Journal of the American Heart Association

ORIGINAL RESEARCH

Ischemic ST-Segment Depression Maximal in V1–V4 (Versus V5–V6) of Any Amplitude Is Specific for Occlusion Myocardial Infarction (Versus Nonocclusive Ischemia)

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BACKGROUND: Occlusion myocardial infarctions (OMIs) of the posterolateral walls are commonly missed by ST-segment–elevation myocardial infarction (STEMI) criteria, with >50% of patients with circumflex occlusion not receiving emergent reperfusion and experiencing increased mortality. ST-segment depression maximal in leads V1–V4 (STDmaxV1–4) has been suggested as an indicator of posterior OMI.

METHODS AND RESULTS: We retrospectively reviewed a high-risk population with acute coronary syndrome. OMI was defined from prior studies as a culprit lesion with TIMI (Thrombolysis in Myocardial Infarction) 0 to 2 flow or TIMI 3 flow plus peak troponin T >1.0 ng/mL or troponin I >10 ng/mL. STEMI was defined by the Fourth Universal Definition of Myocardial Infarction. ECGs were interpreted blinded to outcomes. Among 808 patients, there were 265 OMIs, 108 (41%) meeting STEMI criteria. A total of 118 (15%) patients had "suspected ischemic" STDmaxV1–4, of whom 106 (90%) had an acute culprit lesion, 99 (84%) had OMI, and 95 (81%) underwent percutaneous coronary intervention. Suspected ischemic STDmaxV1–4 had 97% specificity and 37% sensitivity for OMI. Of the 99 OMIs detected by STDmaxV1–4, 34% had <1 mm ST-segment depression, and only 47 (47%) had accompanying STEMI criteria, of which 17 (36%) were identified a median 1.00 hour earlier by STDmaxV1–4 than STEMI criteria. Despite similar infarct size, TIMI flow, and coronary interventions, patients with STEMI(–) OMI and STDmaxV1–4 were less likely than STEMI(+) patients to undergo catheterization within 90 minutes (46% versus 68%; P=0.028).

CONCLUSIONS: Among patients with high-risk acute coronary syndrome, the specificity of ischemic STDmaxV1–4 was 97% for OMI and 96% for OMI requiring emergent percutaneous coronary intervention. STEMI criteria missed half of OMIs detected by STDmaxV1–4. Ischemic STDmaxV1–V4 in acute coronary syndrome should be considered OMI until proven otherwise.

Key Words: acute coronary syndromes ■ non–ST-segment–elevation myocardial infarction ■ occlusion myocardial infarction ■ posterior myocardial infarction ■ ST-segment elevation myocardial infarction ■ ST-segment depression ■ subendocardial ischemia

Acute coronary occlusion myocardial infarction (OMI) requires immediate diagnosis and management. Patients with potential symptoms of OMI are immediately evaluated with the ECG. The most widely recognizable and accepted ECG feature of OMI is ST-segment elevation (STE), meeting criteria specified in the Fourth Universal Definition of Myocardial Infarction. However, not all OMIs manifest STE meeting these criteria, or even any STE at all. We have already shown that ECG manifestations of OMI that go beyond the ST-segment–elevation myocardial infarction (STEMI) criteria are more than twice as

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Supplementary Material for this article is available at https://www.ahajournals.org/doi/suppl/10.1161/JAHA.121.022866

For Sources of Funding and Disclosures, see page 12.

JAHA is available at: www.ahajournals.org/journal/jaha

CLINICAL PERSPECTIVE

What Is New?

- In a cohort with high-risk acute coronary syndrome in the emergency department, precordial ischemic ST-segment depression maximal in V1–V4 (rather than V5–V6) had 96% specificity for occlusion myocardial infarction (OMI) that underwent percutaneous coronary intervention.

What Are the Clinical Implications?

- ST-segment–elevation myocardial infarction criteria missed half of OMIs detected by ST-segment depression maximal in leads V1–V4, and patients with non–ST-segment–elevation myocardial infarction with delayed management of OMI are known to experience increased mortality compared with non–ST-segment–elevation myocardial infarction without OMI.
- These data support that ischemic ST-segment depression maximal in V1–V4 in acute coronary syndrome is concerning for posterior OMI until proven otherwise and should prompt consideration of emergent reperfusion therapy even in the absence of ST-segment–elevation myocardial infarction criteria.

Nonstandard Abbreviations and Acronyms

OMI	Occlusion Myocardial Infarction
STD	ST-segment depression
STDmaxV1–4	ST-segment depression maximal in leads V1–V4
STDmaxV5–6	ST-segment depression maximal in leads V5–V6

sensitive, and more accurate, for the ECG diagnosis of OMI than are STEMI criteria.[1] Among the many OMIs not captured by the STEMI criteria is OMI of the "posterior" wall, which accounts for ≈10% of all OMIs.[2-6] The posterior wall has been reclassified as part of the lateral wall,[7] but we continue to refer to the portion of the lateral myocardium that does not face any overlying leads of the standard 12-lead ECG (and thus cannot manifest STE when that wall has subepicardial/transmural ischemia) as the "posterior" wall. Such subepicardial ischemia of the posterior wall can only manifest ST-segment depression (STD) on the standard 12-lead ECG, without any STE elsewhere, and in such a case it may be referred to as "isolated" posterior OMI, and may be attributable to acute occlusion of a variety of posterior branches of either the right coronary or left circumflex artery. OMIs of the posterior and lateral walls are the most commonly missed OMIs, with >50% of circumflex occlusions not receiving emergent reperfusion, partly because isolated posterior OMIs are not identified by STE, and partly because, when there is any STE, its voltage does not meet STEMI criteria.

When balloon occlusion of coronary arteries results in transmural ischemia proven by intracoronary STE, surface electrodes register STE during circumflex occlusions in only 32%, compared with 84% and 92% for left anterior descending artery (LAD) and right coronary artery occlusions, respectively.[8] Schmitt et al showed that only 46% of patients with complete left circumflex artery (LCX) occlusion manifested diagnostic STE on the standard 12 leads, and this increased by about 10% with addition of extra leads.[8,9] From et al[10] and Huey et al[11] observed 45% and 48% sensitivity, respectively, of diagnostic STE criteria for circumflex occlusion. Despite the fact that LCXs are less likely to manifest diagnostic STE and the most likely to be missed by STEMI criteria, they have no difference in severity of myocardial infarction (MI), prognosis, or benefit from emergent reperfusion compared with other large-vessel coronary occlusions.[3,11,12] Circumflex occlusions without STE have the same amount of myocardium at risk as those *with* STE, and the same amount of salvage with reperfusion therapy.[3] Pride et al[13] performed a substudy of TRITON-TIMI-38 (Trial to Assess Improvement in Therapeutic Outcomes by Optimizing Platelet Inhibition with Prasugrel-Thrombolysis in Myocardial Infarction) trial, including patients with "isolated precordial ST depression" and a nonurgent angiogram (median time, 29 hours), showing that approximately one-third of such patients with acute MI (AMI) had acute coronary occlusion. These patients with missed occlusions had worse outcomes than patients with STD without occlusion. Because >30% of occluded arteries will spontaneously reperfuse (autolyze) within 24 hours, many of those with open arteries would have been occluded at the time of ECG recording, so this study supports that approximately two-thirds of patients with STD in V1–V4 have acute occlusion.

