Improving the Diagnosis of Culprit Left Circumflex Occlusion With Acute Myocardial Infarction in Patients With a Nondiagnostic 12-Lead ECG at Presentation: A Retrospective Cohort Study

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Background—Left circumflex culprit is often missed by the standard 12-lead ECG. Extended lead systems (body surface potential map [BSPM]) should improve the diagnosis of culprit left circumflex stenosis with myocardial infarction.

Methods and Results—Retrospective analysis of a hospital research registry (August 2000–August 2010) comprising consecutive patients with (1) ischemic-type chest pain at rest; (2) 12-lead ECG and 80-lead BSPM at first medical contact; and (3) cardiac troponin-T 12 hours after symptom onset and/or creatine kinase MB fraction, were undertaken. Enrolled in the cohort were patients with culprit left circumflex stenosis (thrombolysis in myocardial infarction flow grade 0/1) at angiography. Acute myocardial infarction AMI was defined as cardiac troponin-T ≥0.1 µg/L and/or creatine kinase MB fraction >2 upper limits of normal. Enrolled were 482 patients: 168 had exclusion criteria. Of the remaining 314 (age 64±11 years; 62% male), 254 (81%) had AMI: of these, 231 had BSPM STE—sensitivity 0.91, specificity 0.72, positive predictive value 0.93, negative predictive value 0.65, and c-statistic 0.803 for AMI ($P<0.001$). Of those with BSPM STE and AMI (n=231), STE was most frequently detected in the posterior (n=111, 48%), lateral (n=53, 23%), inferior (n=39, 17%), and right ventricular (n=21, 9%) territories.

Conclusions—Among patients with 12-lead ECG non-ST-segment–elevation myocardial infarction and culprit left circumflex stenosis, initial BSPM identifies ST-segment elevation beyond the territory of the 12-lead ECG. Greater use of the BSPM may result in earlier identification of AMI, which may lead to more rapid reperfusion. (*J Am Heart Assoc*. 2019;8:e011029. DOI: 10.1161/JAHA.118.011029.)

Key Words: acute coronary occlusion • acute myocardial infarction • body surface potential mapping • left circumflex artery

In acute coronary syndrome patients, prompt diagnosis of acute coronary occlusion with early reperfusion is associated with a reduction in morbidity and mortality.[1–3] Current clinical practice relies upon initial electrocardiographic changes for the diagnosis of ST-segment elevation (STE) myocardial infarction (STEMI). However, the initial 12-lead ECG has limited sensitivity (50%–60%) for STEMI diagnosis.[4]

In patients with initial non-STEMI diagnosis, ≈25% have an acute coronary occlusion at angiography, 33% of whom have a left circumflex (LCx) as culprit.[5] Undiagnosed acute coronary occlusion at onset of myocardial infarction (ie, missed STEMI) is unsurprisingly associated with high morbidity and mortality,[6–8] with patients presenting as non-STEMI on initial 12-lead ECG representing a high-risk patient group: ≥12.2% die or have a nonfatal acute myocardial infarction (AMI) at 6 months.[4,7]

In addition, the majority of deaths from coronary artery disease occur outside the hospital and are mainly sudden. Following return of spontaneous circulation post cardiac arrest, emergency angiography is currently advocated in patients with STE on the standard 12-lead ECG.[9] This has been shown to improve clinical and neurological outcomes in both the short and medium terms.[10]

In patients with ischemic-type chest pain at rest, one method to improve detection of STE and thus the diagnostic capability of the 12-lead ECG is to sample over a larger thoracic surface area. Previous approaches have focused on the placement of a limited number of additional leads to cover the right ventricle (V_3R–V_5R) and the posterior left ventricular wall (V_7–V_9).

In consecutive patients with chest pain,[4,11,12] sampling a larger and more comprehensive volume of the thorax with multiple leads—a technique known as body surface potential mapping (BSPM)—has been shown to improve the sensitivity

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Clinical Perspective

What Is New?

- In acute myocardial infarction, culprit left circumflex artery occlusion is often "missed" by the standard 12-lead ECG.
- In our study, we have shown that extended 80-lead body surface potential maps improve ST-segment–elevation myocardial infarction diagnosis in these patients at presentation.

What Are the Clinical Implications?

- Early identification and reperfusion of an occluded coronary artery are known to improve morbidity and mortality.
- Body surface potential maps at presentation can identify patients with acute left circumflex occlusion that would have otherwise been "missed" by the 12-lead ECG.
- Triage to more timely revascularization has potential to improve clinical outcomes and long-term prognosis in these patients.

