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Pre-hospital 12-Lead ST-Segment Monitoring Improves the Early Diagnosis of Acute Coronary Syndrome

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Abstract

Aims/Methods—We studied 620 patients who activated ‘911’ for chest pain symptoms to determine the sensitivity and specificity of 12-lead ECG ST-segment monitoring in the pre-hospital period (PH ECG) for diagnosing acute coronary syndrome (ACS), and to assess whether the addition of PH ECG signs of ischemia/injury to the initial hospital 12-lead ECG obtained in the emergency department (ED) would improve the diagnosis of ACS.

Results—The sensitivity and specificity of the PH ECG were 65.4% and 66.4%. There was a significant increase in sensitivity (79.9%) and decrease in specificity (61.2%) when considered in conjunction with the initial hospital ECG ($p < 0.001$). Those with PH ECG ischemia/injury were more than 2.5 times likely to have an ACS diagnosis than those who had no PH ECG ischemia/injury ($p < 0.001$).

Conclusions—PH ECG data obtained with 12-lead ST-segment monitoring provides diagnostic information about ACS above and beyond the initial hospital ECG.

Introduction

Recent American Heart Association guidelines recommend acquisition of a pre-hospital electrocardiogram (PH ECG) for all patients with symptoms of acute coronary syndrome (ACS) who are transported by ambulance to the emergency department (ED) (1). Transmitting a PH ECG to the ED has the potential to facilitate earlier diagnosis and reduce time to treatment for patients with ACS (2–5). The standard 12-lead ECG is the gold standard for electrocardiographic detection of acute myocardial ischemia/injury and is reported to be the single most important method to rapidly identify ACS in the ED (6). However, the standard ECG has limited sensitivity (30%–70%) and specificity (70%–95%) (4, 5, 7–9) that results in 2% to 5% of patients with ACS being erroneously discharged from the ED, and 70% of patients admitted for suspect ACS not having it (7).

There is little information about the diagnostic value of ECGs triggered by ST-segment monitoring that have been acquired in the pre-hospital setting. Therefore, the aims of this analysis of 12-lead ST-segment monitoring PH ECG data were to: 1) determine the

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sensitivity and specificity of one or more PH ECGs for diagnosing ACS, and 2) determine whether PH ECG data in conjunction with the initial hospital ECG improves diagnostic accuracy for ACS.

Materials/methods

Data for this analysis were obtained from the ST SMART (Synthesized Twelve-lead ST Monitoring and Real-time Tele-electrocardiography) Trial, a prospective randomized clinical trial in Santa Cruz County, California. Santa Cruz County is a large county that includes both urban and rural areas. The total population is estimated to be about 250,000 persons, of which half live in rural coastal mountain areas serviced by winding roads, resulting in long pre-hospital transport times (median '911' to hospital time, 40 minutes). There are two hospitals and a total of 26 emergency vehicles serving the entire county.

The primary aims of the ST SMART Trial were to compare patients with and without PH ECG in paramedic scene time, hospital time to treatment, and survival over the period of the study (10). Patients were enrolled in the study from 2003 to 2008 and underwent follow-up for up to 4 years. Enrollment for the study occurred 7 days a week, 24 hours a day. Paramedics in the field were trained to identify all persons 30 years of age and older who activated 911 with complaints of non-traumatic chest pain, anginal equivalent symptoms such as new onset shortness of breath (not due to chronic lung disease or asthma), and syncope (not due to drug overdose or intoxication). Paramedics initiated special 12-lead ST-segment monitoring and automatic mobile telephone transmission software. Patients randomized to the experimental group had their initial PH ECG and any subsequent ST-event ECGs printed out in the destination ED with an audible voice alarm stating "incoming ECG from the field". Patients in the control group had an ECG after hospital arrival, which was standard of care in the county (10). The Institutional Review Boards at the University of California, San Francisco, and the participating hospitals approved the study. A waiver of patient consent in the field was granted to avoid delays in patients reaching the hospital. Community consent was obtained by placing notices in country newspapers (including a Spanish-language publication) and by posting information on the hospitals' and the Emergency Medical System (EMS) agencies' websites (10). Research nurses obtained written consent from patients meeting inclusion criteria after admission to the ED.