Baseline ECGs with a normal QRS have significant baseline (nonischemic) STE in V2 and V3, and in most men, the STE is >1 mm[14,15]; hence, the Universal Definition of Myocardial Infarction considers STE in V2 and V3 to be normal (<1.5 mm for women, <2.5 mm for men aged <40 years, and <2.0 mm for men aged >40 years).[16] For this reason, any ST-segment depression, even <0.1 mV (1 mm) in V1–V4 is abnormal and, in the right clinical situation, is suspicious for posterior OMI. Nevertheless, previous studies that resulted in poor sensitivity of STD for posterior MI required 1 mm in 2 consecutive leads.[17]

The STD of posterior OMI is reciprocal to an STE vector of subepicardial (transmural) ischemia directed posteriorly, and thus manifesting as STD in precordial

leads. However, precordial STD may be attributable to either OMI or subendocardial ischemia. Evidence suggests that the STD of posterior OMI is maximal in V1–V4, whereas the STD of subendocardial ischemia is maximal in V5 and V6.[18,19]

The Fourth Universal Definition of Myocardial Infarction[16] recommends ≥0.5 mm STE in leads V7–V9 as an extension of the STEMI criteria; however, it states that isolated STD in V1–V3 "may indicate left circumflex occlusion" but is "nonspecific." This is contrary to angiographic balloon occlusion studies, in which it is specific. We are unaware of any study showing that STD maximal in V1–V4 is common in nonocclusive acute coronary syndrome (ACS); rather, nonocclusive ACS is more likely to have ST-segment depression maximal in leads V5–V6 (STDmaxV5–6).[18]

The 2013 American College of Cardiology/American Heart Association STEMI Guidelines[20] give no formal recommendations for posterior STEMI as a STEMI equivalent (neither using STD in V1–V4 nor STE in V7–V9), and simply state "ST depression in two precordial leads (V1–V4) may indicate transmural posterior injury." Although posterior ECG criteria are specifically avoided as part of the STEMI criteria in their recommendation for administering thrombolytics, they also give a class III harm recommendation, stating "Fibrinolytic therapy should not be administered to patients with ST depression except when a true posterior (inferobasal) MI is suspected."

Finally, the National Cardiovascular Data Registry guidelines[21] are the only guidelines of which we are aware that formally recommend isolated ST depression as a STEMI equivalent, stating "ST elevation in the posterior chest leads (V7 through V9), or ST depression that is maximal in V1–V3, without ST segment elevation in other leads, demonstrating posterobasal myocardial infarction, is considered a STEMI equivalent and qualifies the patient for reperfusion therapy."

Given the importance of missed OMI and the conflicting guideline recommendations with a paucity of evidence, we sought to further evaluate the diagnostic accuracy of ST-segment depression maximal in leads V1–V4 (STDmaxV1–4) for the identification of OMI.

METHODS

Study Design and Setting

This was a planned substudy of the Diagnosis of Occlusion MI and Reperfusion by Interpretation of the Electrocardiogram in Acute Thrombotic Occlusion database, which is a 2-site collaboration designed to study ECG findings in OMI. Stony Brook University Hospital is a suburban, academic hospital and a regional cardiac catheterization referral center. Hennepin Healthcare Medical Center is an urban academic hospital with a cardiac catheterization laboratory. Both emergency departments (EDs) have >100 000 visits per year. Institutional review board approval was obtained at both sites, and there was no extramural funding. Informed consent was waived by both institutional review boards for this retrospective study.

Selection of Participants

First, each site accessed the cardiac catheterization laboratory activation database (all urgent and emergent left heart catheterizations during 1 year), which provided both cases (OMI) and controls (without OMI). At Stony Brook University Hospital, we added a previously collected prospective cohort of ED patients who were admitted to the cardiology service with suspected ACS during a 6-month time period (again contributing both cases and controls). To ensure that the final cohort also contains a substantial number of control patients with abnormal ECGs, we added additional controls from Hennepin Healthcare Medical Center by searching the Use of TROPonin In Acute coronary syndromes (UTROPIA) database[22] for patients without OMI but with STE, STD, or T-wave inversion, one-third of whom had adjudicated nonocclusion MI. Patients were excluded if there were no ECGs in the electronic medical record or if there was insufficient retrospective information available to determine the primary outcome (the presence or absence of our OMI definition).

Data Collection and Measurements

Chart review was performed by 4 emergency medicine residents after training with a standardized data coding manual. Primary and senior authors (H.P.M. and S.W.S.) were available for on-demand questions and retraining as necessary. Demographics, clinical and laboratory results, serial ECGs, and angiographic findings were collected using Research and Electronic Data Capture. All available transfer, prehospital, and study site ECGs were collected for each patient.

ECG interpretation included various predefined ECG findings, objective measurements, and subjective interpretations, and was performed by S.W.S. and H.P.M. blinded to all patient information, except age and sex (necessary for interpretation of STEMI criteria, which are age and sex based). S.W.S. interpretations were used for all analyses of OMI versus not OMI and evaluations of STE criteria. Serial ECGs were interpreted sequentially, and initially blinded to the baseline ECG (when available from a previous visit), then unblinded to that baseline ECG for a separate interpretation. The interpreter could not go back and change an interpretation once entered, just as a clinician must interpret each ECG as it is chronologically recorded in real time. STEMI criteria were defined according to the fourth universal definition of

MI, and thus measured in millimeters using the QRS onset (PQ junction) and the J-point. If any ECG before the angiogram met STEMI criteria, the patient was considered to be STEMI(+); if not, then the patient was considered STEMI(–). Interobserver variation to the nearest 0.5 mm has previously been established in our author group.[23] We assessed interobserver reliability for OMI between H.P.M. and S.W.S. for all cases interpreted by both. Furthermore, all 108 consecutive OMI cases from the prospective cohort were reviewed for STEMI criteria by a cardiologist (J.A.K.) blinded to the outcome and the study goals. Specific ECG measurements and observations included STE meeting STEMI criteria, subtle STE not meeting criteria, hyperacute T waves (including de Winter pattern), reciprocal STD and/or negative hyperacute T waves, STD maximal in V1–V4 indicative of posterior OMI, suspected acute pathologic Q waves (Q waves associated with subtle STE, which cannot be attributed exclusively to old MI), terminal QRS distortion,[24] any STE in inferior leads with any STD or T-wave inversion in lead aVL, and positive modified Sgarbossa criteria for patients with left bundle-branch block or ventricular paced rhythm.

ST-Segment Depression Maximal in Leads V1–V4

Furthermore, H.P.M. also reviewed all ECGs from all patients, also blinded to outcome, to specifically evaluate for the presence of any STDmaxV1–4, STDmaxV5–6, or specific lead where STD was maximal, and to note any secondary, or nonischemic, cause of the STDmaxV1–4 or STDmaxV5–6, if applicable (eg, "secondary," or "nonischemic," refers to STD that is secondary to an abnormal QRS, such as right bundle-branch block [RBBB] or paced rhythm). "Any STDmax V1–V4" refers to any STD of any magnitude that is maximal in V1–V4 in any context (even if clearly secondary to the QRS complex, such as in RBBB). STDmaxV1–4 that could not be explained by a nonischemic cause was classified as primary STD, not secondary to an identifiable nonischemic source, and referred to as "suspected ischemic STDmaxV1–4." There was no minimal magnitude of STD required for our interpreters to diagnose STD (ie, even if the deepest STD was <0.5 mm, for example, the interpreter reported STD). When it was uncertain whether maximal STD was in V4 or V5, the lead with greatest STD in proportion to the QRS complex was chosen.