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of AMI diagnosis over that of the 12-lead ECG. This is particularly so in those presenting with ST-segment depression (STD) only on 12-lead ECG.[13] Furthermore, the OCCULT-MI (Optimal Cardiovascular Diagnostic Evaluation Enabling Faster Treatment of Myocardial Infarction) trial, a large prospective study of 1829 high-risk patients presenting to 12 high-volume tertiary care center emergency departments in the United States, showed that BSPM increased STEMI diagnosis by 27.5% over that of the standard 12-lead ECG[14] and that patients with BSPM-only STE and AMI had increased risk (odds ratio 3.4) of death and recurrent AMI at 30 days because of a delayed revascularization strategy.[14]

In the current study, we hypothesized that BSPM acquisition would improve AMI diagnosis in patients with 12-lead ECG non-STEMI and culprit LCx stenosis. Furthermore, we sought to determine whether a BSPM-based approach would have a diagnostic advantage over recording additional right ventricular (RV) and posterior ECG leads only.

Methods

The data, analytic methods, and study materials will not be made available to other researchers for purposes of reproducing the results or replicating the procedure.

Study Population

From our hospital research registry of clinical BSPM trials, we studied retrospectively all patients with chest pain admitted to our coronary care unit between August 2000 and August 2010 using either the emergency department or mobile

coronary care unit who underwent BSPM. Those who fulfilled the following criteria entered into the study:

1. Typical ischemic-type chest discomfort of ≥20 minutes duration, occurring at rest and presenting within 12 hours of symptom onset;
2. 12-lead ECG and 80-lead BSPM performed at first medical contact;
3. Blood sampled for cardiac troponin T (cTnT) 12 hours post symptom onset and/or creatine kinase MB fraction (CK-MB); and
4. LCx artery culprit stenosis at angiography during index hospitalization.

Patients were excluded from analysis if they had any of the following: 12-lead ECG STEMI[15] or any condition precluding assessment of the ST-segment (eg, left bundle branch block, right bundle branch block, left ventricular hypertrophy, concomitant digitalis therapy or ventricular pacing); prior history of coronary bypass surgery; received fibrinolysis, nitrates, or glycoprotein IIb/IIIa inhibitor before initial 12-lead ECG or BSPM; or >15 minutes delay between initial 12-lead ECG and BSPM recording) (Figure 1).

Demographic data and risk factors for coronary artery disease were documented (Table 1).

Twelve-Lead ECG Analysis

A 12-lead ECG was recorded at first medical contact (25 mm/s and 10 mm/mV). ST-segment shifts were measured at the J-point for ST-segment elevation (STE) and 80 ms after the J-point for STD using the preceding TP segment as a baseline[16] by a cardiologist who was blinded to all other clinical data. STD ≥0.05 mV assessed in leads V_1 to V_3 was considered suggestive of posterior myocardial ischemia.[15,17] STE was considered suggestive of acute coronary artery occlusion if present in 2 contiguous ECG leads with the cut points:
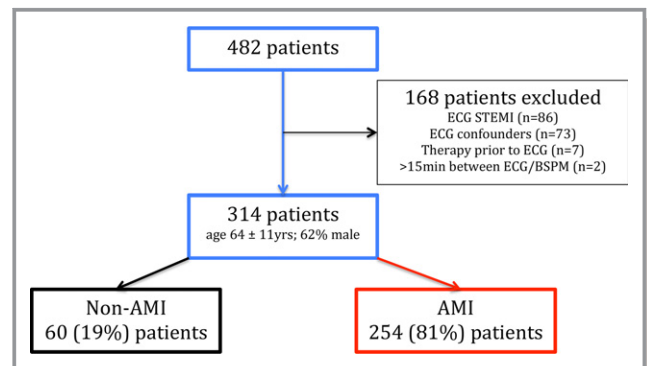

Figure 1. Patient enrollment methodology. AMI indicates acute myocardial infarction; BSPM, body surface potential map; STEMI, ST-segment–elevation myocardial infarction.