The ECGs analyzed in the present analysis included all those acquired and stored in the pre-hospital ECG device as well as the initial hospital-acquired ECG for all patients. There were a total of 794 patients enrolled in the ST SMART trial. Of 794 patients enrolled, a total of 115 patients were excluded from the analysis because of left bundle branch block (LBBB), left ventricular hypertrophy (LVH), or ventricular paced rhythm, all known confounders of the ST segment (11). An additional 59 patients were excluded because they were enrolled in the study more than once, resulting in a final cohort of 620 patients with accompanying ECGs analyzed for this analysis.

Electrocardiographic procedures/analysis

All 26 paramedic-staffed emergency vehicles responding to 911 calls in the county were equipped with special study-modified portable monitor-defibrillator devices (Lifepak12, Physio-Control, Redmond, Washington). The study device software enabled the following: 1) synthesis of a 12-lead ECG from five electrodes, 2) continuous measurement of ST amplitudes (J + 60 milliseconds) every 30 seconds in all 12 leads, and 3) automatic storage and transmission of an ECG to the destination ED if there was a change in ST amplitude of 0.2 mV in 1 lead or 0.1mV in 2 contiguous leads lasting 2.5 minutes (10). The portable monitor-defibrillator study device collected 20 seconds of electrocardiographic data and then selected the 10 seconds with the best signal-to-noise ratio to develop a noise-free

median beat from which all 12-lead ST-segment measurements were obtained. If the initial 20 second sample was noisy, the device automatically analyzed the subsequent 20 seconds of data (10). All county paramedics were taught to apply the 5 electrodes and manually initiate transmission of the initial PH ECG for patients with ACS symptoms (10). Any subsequent ST-event PH ECGs were transmitted automatically without paramedic decision-making. To ensure successful PH ECG transmissions, the device automatically redialed up to 3 attempts if the EMS vehicle was in a location where mobile telephone communication was unavailable. The study device used a bandwidth of 0.05 to 150 Hz, which is the filtering recommended for diagnostic standard 12-lead ECGs (10). A validation study was conducted to test the reduced lead ECG developed for the ST SMART Trial and standard ECGs (12). A high percentage of agreement between the synthesized pre-hospital ECG and standard 12-lead ECG was determined for ST/T wave changes of acute myocardial ischemia, cardiac rhythm, bundle branch block, and prior myocardial infarction.

All PH ECG data were stored electronically in a central computer and analyzed offline (CodeStat Suite version 8.0, Physio-Control, Redmond, Washington). All ECGs were manually read by the investigator [JZH] using the universal criteria for the diagnosis of ACS defined by the European Society of Cardiology and American College of Cardiology Committee (9). An expert [CES] conducted random audits of ECG analysis to confirm the diagnosis of ACS and establish inter-rater reliability. Universal criteria were developed to increase the sensitivity and specificity of the ECG for ACS by recognizing gender and lead differences. They include the following:

1. New ST elevation at the J point in two contiguous leads with the cut-off points: 0.2 mV in men or 0.15 mV in women in leads V₂ through V₃ and/or 0.1 mV in other leads.
2. New horizontal or down-sloping ST depression 0.5 mV in two contiguous leads; and/or T inversion 0.1 mV in two contiguous leads with prominent R-wave or R/S ratio >1.

