Body Surface Potential Mapping Improves Diagnosis of Acute Myocardial Infarction in those with Significant Left Main Coronary Artery Stenosis

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Abstract

Significant stenosis of the left main coronary artery is encountered at angiography in approximately 0.5% of patients with acute myocardial infarction. Non-invasive diagnosis of left main coronary artery stenosis is challenging. It may result in ST-segment abnormalities on initial 12-lead electrocardiogram, e.g. ST-elevation in lead aVR, anterolateral ST depression and/or T-wave inversion. Acute myocardial infarction as a consequence of left main coronary artery obstruction has a poor prognosis and thus early diagnosis is essential to expedite prompt revascularisation.

In this study, we have shown that in patients with acute chest pain and significant left main coronary artery stenosis, ST-segment elevation detected on Body Surface Potential Mapping has an improved sensitivity (93%) over standard ECG in the diagnosis of acute myocardial infarction.

1. Introduction

Currently, the 12-lead ECG is at the centre of the therapeutic decision pathway for acute coronary syndromes since evidence suggests that ST-segment elevation (STE) identifies patients who benefit from early reperfusion therapy [1]. ST elevation myocardial infarction (STEMI) using the standard 12-lead ECG requires ≥0.1mV STE in one or more of leads I, II, III, aVL, aVF, V_5, V_6 or ≥0.2mV STE in one or more of leads $V_1 - V_4$ [2]. In the context of a non-diagnostic 12-lead ECG, e.g. ST depression (STD), T-wave inversion (TWI) and/or STE in lead aVR, the additional lead set provided by the Body Surface Potential Map (BSPM) can identify acute STEMI in areas beyond the standard 12-lead set of the ECG [3], in particular the high right anterior, posterior and right ventricular territories [4].

In most individuals, the left main stem (LMS) coronary artery supplies a significant proportion of the left ventricle (LV) [5] in particular the right upper side of the heart, i.e. the outflow tract of the right ventricle and the basal part of the interventricular septum [6], with ~75% of LV mass dependent upon it for perfusion [7]. LMS disease is usually accompanied by right coronary artery dominance, well-developed collateral channels and significant disease elsewhere in the coronary tree leading to symptoms and presentation prior to complete LMS obstruction [7]. Despite being recognized as the most critical coronary lesion, acute myocardial infarction (AMI) in the presence of significant LMS stenosis continues to be a diagnostic challenge.

We hypothesized that the additional lead set provided by the BSPM will improve detection of STE, particularly in the high right anterior region – a finding consistent with the injury vector associated with both STE in lead aVR and STD in V_4-V_6 on 12-lead ECG.

2. Methods

2.1. Study population

Between January 2000 and January 2010 we retrospectively studied all patients admitted to our coronary care unit using either the emergency department or mobile coronary care unit (MCCU). Patients were excluded from analysis if they were unable to provide informed consent or had any of the following: conditions precluding STE on ECG, i.e. left bundle branch block defined as QRS duration ≥ 120ms, QS or rS wave in lead V_1 and slurred R waves in leads I and V_5 or V_6 [8], right bundle branch block defined as QRS duration ≥ 120ms, rSR' complex in leads V_1 and V_2 and S waves in leads I and V_5 or V_6 [8], left ventricular hypertrophy defined as a sum of the R wave in leads V_5 or V_6 and S wave in V_1 ≥ 3.8mV [9], digitalis therapy or ventricular pacing (247 patients); had received fibrinolytic therapy, nitrates or glycoprotein IIb/IIIa inhibitors prior to initial ECG or BSPM; prior history of coronary artery bypass grafting (CABG) surgery; BSPM recorded >15mins after initial 12-lead ECG. Those who fulfilled the following criteria were studied:

1. Typical ischaemic-type chest discomfort of ≥20minutes duration, occurring at rest and within 12 hours of onset of symptoms

2. Twelve-lead ECG and BSPM available at first medical contact
3. Blood sampled for cardiac troponin T (cTnT) ≥ 12hrs post-symptom onset
4. Coronary angiography during index hospitalization showing significant LMS stenosis

Demographic data and risk factors for coronary artery disease were also collected.

2.2. Twelve-lead ECG analysis

A 12-lead ECG was recorded at first medical contact (25mm/s and 10mm/mV). ST segment shifts were measured at the J-point for STE and 80ms after the J-point for STD using the preceding TP segment as a baseline [10] by a cardiologist who was blinded to all other clinical data. STE consistent with AMI (STEMI) was defined using the Minnesota code 9-2 [2] as ≥0.1mV STE in one or more of leads I, II, III, aVL, aVF, V_5, V_6 or ≥0.2mV STE in one or more of leads V_1 – V_4. STE in lead aVR was defined as ≥0.05mV [11]. STD was ≥0.05mV. TWI was ≥0.1mV, but was not assessed in leads III or V_1.