Table 1. Baseline Characteristics

	AMI (n=254)	Non-AMI (n=60)	P Value
Age, y	63±12	66±13	NS
Male sex, n (%)	152 (60)	43 (72)	0.034
BMI, kg/m^2	22±5	21±4	NS
Risk factors, n (%)			
Hypertension	164 (65)	28 (47)	0.035
Hyperlipidemia	148 (58)	23 (38)	0.042
Current smoker	127 (50)	26 (43)	0.037
Diabetes mellitus	88 (35)	22 (37)	NS
Family history of CAD	63 (25)	17 (28)	NS
Past medical history, n (%)			
Prior MI	23 (9)	10 (17)	0.048
Prior angina	58 (23)	15 (25)	NS
Prior PCI	20 (8)	12 (20)	0.041
Multivessel disease, n (%)	30 (12)	7 (12)	NS
GFR, mL/min	55±5	47±12	NS
Time to treatment, median (IQR)			
Symptom onset to first medical contact, h	1.2 (0.9, 1.7)	1.4 (1.0, 1.9)	NS
First medical contact to 12-lead ECG, min	8 (5, 11)	10 (6, 12)	NS
12-lead ECG to angiography, h	23 (21, 32)	21 (19, 30)	NS

Results are expressed as number (%), mean±SD, or median (interquartile range [IQR]). AMI indicates acute myocardial infarction; BMI, body mass index; CAD, coronary artery disease; GFR, glomerular filtration rate; MI, myocardial infarction; NS, not significant; PCI, percutaneous coronary intervention.

≥0.1 mV in all leads other than V_2 to V_3 where the following cut points apply: ≥0.25 mV in men <40 years, ≥0.2 mV in men ≥40 years, or ≥0.15 mV in women.[15]

Left ventricular hypertrophy was defined as a sum of both the R wave in leads V_5 or V_6 and S wave in V_1 >3.8 mV.[18] Left bundle branch block was defined as QRS duration ≥120 ms, QS or rS wave in lead V_1, and slurred R waves in leads I and V_5 or V_6.[18] Right bundle branch block was defined as QRS duration ≥120 ms, rSR' complex in leads V_1 and V_2, and S waves in leads I and V_5 or V_6.[18]

BSPM Analysis

The BSPM was recorded with a flexible plastic anterior and posterior electrode harness and a portable recording unit (Heartscape Technologies, Inc.). The anterior harness contains 64 electrodes, including 3 proximal bipolar limb leads (Mason-Likar position) and a posterior harness with 16 electrodes (Figure 2).[11] This lead configuration enables

recording of 77 unipolar ECG signals with respect to the Wilson central terminal. During the interpretation process, the electrodes were defined to represent anterior, lateral, inferior, high right anterior, RV, and posterior epicardial regions.[11,12] Harness application takes 3 to 4 minutes. BSPMs were recorded over 5 to 10 s at a sampling rate of 1 kHz and a bandwidth of 0.05 to 100 Hz and transferred into digital format for core laboratory analysis.[17]

The BSPMs were uploaded and displayed on an IBM-compatible computer running PRIME analysis software. All 80 leads were manually checked and those of unacceptable quality (ie, where noise or movement artifact disallowed recognition of QRST variables) were marked and substituted using linear grid interpolation.[18] Any BSPM with >6 leads requiring interpolation were disregarded and these patients were excluded from analysis. Printouts were obtained from the processed BSPM of the 80-lead ECG and a color-contour map displaying the amount of STE at the J point (ST0 isopotential map). Using the 80-lead BSPM and color-contour map, a single cardiologist familiar with BSPM interpretation and blinded to both the clinical details and 12-lead ECG coded the BSPM diagnosis as AMI or non-AMI and defined the infarct location (mean interpretation time: 6 minutes). STE was measured at the ST0 point and defined by the following thresholds: anterior ≥0.2 mV elevation; lateral/inferior/high right anterior/RV ≥0.1 mV elevation; posterior ≥0.05 mV elevation. Infarct location was assessed using the ST0 isopotential color-contour map. The area with maximal STE (maxima) on BSPM defined the principal infarct location; with other STE locations, above the respective thresholds but less than the maxima, also coded for each patient.

All BSPMs were retrospectively analyzed to assess STE in the extended 12-lead ECG leads, ie, V_3R to V_5R (BSPM leads 5, 74, and 69, respectively)[11] and V_7 to V_9 (BSPM leads 63, 65, and 66, respectively).[11] STE, measured at the J point (ST0 isopotential map), was considered significant if ≥0.05 mV in any of the RV chest leads (V_3R–V_5R) and/or any of the posterior chest leads (V_7–V_9).

AMI Definition

Diagnosis of AMI was made when cTnT ≥0.1 μg/L (Roche Diagnostics, Switzerland) and/or CK-MB >2 upper limit of normal (Roche Diagnostics, Switzerland).