Outcomes

The diagnosis of OMI was adjudicated by structured chart review. In patients who were determined to not have OMI, the diagnosis of AMI was the following: at Stony Brook University Hospital, it was determined by the final diagnosis on the patient's record; and at Hennepin Healthcare Medical Center, it was determined by strict adjudication of all clinical data, performed by 3 adjudicators as part of the UTROPIA study. Outcomes used to ascertain the presence of OMI on the ECG cannot be based solely on TIMI flow of the lesion at the time of the angiogram because the state of the artery frequently differs between the time of the ECG and the time of the angiogram. Proven STEMI has an open artery in 19% to 36% of cases, depending on whether it is TIMI –1, –2, or –3 flow. Karwowski et al showed that only 64% of 4581 STEMIs had TIMI 0 flow on angiogram.[25] Stone et al found that 72% have TIMI 0 or 1 flow.[26] Finally, Cox et al found that 80% had TIMI 0, 1, or 2.[27] Thus, ≈20% of true STEMIs have TIMI 3 flow at immediate angiogram. As such, the definition of OMI was reproduced from prior studies,[23,28,29,30,31,32] composed of either (1) "confirmed OMI" on cardiac catheterization (defined as an acute culprit lesion with TIMI 0–2 flow) or (2) "presumed OMI with significant cardiac outcome," defined as any of the following: (a) acute but nonocclusive (TIMI >2) culprit lesion with highly elevated cardiac troponin (contemporary cardiac troponin T ≥1.0 ng/mL [Roche Diagnostics Elecsys; reference range, ≤0.01 ng/mL] or contemporary cardiac troponin I ≥10.0 ng/mL [Abbott Architect 4th generation; reference range, ≤0.030 ng/mL]); (b) if no angiography, then highly elevated cardiac troponin and a new or presumed new regional wall motion abnormality on echocardiography; or (c) ECG positive for STEMI with death before attempted emergent catheterization. Despite the fact that OMI cannot be based solely on TIMI 0 to 1 flow of the culprit lesion for the reasons explained above, we also presented TIMI 0 to 1 culprit lesions as a dedicated outcome. Formal adjudication was made with all available data, including ECGs, cardiac troponins, echocardiograms, and angiogram results. If TIMI flow was not reported, the cine angiogram was reviewed by a cardiologist (G.R.S.) with experience interpreting angiograms. The definition of "highly elevated" cardiac troponin was chosen previously as the most accurate cutoff for differentiating STEMIs from non–ST-segment–elevation myocardial infarctions using various cardiac troponin assays,[33-37] and has subsequently been internally and externally validated.[23,28,30,32,38,39]

Statistical Analysis

We calculated summary statistics for cases and controls, and diagnostic utility statistics (sensitivity, specificity, and accuracy) for each ECG finding. Interobserver agreement was calculated using κ values for categorical variables. Subject characteristics and outcomes were compared between groups using Mann-Whitney *U* or Kruskal–Wallis tests for continuous measurements and Pearson χ^2 or Fisher exact test

for categorical measures. All tests were 2 sided, and statistical significance was accepted at the 0.05 level. Descriptive statistics were performed in Research and Electronic Data Capture, whereas other statistical tests were performed with Microsoft Excel (Version 1905; Redmond, WA).

The primary analysis was the specificity of suspected ischemic STDmaxV1–4 for OMI. Secondary goals included the specificity for OMI of any STDmaxV1–4 (whether classified as "suspected ischemic" or "nonischemic") morphologic characteristics of posterior OMI (QRS, ST-segment, and T-wave morphologic characteristics), comparison of patients with STDmaxV1–4 versus those with STDmaxV4–6, and comparison of patients with STDmaxV1–4 with concomitant STEMI criteria versus those without STEMI criteria.

RESULTS

Our database included 808 patients with a total of 3421 ECGs. Among 808 patients, there were 396 with acute MI as a final diagnosis. A total of 265 patients met our primary outcome criteria for OMI, with only 108 (41%) meeting STEMI criteria. The cardiologist (J.A.K.) who reviewed 108 consecutive OMI cases from Stony Brook University Hospital classified fewer cases as STEMI(+) (59 versus 67, or 55% versus 62%) compared with either of our primary interpreters. Population characteristics are shown in Table 1.

Table 1. Patient Characteristics for All Patients and Patients With OMI Specifically

Characteristics	All patients (N=808)	All patients with OMI (n=265)
Age, mean (SD), y	62 (14)	63 (13)
Women	265 (33)	62 (23)
Black	152 (19)	30 (11)
White	576 (71)	205 (77)
Hispanic	51 (6)	18 (7)
Known CAD	318 (39)	182 (69)
CKD	85 (11)	27 (10)
CHF	121 (15)	21 (8)
Diabetes	275 (34)	82 (31)
Hyperlipidemia	450 (56)	154 (58)
Hypertension	565 (70)	183 (69)
Obesity	388 (48)	123 (46)
Tobacco use	483 (60)	161 (61)
Family history of CAD	313 (39)	118 (45)
Presence of chest pain	638 (79)	230 (87)
Presented in arrest	12 (1.8)	11 (5.1)
Catheterization laboratory activated in ED	218 (27)	176 (66)
Precatheterization cardiac arrest	41 (5)	30 (11.3)
Acute MI on final discharge	396 (49)	265 (100)
Cardiac catheterization performed	635 (79)	261 (99)

Data are given as number (percentage), unless otherwise indicated. CAD indicates coronary artery disease; CHF, chronic heart failure; CKD, chronic kidney disease; ED, emergency department; MI, myocardial infarction; and OMI, occlusion MI.

Interrater Reliability

For the 250 Hennepin Healthcare Medical Center patients interpreted by both S.W.S. and H.P.M., there was 97.2% agreement for the determination of STEMI criteria (κ=0.893), and 94% agreement for the classification of OMI (κ=0.893). Interobserver variation to the nearest 0.5 mm, as well as ST-T wave morphologic agreement, has been previously established within our author group, including specifically between H.P.M. and S.W.S.[23,28,40,41,42]

All Categories of Patients with STDmaxV1–4

A total of 147 (18%) patients had any STDmaxV1–4, of whom 29 were (blindly, by ECG only) classified as "nonischemic" (either identical to available baseline ECG or explained by non-OMI diagnosis). Table 2 shows the clinical outcomes of this group as well as the following subgroups based on ECG interpretation.

Any STDmaxV1–4 (Including Both Nonischemic and Suspected Ischemic)

A total of 147 (18%) patients had STDmaxV1–4, of whom 135 (92%) underwent angiogram, 116 (79%) had an acute culprit lesion, 102 (69%) met our definition of OMI, 101 (68%) underwent percutaneous coronary intervention (PCI), and 70 (48%) had TIMI 0 or 1 culprit flow before intervention. The specificity, sensitivity, and positive likelihood ratio of any STDmaxV1–4 for OMI, regardless of classification (nonischemic or suspected ischemic), was 91.7%, 38.5%, and 4.64 (see all contingency tables in the online appendix in Table S1).

STDmaxV1–4 Classified as Nonischemic Because of Suspected Non-OMI Diagnosis

Of the 147 patients with STDmaxV1–4, 29 were classified as "nonischemic," either baseline or explained by non-OMI diagnosis (17 RBBB, 2 ventricular paced rhythm, 3 nonspecific interventricular conduction delay, 2 hypokalemia [both verified true], 1 juvenile T-wave pattern, 1 left ventricular [LV] aneurysm pattern, and 3 uncategorized but proven identical to prior available baseline ECG). Of these 29 patients with explanations for STDmaxV1–4, 3 had OMI (in other words,

Table 2. Clinical Outcomes for Each Group of STDmaxV1–4

Variable	Any STDmaxV1–4, including "secondary" STD	Suspected "primary" ischemic STDmaxV1–4	Any STDmaxV1–4 and subjective interpretation of OMI
Total, N	147	118	112
Angiogram	135 (92)	113 (96)	110 (98)
Acute culprit	116 (79)	106 (90)	104 (93)
OMI	102 (69)	99 (84)	99 (88)
TIMI 0/1	70 (48)	68 (58)	68 (61)
PCI	101 (68)	95 (81)	95 (85)
Specificity of ECG finding for OMI requiring PCI, %	91.5	96.0	96.9
Specificity of ECG finding for OMI, %	91.7	96.5	97.6
Sensitivity of ECG finding for OMI, %	38.5	37.4	37.4
LR+ of ECG finding for OMI	4.64	10.67	15.60
OR of ECG finding for OMI	6.93	16.45	24.31
Triple-vessel or left main ACS	15 (10)	12 (10)	12 (11)
NOMI	26 (18)	16 (14)	12 (11)
No AMI	19 (13)	3 (1)	1 (1)
Echocardiogram	130 (88)	111 (94)	107 (96)
Wall motion abnormality, n/total (%)	98/130 (75)	90/111 (81)	87/108 (81)

Data are given as number (percentage), unless otherwise indicated. ACS indicates acute coronary syndrome; AMI, acute myocardial infarction; LR+, positive likelihood ratio; NOMI, nonocclusion myocardial infarction; OMI, occlusion myocardial infarction; OR, odds ratio; PCI, percutaneous coronary intervention; STD, ST-segment depression; STDmaxV1–4, STD maximal in leads V1–V4; and TIMI, Thrombolysis in Myocardial Infarction.

the "nonischemic" diagnosis falsely reassured against OMI).