The American College of Cardiology key data elements for measuring clinical management and outcomes of patients with ACS were used to define study variables (final diagnosis) and patient characteristics (demographics, cardiac history, coronary risk factors) (13). Patients were assigned a final diagnosis based on their initial hospital ECG analysis, biomarker evidence of necrosis, and medical record review. ACS diagnoses were classified as 1) ST-elevation myocardial infarction (STEMI), 2) non-ST elevation myocardial infarction (NSTEMI), 3) myocardial infarction (MI) of uncertain type, and 4) unstable angina. Unstable angina was further subcategorized into 1) Definite/probable unstable angina or, 2) possible unstable angina. Of these, only the subcategory of definite/probable unstable angina was included in the ACS cohort for this analysis.

Statistical analysis

All data analyses were performed with SPSS software, version 17.0. Descriptive statistics were used to report demographic and clinical information. ECG signs of ischemia/injury (ST elevation, ST depression, T-wave inversion) were collapsed into dichotomous variables for analysis (ACS diagnosis yes/no) to calculate sensitivity, specificity, and positive/negative predictive values.

Logistic regression analysis was used to determine if PH ECG evidence of ischemia/injury added to the initial hospital ECG improved the ability to predict a diagnosis of ACS. In this regression, the dependent variable was ACS diagnosis yes/no. In the first step, a variable for the initial hospital ECG was entered to determine how well it predicted an ACS diagnosis by itself. In the second step, a variable for PH ECG was entered. Odds ratios with 95% CIs

were calculated. The Omnibus Test of Model Coefficients was used to assess the fit of the final model.

Results

Subject characteristics

A total of 620 enrolled patients were included in this analysis. Average age was nearly 70 years, with slightly more male predominance (Table 1). Ethnicity included 92% White, 3% Latino, 2% Asian, 1% Black, and 2% mixed or unknown. A history of hypertension was common, and one third of patients either currently smoked or had a history of smoking. There were 179 patients (28.9%) with a final ACS diagnosis (Table 2).

Sensitivity and Specificity

The sensitivity and specificity of the PH ECG for a diagnosis of ACS was determined to be 65.4% and 66.4%, respectively. There was a significant increase in sensitivity with a reduction in specificity when both the PH ECG and standard ECG were considered together (Table 3). Specifically, the sensitivity increased to nearly 80% while the specificity decreased to 61.2% when *either* the PH ECG or the initial hospital ECG had evidence of myocardial ischemia/injury.

The positive predictive value of the PH ECG was determined to be 68%, indicating that of those predicted to have an ACS diagnosis, the model accurately identified 68% of them. The negative predictive value was 80%, which means the model accurately identified 80% of those predicted not to have an ACS diagnosis.

Predictor of ACS

PH ECG evidence of ischemia/injury was an independent predictor for an ACS diagnosis. Moreover, adding PH ECG evidence of ischemia/injury to the initial hospital ECG significantly improved the overall model fit ($p < 0.001$), making the model a better predictor for an ACS diagnosis when PH ECG ischemia was included than initial hospital ECG data alone (Table 4).

Figure 1 shows three ECGs from a 75 year old male whose wife called 911 from their vacationing campsite because her husband developed chest pain and diaphoresis. The patient had a long cardiac history dating back to the 1970's with hypertension, myocardial infarction, coronary bypass surgery on three occasions, and PCI on two occasions; however, he was currently well and physically active. When EMS arrived, the patient had 7 out of 10 chest pain. A PH ECG was transmitted to the receiving computer that randomized the patient to the control group, which meant that the ECG was not printed out in the ED but stored for later research analysis. This stored PH ECG (Figure 1a) showed down-sloping ST-segment depression and/or T wave inversion in leads I, II, aVF and V₅₋₆. The patient was administered oxygen, aspirin, and a total of four doses of nitroglycerin spray en route to the hospital. The ST-segment monitoring algorithm in the ST SMART device stored an ECG whenever there was a change in ST amplitude lasting at least 2.5 minutes. Because the patient's ST-segment depression was resolving en route to the hospital, it triggered a total of five stored ECGs.