Coronary Angiography Criteria

All patients underwent coronary angiography during index admission. Stenosis was considered clinically significant if the luminal diameter was narrowed ≥70% in any projection. The
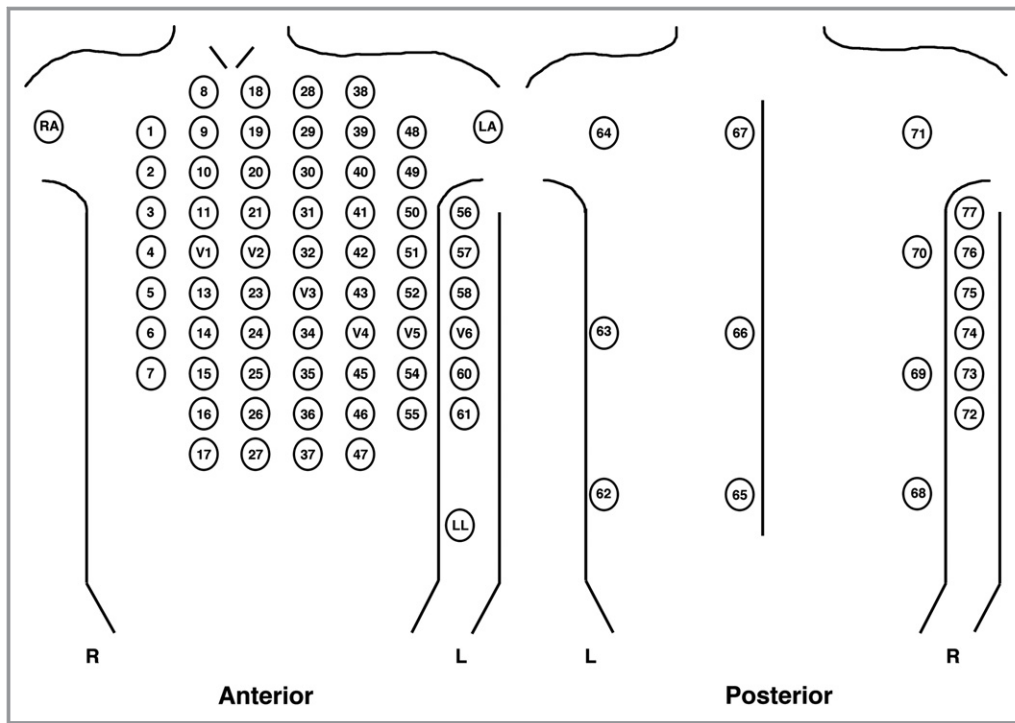
Figure 2. Schematic of the 80-lead body surface potential map electrode positions.

infarct-related artery was defined as that with the most severe stenosis (ie, culprit). In addition, flow in the infarct-related artery was graded according to the thrombolysis in myocardial infarction flow grade criteria.[19] Only patients in whom the LCx was considered the culprit infarct-related artery with thrombolysis in myocardial infarction flow grade 0/1 were included.

Ethics

The Northern Ireland Regional Ethics Committee and the Institutional Research Committee of the Royal Victoria Hospital, Belfast, approved the study. All patients gave informed consent to BSPM and inclusion in a hospital research registry.

Statistical Analysis

Baseline categorical variables were analyzed by χ^2 and continuous clinical variables by analysis of variance. Sensitivity and specificity of the various diagnostic methods were calculated by comparing the prediction of AMI against cTnT/CK-MB criterion standard. Receiver operating characteristic curve analysis was undertaken. The area under each curve (c-statistic) was determined, and the sensitivity and specificity (with 95% CIs) were calculated for optimal cut-off values. Statistical analysis was performed using SPSS version 17.0 for Windows (SPSS Inc, Chicago, IL). A $P<0.05$ was taken as statistically significant.

Results

Baseline Characteristics

Of the 482 patients who entered the study, 168 were excluded from analysis because of STEMI (n=86), 12-lead ECG confounders (n=73), therapy before 12-lead ECG and/or BSPM (n=7), and a delay >15 minutes between 12-lead ECG and BSPM (n=2). No patient was excluded for having >6 leads of poor quality on BSPM. Thus, 314 patients met the study criteria (62% male; age 64±11 years) (Figure 1), 254 (81%) of whom had AMI by cTnT/CK-MB definition. Patients with AMI had higher incidence of hypertension, dyslipidemia, and cigarette smoking than non-AMI patients (Table 1). However, non-AMI patients were more likely to be male and have a prior history of AMI and/or percutaneous coronary intervention (PCI) than those with AMI. Otherwise, baseline characteristics between groups were similar.