2.3. BSPM analysis

The BSPM comprises 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. This lead configuration enables recording of 77 unipolar ECG signals with respect to the Wilson central terminal. During the interpretation process the electrodes are defined to represent anterior, lateral, inferior, high right anterior, right ventricular and posterior epicardial regions [9]. All 80 leads were manually checked and those of unacceptable quality, i.e. where noise or movement artefact disallowed recognition of QRST variables, were marked and substituted using linear grid interpolation. Any BSPM with >6 leads requiring interpolation were disregarded and these patients excluded from analysis. Printouts were obtained from the processed BSPM of the 80-lead ECG and a colour-contour map displaying the amount of STE at the J point (ST0 isopotential map). The result of the PRIME™ diagnostic algorithm was noted. Using the 80-lead BSPM and colour-contour map, a single cardiologist familiar with BSPM interpretation and blinded to the clinical details, 12-lead ECG and PRIME™ diagnostic algorithm result coded the BSPM diagnosis as AMI or non-AMI and defined the infarct location. STE was measured from the ST0 point and defined by the following thresholds: anterior ≥0.2mV elevation; lateral/inferior/high right anterior/right ventricular ≥0.1mV elevation; posterior ≥0.05mV elevation; with infarct-location described by the ST0 isopotential colour-contour map.

2.4 Acute myocardial infarction

Diagnosis of AMI was made when cTnT ≥0.03μg/L.

2.5 Coronary Angiography

All patients underwent coronary catheterization during index admission. All coronary angiograms were evaluated by one cardiologist blinded to all other clinical data. The stenosis in the LMS was considered significant if the luminal diameter was narrowed ≥70% in any projection. The infarct related artery was defined as that with the most severe stenosis. When the LMS was considered to be the infarct-related artery, the stenosis was ≥ 70% luminal diameter.

3. Results

3.1. Baseline characteristics

During the study period, 108 patients (age 67 ± 11; 84% male) fulfilled the study criteria, i.e. had significant LMS stenosis on coronary angiography. Baseline clinical and demographic characteristics are shown in Table 1. Of note were: (a) a preponderance to male gender (84%); (b) a higher proportion with hypertension (61%); and (c) a high proportion with renal dysfunction (mean eGFR 47 ±12 ml/hr). Prior histories of angina, AMI and/or Percutaneous coronary intervention were relatively infrequent, suggesting index presentation in this patient group. Furthermore, admission via the Mobile Coronary Care Unit was common (40%).

3.2. Twelve-lead ECG diagnosis

In diagnosis of AMI, the performance of various 12-lead ECG abnormalities are summarised in Table 2. Using the Minnesota 9-2 criteria, 11 patients were classified as having STEMI at presentation, with 9/11 (82%) patients having AMI, i.e. cTnT ≥ 0.03μg/L (sensitivity 11%, specificity 88% for AMI diagnosis). Of those with AMI, 74/83 (89%) patients did not have STEMI by Minnesota 9-2 criteria definition on the initial 12-lead ECG, i.e. NSTEMI diagnosis. STE ≥0.05mV in aVR was detected in 19 patients, 17 (89%) of these having AMI. STE aVR had sensitivity 20% and specificity 88% for AMI diagnosis. STD in ≥2 contiguous leads was detected in 46 patients, 41 (89%) of whom had AMI, giving sensitivity 49% and specificity 71% for the diagnosis. TWI in ≥2 contiguous leads was detected in 30

patients, 26 (87%) of these having AMI, giving sensitivity 31% and specificity 76% for the diagnosis.

Table 1. Baseline clinical and demographic characteristics

	Patients with significant LMS stenosis (n=100)
Age (mean ± SD)	67 ± 11
Male	84%
Admitted via MCCU	40%
AMI	83%
Risk Factors:	
Hypertension	61%
Hyperlipidaemia	32%
Diabetes mellitus	40%
GFR (ml/hr) [mean±SD]	47 ± 12
Current smoker	36%
Family history of IHD	43%
BMI (kg/m^2)	29.1 ± 4.2
Previous angina	22%
Previous AMI	11%
Previous PCI	5%

Values are number of patients (percentage) or mean ± SD.
AMI = acute myocardial infarction; BMI = Body mass index; GFR = glomerular filtration rate; IHD = ischaemic heart disease; MCCU = mobile coronary care unit; PCI = percutaneous coronary intervention; SD = standard deviation

Table 2. Univariate analysis of initial 12-lead ECG for the diagnosis of AMI

	Patients with significant LMS stenosis (n = 100; 83 with AMI, 17 without AMI)		
	n	Sens	Spec
STEMI (Minnesota 9-2 code)	11	11%	88%
STE in aVR ≥0.05mV	19	20%	88%
STD ≥0.05mV in ≥2CL	46	49%	71%
TWI ≥0.1mV in ≥2CL	30	31%	76%

CL = contiguous leads; n = number; Sens = sensitivity; Spec = specificity

3.3. Body surface potential map diagnosis

Of the 100 patients with significant LMS stenosis, 82 and 80 patients were classified as having ST-segment elevation AMI by the physician on analysis of the 80-lead ECG and the PRIME™ diagnostic algorithm respectively (Table 3). Of those with AMI, physician's diagnosis of STE on BSPM occurred in 77 patients, and had sensitivity 93% and specificity 71% for the diagnosis.