The initial hospital standard 12-lead ECG (Figure 1b) showed prior inferior and antero-septal myocardial infarction, isolated T wave inversion in leads I and aVL, and non-specific T wave abnormalities in leads V₅₋₆. The patient no longer had chest pain and his serum biomarkers were negative. The patient's ECG findings were considered congruent with his past medical history and not indicative of ACS. He was admitted to the hospital for further

observation with a diagnosis of “recurrent angina” and his outpatient oral medications were resumed (beta blocker, diuretic, ACE inhibitor, and anticoagulant).

Thirty hours after admission near midnight, the patient developed severe chest pain and hypotension. A “stat” ECG (Figure 1c) showed down-sloping ST-segment depression similar to his PH ECG. The patient was taken to the cardiac catheterization laboratory at 2 am where his hemodynamic status deteriorated further, requiring insertion of an intra-aortic balloon pump, intubation, and about five minutes of cardiopulmonary resuscitation. His cardiac catheterization revealed occlusions of the right coronary artery and several bypass grafts, and a high-grade stenosis of the left main coronary artery. Following a stent of the left main coronary artery, the patient had a complicated hospital course with cardiogenic shock, ventricular tachycardia, congestive heart failure, renal failure requiring hemodialysis, and aspiration pneumonia with acute respiratory failure. His peak troponin occurred on hospital day three at 496 ng/ml (expected range 0.0–2.3) and his creatinine kinase peaked at 3,015 IU/L (expected range, 24–195). The patient was discharged after 16 days with follow up to cardiac electrophysiology for an implantable cardioverter defibrillator.

In retrospect, it is evident that this patient was experiencing unstable angina because his PH ECG showed dramatic down-sloping ST-segment depressions that were dynamically changing over the pre-hospital period. However, because his ST-segment depressions had virtually resolved by the time of his initial ED ECG, he was not recognized as an ACS patient nor treated according to the guidelines for NSTEMI/unstable angina. It is likely that if the treating physicians had seen the PH ECGs in this patient, more aggressive therapy would have been initiated earlier and that may have prevented the massive and complicated infarction that occurred 30 hours after hospital admission.

Discussion

To our knowledge, this study is the first to report improved sensitivity (nearly 80%) of the ECG for an ACS diagnosis when using ST-segment monitoring-triggered ECGs from the field in conjunction with the initial hospital ECG. The dynamic ST-segment changes between pre-hospital and hospital ECGs were a critical discriminator for an ACS diagnosis. Our findings are in agreement with a study conducted by Kudenchuk and colleagues (5) who reported that when serial ST/T changes between a PH ECG and hospital ECG were considered, the sensitivity significantly increased from 80% to 87%, with a reduction in specificity from 60% to 50% (5). Unlike our study that utilized ST-segment triggered PH ECG, Kudenchuk and colleagues evaluated a conventional 12-lead PH ECG obtained by paramedics in the ambulance. A likely explanation for Kudenchuk et al’s higher sensitivity and lower specificity compared with our results is that they included any sign of ischemic abnormality, not just that for acute ischemia/injury. However, both studies confirm that serial ECG recordings enhance the diagnostic sensitivity for ACS, as compared to a single tracing.

We also found that PH ECG evidence of ischemia/injury is a significant independent predictor of an ACS diagnosis. Those with PH ECG ischemia were 2.5 times more likely to have an ACS diagnosis than those without PH ECG ischemia/injury. In addition, adding PH ECG evidence of ischemia/injury to a model containing the initial hospital ECG significantly improved the ability to predict an ACS diagnosis. These are important findings since nearly one third of patients diagnosed with ACS present without chest pain and have ischemic episodes that are silent, thus easily missed by a single “snap shot” ECG (14). Two studies have evaluated the diagnostic value of an ECG in the pre-hospital setting. Grijseels and colleagues (15) conducted a study to evaluate five previously developed algorithms for the diagnosis of ACS in the pre-hospital setting. They reported the sensitivity (43%–77%)