BSPM Diagnosis

Of the 254 (81%) who had AMI, 231 patients had BSPM STE——sensitivity 0.91 (95% CI: 0.77–1.00), specificity 0.72 (95% CI: 0.61–0.85), positive predictive value 0.93 (95% CI: 0.84–1.00), negative predictive value 0.65 (95% CI: 0.56–0.85), and c-statistic 0.803 (95% CI: 0.700–0.907) for AMI diagnosis (*P*<0.001). Of those with BSPM STE and AMI (n=231), STE maxima were most frequently detected in the posterior (n=111, 48%), lateral (n=53, 23%), inferior (n=39, 17%), RV

(n=21, 9%), and high right anterior (n=7, 3%) territories (Table 2, Figures 3 and 4). Furthermore, there was overlap with other territories not reaching the maxima but above the threshold, most frequently occurring in the inferoposterior and inferolateral regions.

Additional analysis of those with posterior AMI was undertaken. Of the 111 (48%) patients with posterior-territory BSPM STE and AMI, 80 (72%) patients had STE in ≥1 of BSPM leads 63, 65, and 66, equivalent to V_7 to V_9 leads, respectively, in whom only 45 (56%) patients had STD in either V_1, V_2, or V_3. In addition, further analysis of those with RV-territory AMI was undertaken. Of the 21 (9%) patients with RV-territory BSPM STE and AMI, 14 (67%) patients had STE in ≥1 of BSPM leads 5, 74, and 69, equivalent to V_3R to V_5R leads, respectively.

Further additional analysis was therefore undertaken with the assumption that in routine clinical practice (1) additional posterior electrodes V_7 to V_9 would be applied when STD was present in either V_1, V_2, or V_3 on 12-lead ECG[15] (ie, 45 patients with V_7 to V_9 STE and AMI in this study); and (2) additional RV electrodes V_3R to V_5R would be applied in patients with inferior territory STE on 12-lead ECG to assess for concomitant RV infarction,[15,20] but no patients with inferior territory STEMI (≥0.1 mV in inferior leads) entered this study as all 12-lead ECG STEMI were excluded at enrollment. Thus, when these 45 patients were excluded from analysis, 209 patients had AMI by cTnT/CK-MB definition (Table 3). Of these, 186 had BSPM STE—sensitivity 0.89, specificity 0.72, positive predictive value 0.92, and negative predictive value 0.65 for AMI diagnosis (P<0.005) (Table 3). However, when all patients with AMI and either STE in ≥1 of BSPM leads 63, 65, 66, ie, V_7 to V_9, with STD in V_1 to V_3 (n=45) or STE in ≥1 of BSPM leads 5, 74, 69, ie, V_3R to V_5R (n=14) were excluded from analysis (n=59), 195 patients had AMI by cTnT/CK-MB definition (Table 3). Of these, 172 patients had BSPM STE and AMI—sensitivity 0.88 and specificity 0.72 for AMI diagnosis (P<0.005) (Table 3).

Furthermore, when all patients with AMI and either STE in ≥1 of BSPM leads 63, 65, 66, ie V_7 to V_9 (n=80) or ≥1 of BSPM leads 5, 74, 69, ie, V_3R to V_5R (n=14) were excluded from analysis (n=94), 160 patients had AMI by cTnT/CK-MB definition (Table 3). Of these, 137 patients had BSPM STE and AMI—sensitivity 0.86 and specificity 0.69 for AMI diagnosis (P<0.005) (Table 3).

Discussion

Current clinical practice guidelines recommend a door-to-balloon time of ≤90 minutes for STEMI patients undergoing primary PCI.[1] Multiple studies have demonstrated increasing morbidity and mortality with treatment delay in such patients,[7] with each 30-minute delay to primary PCI having been estimated to increase the relative risk of 1-year mortality by 7.5%.[7,21] These guidelines also recommend an emergent invasive strategy (<2 hours from hospital admission) for patients with acute coronary syndrome and either hemodynamic instability or features of ongoing ischemia.[22] An early invasive strategy (<24 hours from hospital admission) for high-risk patients with GRACE (Global Registry of Acute Coronary Events) risk score >140, temporal change in troponins, dynamic ST-changes, or prior coronary revascularization is also recommended.[22] Although patients with an acute total occlusion of a coronary artery would likely benefit from either primary or emergent PCI, an absence of characteristic 12-lead ECG changes or hemodynamic instability can exclude them from such an approach. This is increasingly pertinent as there are an increasing number of patients with AMI presenting as non-STEMI, particularly in the elderly population with multiple comorbidities and frailty. Similarly, in primary PCI studies of AMI there is a paucity of patients with LCx occlusion as the culprit vessel.