The first case involved a patient with normal QRS morphologic features who experienced LCX OMI simultaneously with severe hypokalemia (K=2.6 mEq/L), whose ECG was interpreted (without any clinical context) by both H.P.M. and S.W.S. as hypokalemia mimicking posterior OMI.

The other 2 cases were patients with RBBB who experienced LCX and D1 OMIs, respectively, whose ECGs were interpreted by both interpreters as RBBB with STDmaxV1–4 suspected to be the normal (appropriately discordant) STDmaxV1–4 of RBBB. Only in retrospect was it recognized that these patients actually have STDmaxV1–4 that is excessively discordant to the positive RBBB R'-wave, deeper than would be expected for RBBB alone.

Suspected Ischemic STDmaxV1–4

A total of 118 (15%) patients had "suspected ischemic" STDmaxV1–4 without an accompanying ECG feature, such as RBBB, that would explain the STDmaxV1–4 as preexisting. Of these 118 patients, 113 (96%) underwent angiogram, 106 (90%) had an acute culprit lesion, 99 (84%) met our definition of OMI, 95 (81%) underwent PCI, and 68 (58%) had TIMI 0 or 1 culprit flow before intervention. The specificity, sensitivity, and positive likelihood ratio for the diagnosis of OMI of suspected ischemic STDmaxV1–4 were 96.5%, 37.4%, and 10.67, and for OMI requiring PCI the specificity, sensitivity, and positive likelihood ratio were 96.0%, 39.7%, and 9.94.

STDmaxV1–4 and Subjective Interpretation of OMI

Of the 118 with suspected ischemic STDmaxV1–4, 112 (95%) were interpreted as OMI, of whom 110 (98%) underwent angiogram, 104 (93%) had an acute culprit lesion, 99 (88%) met our definition of OMI, 95 (85%) underwent PCI, and 68 (61%) had TIMI 0 or 1 culprit flow before intervention.

Of the 118 patients, 6 were not called OMI by the interpreter, with reasons including the following: 2 cases (1 with a culprit lesion in the left posterolateral artery): there was no other lead with even subtle evidence of OMI; 1 case with no culprit: no agreement on presence of any STD; 1 case with an LCX culprit: opinion that STD was maximal in V5–V6; 1 case with no culprit: associated T-wave morphologic feature that led the reader to interpret the STD as chronic; and 1 case with no culprit: atrial fibrillation with rapid ventricular response. The specificity, sensitivity, and positive likelihood ratio for OMI of STDmaxV1–4 interpreted as diagnostic of OMI were 97.6%, 37.4%, and 15.60, respectively.

Of the 112 patients interpreted as OMI, 12 (11%) had triple-vessel disease or left main ACS, 11 of which were also OMI. Of these 12 patients, 10 were diagnosed with AMI, with the other 2 patients having a slight troponin increase noted during the first few serial troponins (0.00–0.02 ng/mL in one case, and 0.00–0.03 ng/mL in the other case), with no troponins further measured and no formal diagnosis of AMI recorded at discharge. A total of 9 (8%) received coronary artery bypass grafting. A total

of 107 of 112 underwent formal echocardiogram; 87 (81%) had a wall motion abnormality, of which 84 (96%) were located in the inferior, posterior, and/or lateral walls.

Only 6 of the 112 had no OMI, no culprit, and no triple-vessel or left main disease. A total of 5 of these 6 were diagnosed with acute MI, and the remaining patient had normal coronaries on emergent catheterization, without AMI. Thus, only 1 patient of 112 diagnosed with OMI by ECG did not have AMI.

There were 8 patients with STDmaxV1–4 who had posterior leads performed and objectively recorded in the electronic medical record.

A Total of 99 OMIs Detected by Suspected Ischemic STDmaxV1–4

Of the 99 OMIs detected by STDmaxV1–4, 47 (47%) had accompanying STEMI criteria in other locations. Culprit lesions included the right coronary artery (53), left circumflex artery (32), posterior descending artery (7), smaller branches capable of supplying the posterior wall, such as first obtuse marginal, second obtuse marginal, ramus intermedius, and left posterolateral (30), and other vessels (10).

"False Positives": Patients With Suspected Ischemic STDmaxV1–4 But Without OMI

A total of 13 patients had STDmaxV1–4 but did not meet the definition of OMI ("false positives"); all 13 underwent angiogram, 7 had culprit lesions (2 proximal LAD, 1 mid-LAD, 1 LCX, 1 middle right coronary artery, 2 second obtuse marginal, and 1 third diagonal), 6 required PCI, and 1 patient died during index visit.

ECG Findings in Patients With Suspected Ischemic STDmaxV1–4 without concomitant STEMI

Table 3 shows the ECG characteristics of various groups. A total of 52 patients had STDmaxV1–4 without concomitant STEMI criteria, but only 12 of 112 patients (11%) had STDmaxV1–4 without any other subtle signs of OMI, especially hyperacute T waves and subtle STE, in other leads; all 12 of these had at least 1 mm of STDmaxV1–4. A total of 22 patients in the STEMI(–) OMI group had STDmaxV1–4 of <1 mm, but all of these had subtle findings of OMI in other locations. In other words, although the findings may be extremely subtle, they are supported by findings in other leads. In summary, all patients with posterior OMI had either (1) at least 1 mm STDmaxV1–4 or (2) other subtle findings of OMI in addition to any STDmaxV1–4 (even if <1 mm).

Patients With STEMI(+) OMI With STDmaxV1–4 Versus Patients With STEMI(–) OMI With STDmaxV1–4

Of the 99 patients with OMI with suspected ischemic STDmaxV1–4, 47 had concomitant STEMI criteria (STEMI[+] OMI), and the remaining 52 (20% of all 265 OMIs) did not (STEMI[–] OMI); these patients with OMI

Table 3. Specific ECG Characteristics, Including T-Wave and ST-Segment Morphologic Characteristics, of Various Groups of Patients With STDmaxV1–4