and specificity (38%–78%) of the five algorithms and found the presence of various ECG abnormalities in the pre-hospital setting to be most predictive of an ACS diagnosis (15). Specific ECG abnormalities included ventricular rhythm, pacemaker rhythm, presence of Q-waves, right or left bundle branch block, LVH, or ECG signs of ischemia/injury. Unlike our study that represented patients contacting 911 for symptoms of ACS, this study included patients who were referred to the ED from a general practitioner's office. In 2005, Svensson and colleagues (16) examined a cohort of patients similar to ours who activated EMS for symptoms of ACS. They reported on the predictive ability of specific ECG signs of ischemia/injury (ST elevation, ST depression, T-wave inversion) found on a PH ECG for an ACS diagnosis. They determined the presence of ST depression had the highest independent predictive value for an ACS diagnosis (OR 2.62, 1.23–5.58, $p<0.05$)(16). Both of these previous investigations by Grijseels and Svensson differed from our study in that they examined the effects of a *single* “snapshot” 12-lead PH ECG rather than multiple PH ECGs triggered by ST-segment monitoring (15, 16).

The parent study found that pre-hospital ST monitoring with mobile phone transmission of ST alarm ECGs to the destination ED was associated with greater paramedic pre-hospital ECG utilization without clinically significant increases in scene time (10). It also resulted in faster time to first ECG in patients with symptoms of ACS, faster time to first drug in patients with non-STEMI or unstable angina, and a suggested trend for faster door-to-balloon times and lower mortality in patients with STEMI.

Limitations

PH ECGs were recorded using a 5- electrode reduced lead configuration that was specifically developed for the ST SMART study. It is important to consider that different methods of ECG acquisition can result in different ST/T wave morphologies, and therefore should be interpreted with caution. Although the validation study determined that the synthesized PH ECG was comparable to the hospital ECG for diagnosis of ischemia/injury, the validation study did not assess diagnostic agreement for LVH. For this reason, we confirmed the diagnosis in all patients with criteria for LVH on their PH ECG by reviewing the hospital ECGs.

Conclusions

PH ECG data obtained with 12-lead ST-segment monitoring provides information above and beyond the initial hospital ECG obtained in the ED. PH ECG enhances the diagnostic sensitivity for ACS and helps to predict ACS in an early phase of emergency cardiac care.

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Figure 1a Pre-hospital ECG **Figure 1b Initial Emergency Department ECG** **Figure 1c ECG 30 hours after hospital admission with chest pain, hypotension** **Figure 1.**

Potential value of the pre-hospital ECG for ACS patients: a case example

Table 1

Baseline characteristics of the cohort (n=620)

Variable	
Age	69.77 ± 14.5
Male sex	320 (52%)
Smoker	211 (34%)
Diabetes	131 (21%)
Hypertension	381 (62%)
History of MI	118 (19%)
History of CAD	194 (31%)
History of Angina Pectoris	184 (30%)

MI=myocardial infarction

CAD=coronary artery disease

Table 2

Patients with a diagnosis of ACS (n=179)

Diagnosis	
STEMI	47
NSTEMI	54
MI of unknown origin	1
Definite/probable UA	77

ACS=acute coronary syndrome

STEMI=ST-elevation myocardial infarction

NSTEMI=non-ST-elevation myocardial infarction

MI=myocardial infarction

UA=unstable angina

Table 3

Sensitivity and specificity of PH ECG alone and in conjunction with the hospital ECG for an ACS diagnosis (n=620)

	PH ECG	PH or Hosp ECG ischemia	P value
Sensitivity	65.4%	79.9%	<0.001
Specificity	66.4%	61.2%	<0.001

Table 4

Logistic regression analysis for ECG predictors of an ACS diagnosis (n=620)

Predictor variables	Odds ratio (95% CI)	P
Hospital ECG ischemia/injury	6.762 (4.47–10.23)	<0.001
PH ECG ischemia/injury	2.543 (1.70–3.80)	<0.001

Omnibus Tests of Model Coefficients $\chi^2(2) = 20.78, p < .001$

CI = confidence interval