In a recent meta-analysis, patients with non-STEMI and acute coronary occlusion had a mean delay >24 hours to PCI and were consequently at increased risk of both major adverse cardiovascular events (risk ratio 1.41) and all-cause mortality (risk ratio 1.67).[5] Furthermore, the Occluded Artery Trial has shown no benefit from PCI ≥24 hours after symptom onset in patients with an occluded infarct artery.[23] Thus, patients with an acute coronary occlusion but without 12-lead ECG STE (ie, missed STEMI) are more likely to undergo a delayed revascularization strategy that is of limited prognostic impact.

It is well known that the 12-lead ECG is particularly insensitive for LCx occlusion because of the absence of lateral precordial leads and the late depolarization of the lateral wall.[13,24] Classical STE of ≥0.1 mV in at least one of leads I, aVL, V_5, and V_6 is seen in only 48% of patients with LCx occlusion.[13,24,25] In the PARAGON–B (Platelet IIb/IIIa Antagonism for the Reduction of Acute Coronary Syndrome Events in a Global Organization Network) trial of non-STEMI patients

Table 2. AMI Territory as Indicated by BSPM STE Maxima

BSPM STE (Maxima) Territory	AMI (n=231) n (%)
Posterior	111 (48)
Lateral	53 (23)
Inferior	39 (17)
Right ventricular	21 (9)
High right anterior	7 (3)

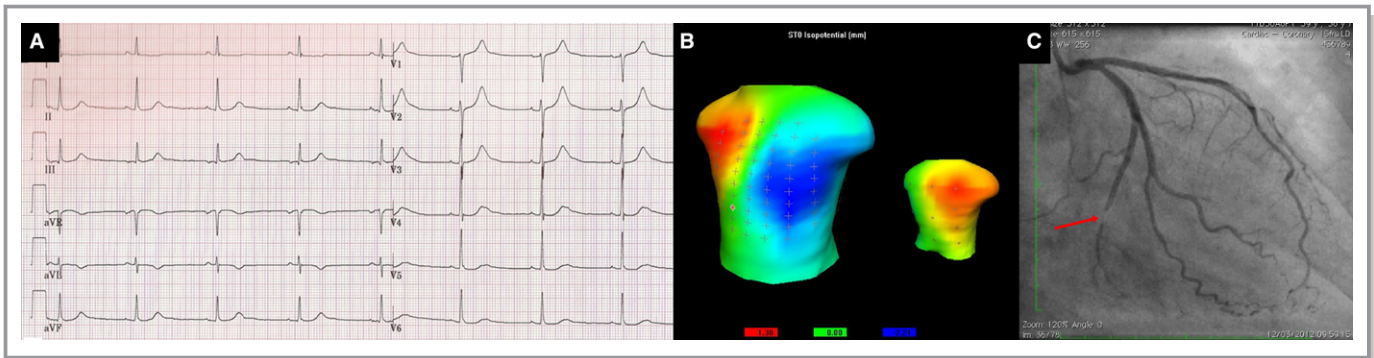AMI indicates acute myocardial infarction; BSPM, body surface potential map; STE, ST-segment elevation.

Figure 3. Case example (**A**) 12-lead ECG showing minimal lateral territory ST-segment sagging (V_5–V_6) with T-wave inversion in lead aVL; (**B**) ST0 isopotential BSPM showing high right anterior and posterior STE (red maxima 1.38 mm)[13]; and (**C**) coronary angiogram showing distal LCx stenosis (red arrow). BSPM indicates body surface potential map; LCx, left circumflex artery; STE, ST-segment elevation.

(n=1957), 27% had an occluded culprit artery at angiography, identified as the LCx in 25.1%.[24] Despite similar in-hospital treatment, those with an occluded culprit artery had larger infarcts, as defined by peak cardiac biomarker titer, and higher risk-adjusted 6-month mortality (hazard ratio 1.72, 95% CI 1.07–2.79) than those with a nonoccluded culprit artery. In addition, the TRITON-TIMI 38 (Trial to Assess Improvement in Therapeutic Outcomes by Optimizing Platelet Inhibition with Prasugrel – Thrombolysis in Myocardial Infarction 38) trial substudy of acute coronary syndrome patients with isolated anterior STD (n=1198) showed that 26.2% of patients had an occluded culprit artery at angiography. Of these, acute LCx occlusion was identified in 48.4%.[7] In this substudy, patients with an occluded culprit artery and elevated biomarkers were significantly more likely to die or have a nonfatal MI at 30 days than those with a patent artery and negative biomarkers (hazard ratio 3.06, 95% CI 1.33–7.03, P=0.008).