Variable	118 Patients with suspected ischemic STDmaxV1–4	99 Patients with suspected ischemic STDmaxV1–4 with OMI	52 Patients with suspected ischemic STDmaxV1–4 with STEMI(–) OMI
Most diagnostic lead	V1: 1 (1)	V1: 1 (1)	V1: 1 (2)
	V2: 63 (53)	V2: 60 (61)	V2: 24 (46)
	V3: 32 (27)	V3: 22 (22)	V3: 14 (27)
	V4: 22 (19)	V4: 16 (16)	V4: 13 (25)
Maximal STD magnitude	STD <1 mm: 45 (38)	STD <1 mm: 34 (34)	STD <1 mm: 22 (42)
	STD ≥1 mm: 73 (62)	STD ≥1 mm: 65 (66)	STD ≥1 mm: 30 (58)
ST-segment morphologic characteristics	Horizontal: 43 (36)	Horizontal: 33 (33)	Horizontal: 16 (31)
	Down-sloping: 46 (39)	Down-sloping: 40 (40)	Down-sloping: 19 (36)
	Up-sloping: 29 (25)	Up-sloping: 26 (26)	Up-sloping: 17 (33)
T-wave morphologic characteristics	Upright: 69 (59)	Upright: 59 (60)	Upright: 35 (67)
	Inverted: 22 (19)	Inverted: 14 (14)	Inverted: 5 (10)
	Biphasic down-up: 27 (23)	Biphasic down-up: 26 (26)	Biphasic down-up: 12 (23)
	Biphasic up-down: 0 (0)	Biphasic up-down: 0 (0)	Biphasic up-down: 0 (0)
R wave amplitude / S wave amplitude >1 in V2 (R/S >1)	R/S >1: 18 (15)	R/S >1: 16 (16)	R/S >1: 7 (14)
	R/S ≤1: 100 (85)	R/S ≤1: 83 (84)	R/S ≤1: 45 (86)

Data are given as number (percentage). OMI indicates occlusion myocardial infarction; STD, ST-segment depression; STDmaxV1–4, STD maximal in leads V1–V4; and STEMI, ST-segment–elevation myocardial infarction.

Table 4. Angiographic Outcomes, Timing, and Peak Troponins Between STEMI(+) and STEMI(–) OMI With STDmaxV1–4

Variable	STEMI(+) OMI with STDmaxV1–4 (posterior OMI with concomitant STEMI of another wall)	STEMI(–) OMI with STDmaxV1–4 (isolated posterior OMI)	Nonocclusion MI*	*P* value comparing STEMI(+) OMI with STEMI(–) OMI
Total, N	47	52	216	Not Applicable
TIMI 0/1 flow, n (%)	32 (68)	35 (67)	0 (0)	0.94
PCI performed, n (%)	45 (96)	44 (85)	62 (29)	0.100
Peak troponin T, median (IQR), ng/mL	3.68 (2.06–5.52)	2.82 (1.02–4.34)	0.13 (0.03–0.34)	0.072
Time from presentation to angiogram, median (IQR), min	41 (22–119)	92.5 (33–854)	1340 (279–3465)	0.013
Angiogram within 90 min, n (%)	32 (68)	24 (46)	24 (11)	0.028

IQR indicates interquartile range; MI, myocardial infarction; OMI, occlusion MI; PCI, percutaneous coronary intervention; STDmaxV1–4, ST-segment depression maximal in leads V1–V4; STEMI, ST-segment–elevation MI; and TIMI, Thrombolysis in Myocardial Infarction.
*Nonocclusion MI is presented for comparison.

were completely missed by STEMI criteria but correctly identified by suspected ischemic STDmaxV1–4. Table 4 shows the differences in interventions, peak troponins, and time to catheterization between the 2 categories.

Between the STEMI(+) and STEMI(–) groups, there was no statistical difference between the rates of TIMI 0 or 1 culprit lesion flow, the rates of requiring PCI, or the median peak troponin; the only statistically significant differences found were that the STEMI(–) group had longer delays to catheterization and lower likelihood of receiving catheterization within 90 minutes. Four example patients with OMI diagnosed by suspected ischemic STDmaxV1–4 are shown in Figures 1 through 3, as well as Figure S1 (supplemental online appendix).

Of the 47 patients with OMI who had STDmaxV1–4 with concomitant STEMI criteria, 30 had both ECG findings simultaneously on the first ECG at arrival. The remaining 17 patients all had STDmaxV1–4 noted earlier than STEMI criteria, with a median (interquartile range) delay of 1.00 (0.42–5.72) hours until STEMI criteria developed on a subsequent serial ECG. Thus, in 69 of 99 patients with OMI (70%), the first or only evidence of OMI was any STDmaxV1–4.

Suspected Ischemic STD Borderline/ Equal Between V4 and V5

Although the determination of STDmaxV1–4 versus STDmaxV5–6 was dichotomized for all other analyses presented, there were 7 patients with STD for which the interpreter noted that STD was basically equal in leads V4 and V5, making this distinction especially difficult in this small subset. All 7 underwent angiography, 5 had culprit lesions (all LCX), 4 had OMI, and 3 received PCI.

STD Maximal in V5–V6

A total of 196 (24.3% of 808) had any objective STDmaxV5–6 (whether classified as ischemic or nonischemic). The interpreter labeled these cases as ischemic (65 [33%]), ischemic and indicative of LAD occlusion pattern (32 [16%]), LV hypertrophy (33 [17%]), ventricular paced rhythm (4 [2%]), LV aneurysm morphologic feature (3 [1.5%]), left bundle-branch block (32 [16%]), RBBB (3 [1.5%]), nonspecific and mild (19 [9.7%]), and other (5 [2.6%]).

A total of 48 of 196 were identified as OMI by expert interpretation, with 22 meeting STEMI criteria in other leads; thus, there were 174 STDmaxV5–6 without STEMI, and 148 STDmaxV5–6 without any signs of OMI elsewhere on the ECG. Of these 148 patients with STDmaxV5–6 and no signs of OMI, 58 were classified as "ischemic" STD and 90 were classified as "secondary" STD (attributed to LV hypertrophy, left bundle-branch block, or ventricular paced rhythm). Outcomes of each subset are shown in Table 5. Figure S2 (supplemental appendix) shows the quintessential case of diffuse ischemic STD with STDmaxV5–6, with reciprocal STE in aVR, representing ischemia not meeting our definition of OMI in the setting of known severe 3-vessel disease and prior coronary artery bypass grafting.

Limitations

Because OMI comprises only ≈2% to 5% of all ED patients with potential ACS, we had insufficient resources to perform a prospective, consecutive cohort study, and instead we performed a retrospective case-control study to maximize both the number of patients with OMI and patients with non-OMI with abnormal ECGs. As a result, our population is a high-risk cohort with ACS, and findings may not apply to undifferentiated ED patients without ACS strongly suspected clinically.

Our study is retrospective and performed at only 2 centers. Few (only 8) of our patients with STDmaxV1–4 had posterior leads performed and recorded in the electronic medical record. Therefore, we were unable to assess the accuracy of posterior lead criteria in