High right anterior, right ventricular and posterior territory STE were frequently encountered, with the combinations of anterior/lateral and right ventricular/posterior occurring occasionally. In those with significant LMS stenosis, STE in either the high right anterior or right ventricular territories was detected using physician's interpretation in 60 (78%) patients. High right anterior or right ventricular territory STE occurred in 65 (84%) of the 77 patients categorised as having AMI and BSPM STE by the physician. Of those with AMI, 9/83 (11%) patients had STEMI by Minnesota criteria, i.e. 74/83 patients were classified as non-STEMI using the initial 12-lead ECG. Of these 74 patients, 60 (81%) had STE in either the high right anterior or right ventricular territories.

Table 3. Univariate analysis of initial BSPM for the diagnosis of AMI

	Patients with significant LMS stenosis (n = 100; 83 with AMI, 17 without AMI)		
BSPM	n	Sens	Spec
Physician's diagnosis	82	93%	71%
PRIME algorithm	80	89%	65%

n = number; Sens = sensitivity; Spec = specificity

4. Discussion

Coronary heart disease remains the leading cause of death in the United States and Europe [3]. The 12-lead ECG is the first-line modality used in the diagnosis of STEMI at the bedside, guiding appropriate therapy and predicting prognosis, yet only 50% of patients with AMI are diagnosed by the initial 12-lead ECG [2]. In LMS stenosis, ischaemia of the basal interventricular septum with an injury vector in the frontal plane pointing in a superior direction towards the right shoulder will result in STE in lead aVR with reciprocal change in the lateral precordial leads (V_4-V_5) [7]. It has been previously suggested that STE in lead aVR > STE in lead V_1 predicts LMS stenosis [5, 12]. In our study, STE aVR ≥0.05mV had low diagnostic sensitivity for AMI (20%). Looking at ST-segment deviation, Mahajan et al [12] showed a trend towards STD ≥0.05mV in leads V_4-V_6, I, II and aVL and that ST-segment deviation in lead V_6 greater than or equal to ST-segment deviation in lead V_1 resulted in a diagnostic sensitivity 81%, specificity 57% and positive predictive value 64% for LMS stenosis (≥50%) in ACS [12]. Typical STEMI on 12-lead ECG has been previously described in the context of acute LMS obstruction [5, 7, 13, 14]. The predominant pattern is anterior territory STE with varying numbers of affected precordial leads usually with accompanying STE in leads I and aVL. In this study, STEMI by Minnesota criteria was only detected in 11% of the study population, having

a sensitivity 11%, specificity 88% for AMI diagnosis (Table 2). Furthermore, in the Global Registry of Acute Coronary Events (GRACE) trial, 771 (43%) patients with a clinical history of ACS and >50% LMS stenosis had STD and 627 (35%) patients had new STE/LBBB on initial 12-lead ECG [15].

BSPM using 80-lead ECG has been shown to be more sensitive for detection of STEMI than 12-lead ECG, while retaining similar specificity, particularly in the high right anterior, posterior and right ventricular territories [3, 8]. The findings of the present study showed that STE was located in either the high right anterior or right ventricular territories in 58% of those with BSPM STE and AMI, whereas reciprocal lateral territory STD in ≥2 contiguous leads on 12-lead ECG was only detected in 41/83 (49%) of those with AMI (Table 2). In comparing the diagnostic performance of the BSPM and the 12-lead ECG univariate analysis, only BSPM STE using both physician's and PRIME algorithm diagnosis had sensitivity > 85%, i.e. 93% and 89% respectively. All 12-lead ECG criteria, i.e. STD, TWI, STE in lead aVR and Minnesota STEMI, performed poorly.

5. Conclusion

Of those with AMI and significant LMS stenosis, only 9/83 (11%) patients were classified as STEMI using 12-lead ECG. Early BSPM in these patients, resulted in the reclassification of 60/74 (81%) patients to STEMI diagnosis by detection of STE beyond the territory of the 12-lead ECG. BSPM has the potential to allow earlier triage and more timely institution of aggressive medical therapy by facilitating emergent revascularisation with the potential to reduce morbidity and improve survival in this high-risk patient group.

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