Martin et al[26] have shown standard 12-lead ECG STE to have only 50% sensitivity for AMI using contrast-enhanced cardiac magnetic resonance imaging as the diagnostic criterion standard. However, sensitivity increased significantly to 84% (P<0.0001) with the inclusion of STD criteria: ≥0.1 mV STD in ≥2 anatomically contiguous leads or in 1 lead that was anatomically contiguous to a lead with STE criteria.[26] Schmitt et al have also shown the 12-lead ECG to have poor diagnostic sensitivity (<50%) for LCx occlusion in patients with acute chest pain and AMI. In their study, STE ≥0.1 mV in the additional posterior leads (V_7–V_9) and RV leads (V_3R–V_6R) improved the diagnostic sensitivity by 11%.[27] Zalenski et al[28] have shown in their study of 345 non-STEMI patients that STE ≥0.1 mV in at least 2 additional nonstandard leads (V_7–V_9 and V_4R–V_6R) improved the diagnostic sensitivity of the ECG by only 8.4% (P=0.03).[28]

The 80-lead BSPM has been shown to have sensitivity 76%, specificity 92%, and c-statistic 0.84 for AMI diagnosis in consecutive patients presenting with acute ischemic-type chest pain at rest.[11] Improvement in sensitivity over the 12-lead ECG (sensitivity 68%) is mainly because of detection of STE in the high right anterior, posterior, and right ventricular territories not identified by the 12-lead ECG.[11] In a prospective, multicenter (US/UK) trial conducted in 647
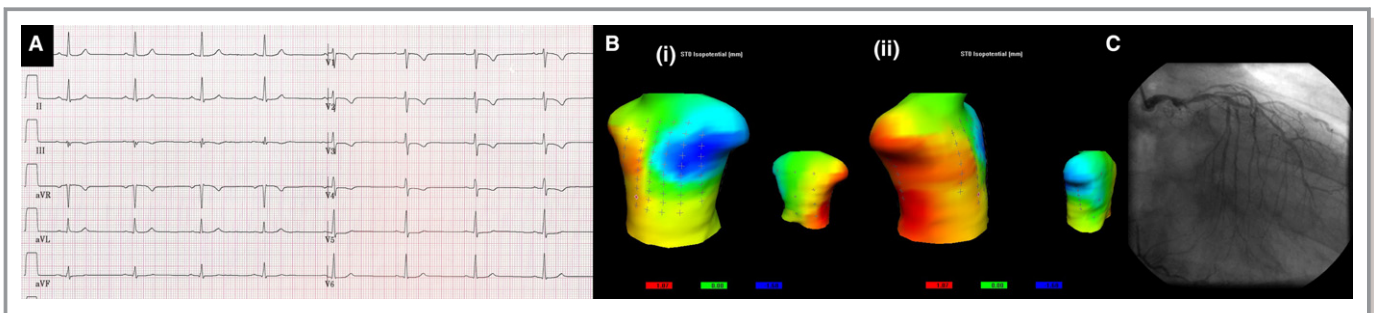

Figure 4. Case example (**A**) 12-lead ECG showing 0.05 mV STD in leads V_3 to V_5 and T-wave inversion in lead III and V_1 to V_4; (**B**) ST0 isopotential BSPM showing (i) anterior territory minima (blue) (−1.68 mm) and (ii) right ventricular and posterior maxima (red) (1.07 mm); and (**C**) coronary angiogram showing culprit occlusion of the proximal LCx, with 60% to 70% stenoses in both the distal LMS and proximal LAD. BSPM indicates body surface potential map; LAD, left anterior descending artery; LCx, left circumflex artery; LMS, left main-stem artery; STD, ST-segment depression.

Table 3. ROC Analysis of BSPM Subsets for AMI Diagnosis

AMI by cTnT/CK-MB Definition (n=254) Exclusions:	AMI by cTnT/CK-MB (n)	BSPM STE and AMI (n)	AMI Diagnosis (95% CI)				
			Sensitivity	Specificity	PPV	NPV	c-Statistic
BSPM STE in leads 63, 65 and/or 66, ie V_7 to V_9 with 12-lead ECG STD in V_1 to V_3 (n=45)	209	186	0.89 (0.76–1.00)	0.72 (0.60–0.86)	0.92 (0.82–1.00)	0.65 (0.51–0.84)	0.775 (0.670–0.883)
BSPM STE in leads 63, 65 and/or 66, ie V_7 to V_9 with 12-lead ECG STD in V_1 to V_3 and BSPM STE in leads 5, 74 and/or 69, ie V_3R to V_5R (n=59)	195	172	0.88 (0.75–1.00)	0.72 (0.61–0.87)	0.91 (0.80–1.00)	0.65 (0.54–0.85)	0.771 (0.638–0.907)
BSPM STE in leads 63, 65 and/or 66, ie V_7 to V_9 and BSPM STE in leads 5, 74 and/or 69, ie V_3R to V_5R (n=94)	160	137	0.86 (0.73–0.98)	0.69 (0.55–0.82)	0.89 (0.79–1.00)	0.65 (0.52–0.87)	0.752 (0.628–0.875)