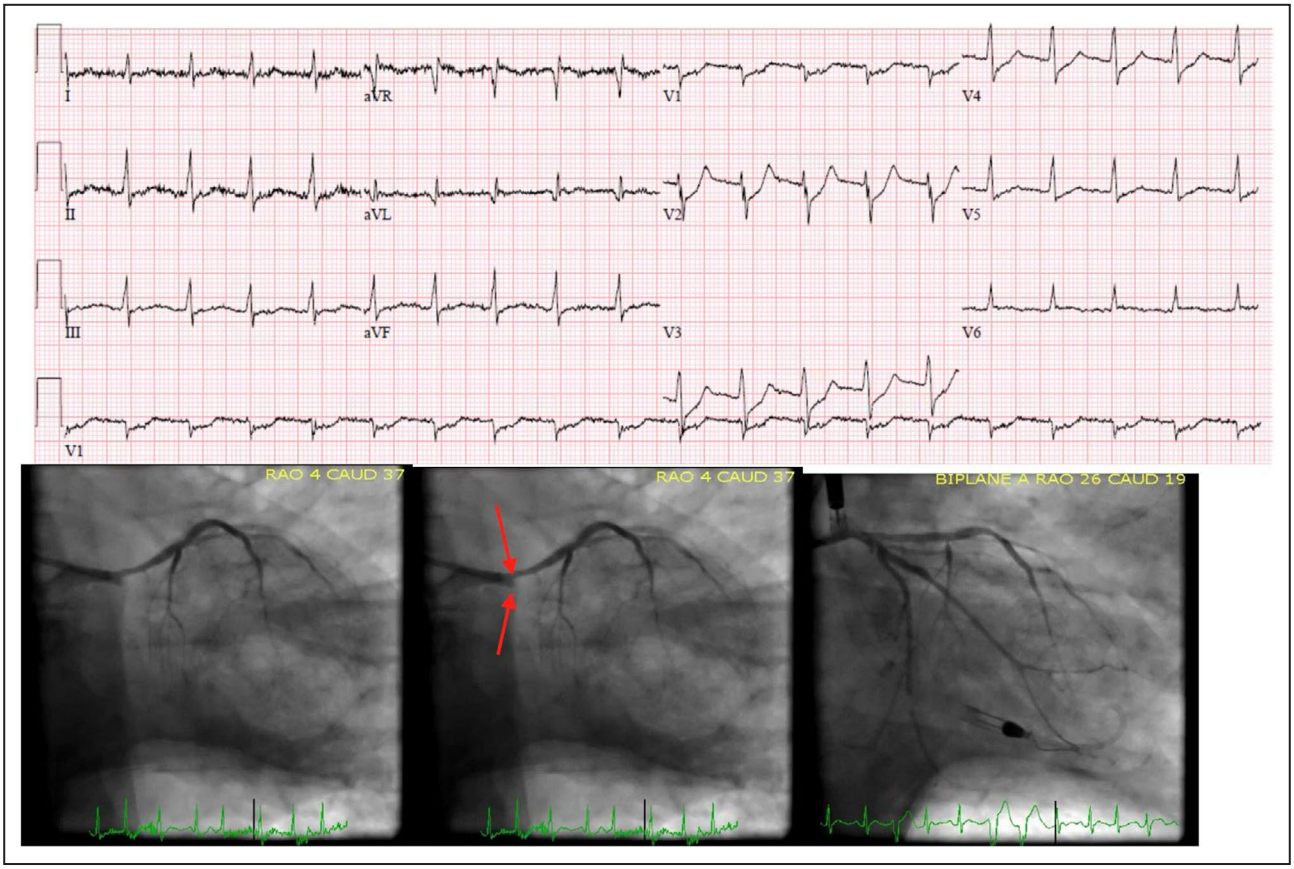

Figure 1. A 58-year-old man presented with 2 hours of constant chest pressure, shortness of breath, and palpitations that started while walking his dog.
The ECG was interpreted as having ST-segment depression maximal in leads V1–V4, and the catheterization laboratory was activated immediately based on this ECG. There is also subtle ST-segment elevation in V6. The ECG was deemed not to meet ST-segment–elevation myocardial infarction criteria, and the catheterization laboratory activation was canceled. Serial ECGs remained unchanged. The patient then experienced a 2-hour delay until angiogram, where a complete occlusion (TIMI [Thrombolysis in Myocardial Infarction] 0) of the ostial left circumflex was found and stented (angiograms shown above). Troponin T was 32.09 ng/mL and increasing but not trended to peak. Despite effective angiographic reperfusion, the patient progressed to cardiogenic shock and died on day 7 of hospitalization.

conjunction with STDmaxV1–4. Although this is a limitation, it is somewhat telling that, in our study of 808 patients with 3241 ECGs, it was extremely rare in actual clinical practice to record and document posterior leads, even in patients with anterior STD. This indicates that further education on posterior leads is needed, or standard 12-lead criteria designed to identify posterior OMI are needed. Our data show that STDmaxV1–4 is an accurate marker of OMI without obtaining posterior leads. More important, there are little data supporting that, when there is ischemic STD in V1–V4, STE in posterior leads helps to differentiate posterior OMI from subendocardial ischemia.[43]

As are all ECG findings when interpreted by humans, the identification of proportionally maximal STD in leads V1–V4 is subjective, requiring dedicated training and experience to accurately identify. This is an important limitation to external validity. However, the current STEMI criteria are also highly subjective, with notoriously poor interrater reliability,[44-47] and never met any external validity standards before becoming the universal approach to ECG ischemia interpretation. The relatively high interrater reliability between our 2 interpreters shows that these skills can be taught and learned with a high level of agreement.

DISCUSSION

In contrast to Pride et al,[13] we found any STDmaxV1–4 to be 92% specific for OMI among ED patients with ACS (increased to 97% for suspected ischemic STDmaxV1–V4). Even in the group of 13 patients with STDmaxV1–4 who did not meet our definition of OMI ("false positives," according to the outcome definition), all required angiogram and half underwent PCI. Several differences between our study and Pride et al could explain the differences in results. First, and most important, the median time from presentation to