AMI indicates acute myocardial infarction; BSPM, body surface potential map; CK-MB, creatine kinase MB fraction; cTnT, cardiac troponin T; NPV, negative predictive value; PPV, positive predictive value; ROC, receiver operating characteristic; STD, ST-segment depression; STE, ST-segment elevation.

patients presenting with acute chest pain, BSPM improved the diagnosis of biomarker-confirmed STEMI by 32.2% when troponin was used, with no significant change in specificity. RV involvement complicating inferior STEMI was detected by BSPM in 22% patients with biomarker-defined MI.[29]

BSPM-only STEMI patients have adverse clinical outcomes similar to those of ECG STEMI patients treated with a delayed or conservative invasive strategy.[14] In the OCCULT-MI trial, BSPM increased STEMI detection by 27.5% over the 12-lead ECG.[14] In this trial, BSPM-only STEMI was associated with an increased risk of death and MI at 30 days (odds ratio 3.4).[14]

Criticism has suggested that BSPM does not add significantly to STEMI diagnosis determined by an extended 18-lead ECG (ie, the standard 12-lead ECG with the addition of V_7 to V_9 and V_3R to V_5R). However, in our study, BSPM at presentation identified STE "missed" by the 12-lead ECG in 231/254 (91%) of those with a culprit LCx stenosis and AMI. Of these, STE was most frequently detected in the posterior (n=111, 48%) territory. However, only 45/111 (41%) had STD in either V_1 to V_3 that would indicate the addition of posterior leads (V_7–V_9) to the standard ECG in clinical practice. Our analysis has shown the 80-lead BSPM to improve detection of culprit LCx occlusion over an extended 18-lead ECG, with sensitivity 0.86, specificity 0.69, and c-statistic 0.752 for AMI diagnosis (Table 3). BSPM superiority over the 18-lead ECG is likely because of the BSPM sampling a greater area of the patients' right side and permitting STE detection in the lower posterior and right posterolateral regions through additional posterior chest lead placement.

In previously published work from our unit, epicardial potentials derived from the BSPM, a technique known as inverse electrocardiography, have been shown to further improve BSPM sensitivity for AMI diagnosis—sensitivity 98%, specificity 79%, positive predictive value 85%, and negative predictive value 97%.[30] Moreover, early BSPM in patients with ventricular fibrillation cardiac arrest and return of spontaneous circulation improve diagnosis of acute coronary occlusion (sensitivity 88%, specificity 100%), with 33% of those studied having culprit LCx occlusion and STE beyond the territory of the 12-lead ECG.[31]

Early BSPM has the potential to identify STE and AMI in those with culprit LCx stenosis and nondiagnostic 12-lead ECG at presentation. These patients would likely benefit from emergent revascularization to improve prognosis and reduce adverse clinical outcomes (ie, late heart failure, malignant arrhythmias, and mortality). Future randomized studies are required to assess whether early BSPM as part of a clinical decision pathway improves prognosis in all patients presenting with acute ischemic-type chest pain.

Limitations

This study is a nonrandomized retrospective analysis and as such it is possible that both identified and unidentified confounders may have influenced the results. Consecutive patients admitted to the coronary care unit with nondiagnostic initial 12-lead ECG at presentation and LCx culprit stenosis

on angiography were studied. This represents a highly selected, potentially high-risk patient group and is not representative of all patients presenting to the emergency department with ischemic-type chest discomfort. In addition, patients had to survive to angiography and the distribution of patent and occluded arteries among patients who died before angiography is not known. Furthermore, 12-lead ECG, BSPM, and angiography were not performed simultaneously; therefore, a patent artery at the time of 12-lead ECG may have occluded in the time from ECG/BSPM to angiography and vice versa. Diagnosis of acute myocardial infarction required cTnT ≥0.1 μg/L 12 hours post symptom onset and did not require temporal change in cTnT.

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Disclosures

None.

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