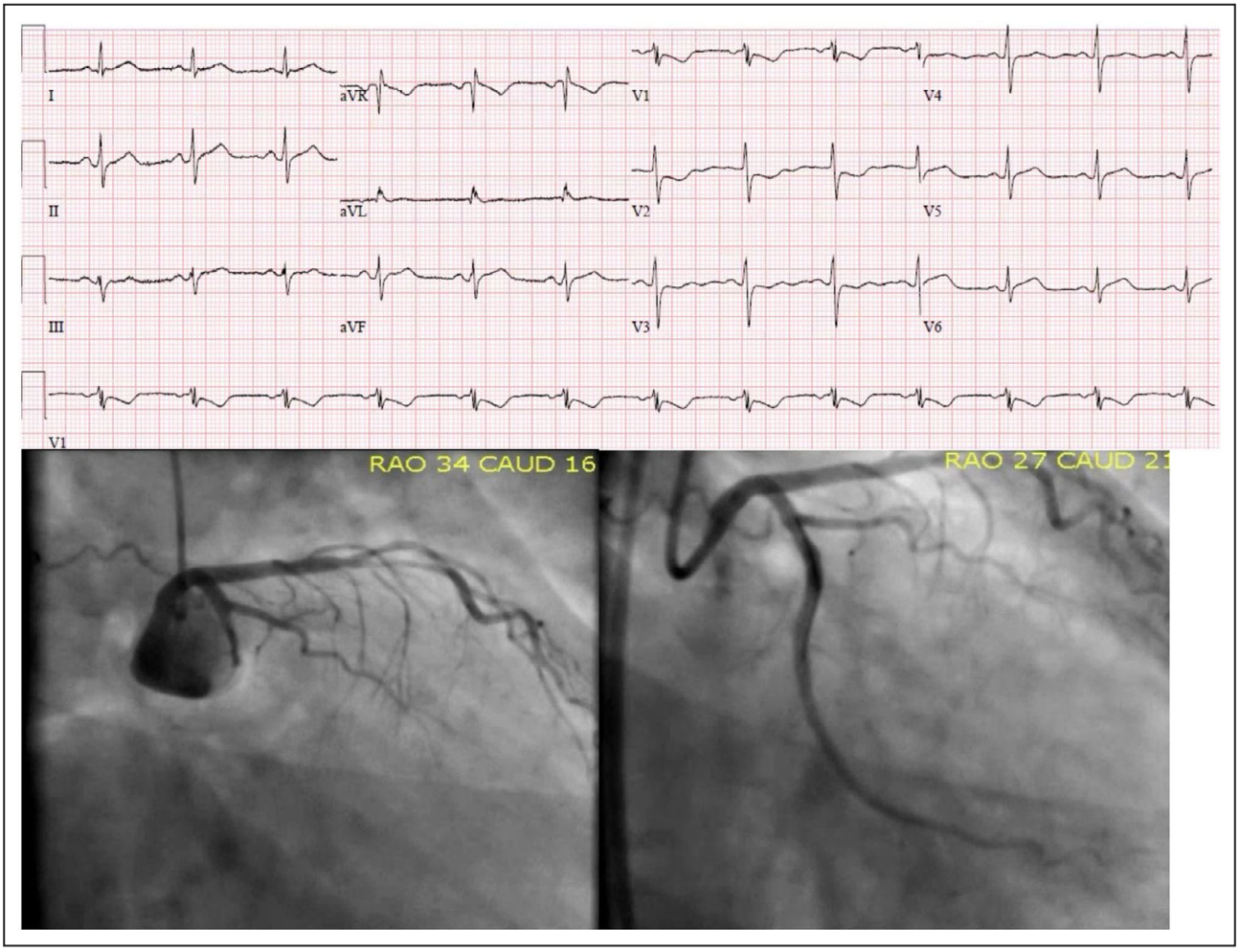

Figure 2. A 65-year-old woman presented with acute onset chest pain at rest for 4 hours.
The triage ECG (shown above) has ST-segment depression in V1–V3, maximal in V1–V2. There is also subtle evidence of inferior and lateral occlusion myocardial infarction with slight ST-segment elevation, ST-segment straightening, and large-volume T wave in II, III, and V6 (without ST-segment–elevation myocardial infarction [STEMI] criteria). Troponin T peaked at 8.45 ng/mL. Because the ECG did not meet STEMI criteria, the patient experienced a 15-hour delay until angiogram, when a complete (TIMI [Thrombolysis in Myocardial Infarction] 0) occlusion of the left circumflex artery was found and stented. The patient survived.

cardiac catheterization in Pride et al was ">29 hours" versus 75 minutes in our study. Because over half of all occluded arteries will spontaneously recanalize by next day angiogram, especially with antiplatelet and antithrombotic therapy, this difference is not unexpected. Such delayed reperfusion often results in irreversibly infarcted myocardium; salvage depends on rapid reperfusion, which was not achieved in Pride et al. Second, in Pride et al, arteries with culprit lesions but with TIMI 2 flow and significantly elevated troponin were not classified as occlusion; including such arteries is essential because 20% of patients with true STEMI who undergo immediate angiogram have TIMI 3 flow.[26,27] Third, Pride et al defined STD as at least 1 mm, did not take proportionality into account, and did not assess the location of maximal STD. Thus, they could have misclassified a patient with STD in leads V5–V6 (not maximal in V1–V4) as "isolated anterior ST depression."

Among the 99 patients with OMI with STDmaxV1–4 (all of whom are presumed to benefit from emergent reperfusion), the 52 (53%) patients lacking STEMI criteria had similarly high peak troponin levels but significantly lower chance of receiving catheterization within 90 minutes of presentation. We believe that these patients in need of emergent reperfusion likely did not receive it because of the absence of STEMI criteria, despite the fact that they could have been identified easily and immediately by STDmaxV1–4, in addition to other subtle signs of OMI. Furthermore, among one-third of those with STEMI criteria, those criteria emerged a median of 1 hour after appearance of STDmaxV1–4; earlier diagnosis by STDmaxV1–4 could have resulted in earlier reperfusion.

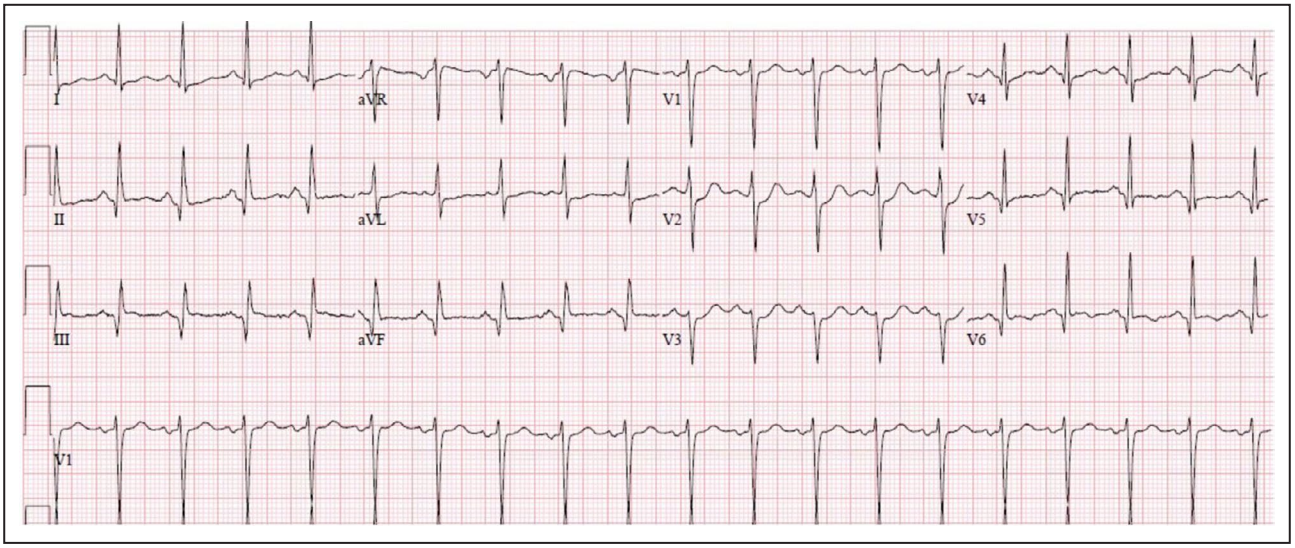

Figure 3. A 47-year-old man presented with chest pain and shortness of breath off and on for the past 24 hours, which had intensified within the past 2 hours.
The triage ECG (shown above) has ST-segment depression (STD) in V1–V4, maximal in V2. There is also subtle ST-segment elevation in lead III, with reciprocal STD in aVL. Because none of his ECGs met ST-segment–elevation myocardial infarction criteria, his angiogram was delayed for 15.68 hours from presentation, when a complete (TIMI [Thrombolysis in Myocardial Infarction] 0) left circumflex artery occlusion was found and stented. Troponin T peaked at 1.92 ng/mL. The patient survived.

Because STDmaxV1–4 is a single ECG finding designed only to detect the electrocardiographically posterior component of an OMI, it is not expected to be a sensitive finding for any OMI (eg, a posterior OMI finding should not be expected to be positive for an anterior OMI). Therefore, the clinical utility of this finding lies in its specificity, not its sensitivity, and our results confirm this with sensitivity of 38.5% and 37.4% for any

Table 5. Clinical Events and Outcomes in Each Subgroup of Patients With STDmaxV5–6, According to ECG Interpretation

Variable	Any STDmaxV5–6 (even with STEMI in other leads)	STDmaxV5–6 without concomitant STEMI criteria (excludes STEMI)	STDmaxV5–6 without concomitant STEMI criteria or other signs of OMI	STDmaxV5–6 deemed "ischemic" but not OMI or STEMI (starting with "ischemic" STDmaxV5–6, excluding STEMI and subtle OMI patterns)	STDmaxV5–6 deemed nonischemic (includes LVH, LBBB, and ventricular paced rhythm)
Total, N	196	174	148 (A+B)	58 (A)	90 (B)
Prior CABG	24 (12)	35 (20)	24 (16)	17 (29)	7 (8)
Catheterization	158 (81)	139 (80)	113 (76)	51 (90)	62 (69)
Culprit	94 (48)	76 (44)	54 (36)	36 (62)	18 (20)
OMI	60 (31)	42 (24)	20 (14)	16 (28)	4 (4)
PCI	79 (40)	62 (36)	41 (28)	27 (47)	14 (16)
3VD/ACS	19 (10)	17 (10)	15 (10)	13 (22)	2 (2)
Peak troponin I (ng/mL), median (IQR)	0.06 (0.02–5.11)	0.06 (0.02–3.49)	0.03 (0.02–0.13)	4.46 (0.24–8.71)	0.03 (0.02–0.06)
Peak troponin T (ng/mL), median (IQR)	0.12 (0.00–0.85)	0.06 (0.00–0.57)	0.03 (0.00–0.21)	0.17 (0.03–0.66)	0.01 (0.00–03)
Index death	10 (5)	9 (5)	8 (5)	7 (12)	1 (1)
Index CABG	4 (2)	4 (2)	4 (3)	2 (3)	2 (2)
Time to catheterization, median (IQR), min	892 (110–3116)	1229 (184–3398)	1462 (287–4218)	1314 (263–2781)	1960 (463–4980)
Catheterization <90 min	34 (17)	20 (11)	10 (7)	3 (5)	7 (8)

Data are given as number (percentage), unless otherwise indicated. 3VD indicates 3-vessel disease; ACS, acute coronary syndrome; CABG, coronary artery bypass grafting; IQR, interquartile range; LBBB, left bundle-branch block; LVH, left ventricular hypertrophy; OMI, occlusion myocardial infarction; PCI, percutaneous coronary intervention; STDmaxV5–6, ST-segment depression maximal in leads V5–V6; and STEMI, ST-segment–elevation myocardial infarction.

STDmaxV1–4 and suspected ischemic STDmaxV1–4, respectively.

Our results appear to confirm that STDmaxV1–4, when not secondary to an abnormal QRS, such as RBBB, is an accurate marker of posterior OMI and is clinically distinct from STDmaxV5–6. STDmaxV5–6 (and lead II), when not secondary to an abnormal QRS, such as LV hypertrophy, is a manifestation of subendocardial ischemia, which can be attributable to (1) supply-demand mismatch from various clinical causes or (2) ACS involving the left main, LAD, or triple-vessel disease). Although 84% of patients with suspected ischemic STDmaxV1–4 had OMI, only 28% of patients with suspected ischemic STDmaxV5–6 had OMI, and those with STDmaxV5–6 had a lower percentage requiring PCI (47% versus 81%; $P<0.001$) and lower median peak troponin T levels (0.17 versus 2.94 ng/mL; $P<0.001$) than those with STDmaxV1–4.

We found that most cases with STDmaxV1–4 were present concomitantly with subtle OMI findings of the inferior and/or lateral walls. Although we found that even <1.0 mm of STDmaxV1–4 was specific for OMI, we could not find any patient with OMI who had both (1) <1.0 mm STDmaxV1–4 and (2) no other findings of OMI in other areas of the ECG. If the interpreter is capable of identifying these other subtle signs of OMI, then we suggest that STDmaxV1–4 <1.0 mm without any other OMI signs (especially in the inferior and/or lateral walls) is unlikely to be posterior OMI based on our cohort. In our cohort, the only ECGs with unexplained STD not classified as OMI were those with <1 mm STD *and* no other subtle signs of OMI (and all 6 were true negatives).

At first glance, multiple prior studies[17,48,49,50] appear to show that STE in posterior leads is more sensitive than STD in anterior leads. However, these studies apply a double standard by examining <1 mm of STE in just a single posterior lead while requiring at least 1 mm of STD in 2 consecutive anterior leads.

Although our extensive electrocardiographic experience suggests that posterior leads add little to no value to expert evaluation for STDmaxV1–4, posterior leads could conceivably be shown to have a role if further studies were to compare them to equally detailed and subtle evaluation of anterior STD. However, on the basis of our findings, we believe STDmaxV1–4 to be highly accurate and sufficient without the need for routine posterior leads.

CONCLUSIONS

Among patients with symptoms suggestive of ACS in the ED, the specificity of suspected ischemic STDmaxV1–4 was 97% for the diagnosis of OMI and 96% for OMI requiring PCI. Many patients with OMI with STDmaxV1–4 had maximal STD <1 mm, and STEMI criteria missed half of OMIs detected by STDmaxV1–4. We found significant utility for differentiating posterior OMI from ischemic STD without OMI by evaluating STD maximal in V1–V4 versus V5–V6. These data justify prospective trials to assess the benefit of emergent reperfusion therapies for patients with non–ST-segment–elevation myocardial infarction with STDmaxV1–4. Until more data are available, any suspected ischemic STD maximal in V1–V4 in the setting of ACS is attributable to OMI until proven otherwise.

ARTICLE INFORMATION

Received June 12, 2021; accepted September 7, 2021.

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Sources of Funding

There was no funding for this study.

Disclosures

None.

Supplementary Material

Table S1
Figure S1–S2

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SUPPLEMENTAL MATERIAL

Contingency Tables

	Any STDmax1-4 +	Any STDmax1-4 -	Total
OMI +	102	163	265
OMI -	45	498	543
Total	147	661	808
	Suspected Ischemic STDmax1-4 +	Suspected Ischemic STDmax1-4 -	
OMI +	99	166	265
OMI -	19	524	543
Total	118	690	808
	Suspected Ischemic STDmax1-4 +	Suspected Ischemic STDmax1-4 -	
OMI requiring PCI	95	144	239
No OMI requiring PCI	23	546	569
Total			
	Expert Subjective Interpretation and STDmax1-4 +	Expert Subjective Interpretation and STDmax1-4 -	
OMI +	99	166	265
OMI -	13	530	543
Total	112	696	808

Table S1: Contingency Tables for all three categories of STDmaxV1-4 presented in the manuscript.

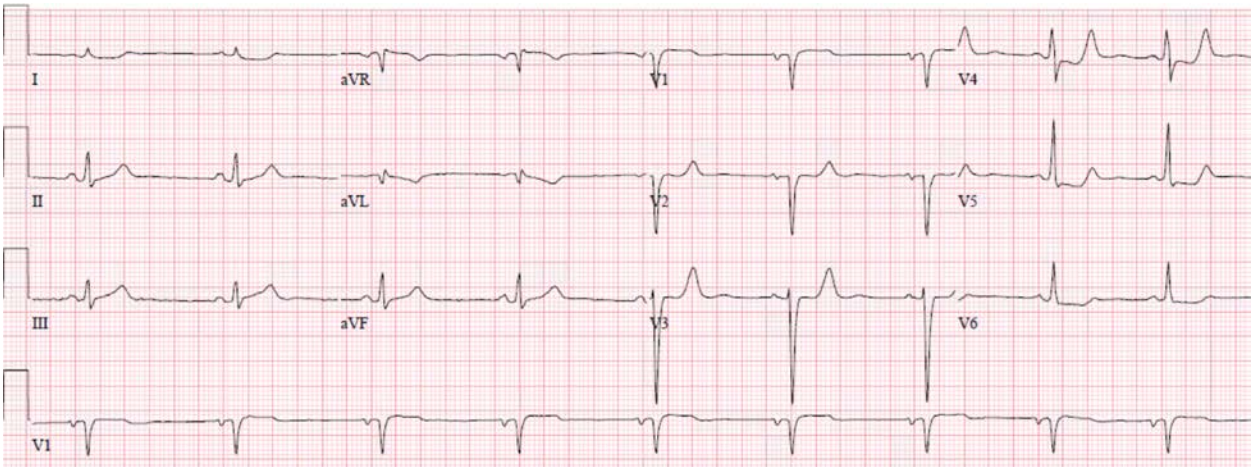

Figure S1. A 65-year-old man presented with 3 hours of chest pain and shortness of breath. The triage ECG (shown above) was interpreted as having STDmaxV1-4 (the STD is present in V2-V6, maximal in V4 in this case). Some interpreters might say that the STD is equal in V4 and V5, but this fails to take into account proportionality. Even if the STD were proportionally equal in V4 and V5, in our study we would err on the side of V4 indicating OMI. There is also subtle evidence of inferior OMI, including very slight STE and proportionally large T waves in lead III and reciprocal STD and negative T wave in lead aVL. The patient suffered a 4.68 hour delay from the ECG to catheterization, where a total (TIMI 0) occlusion of the PDA was found and stented. Troponin T peaked at 3.41 ng/mL. The patient survived.

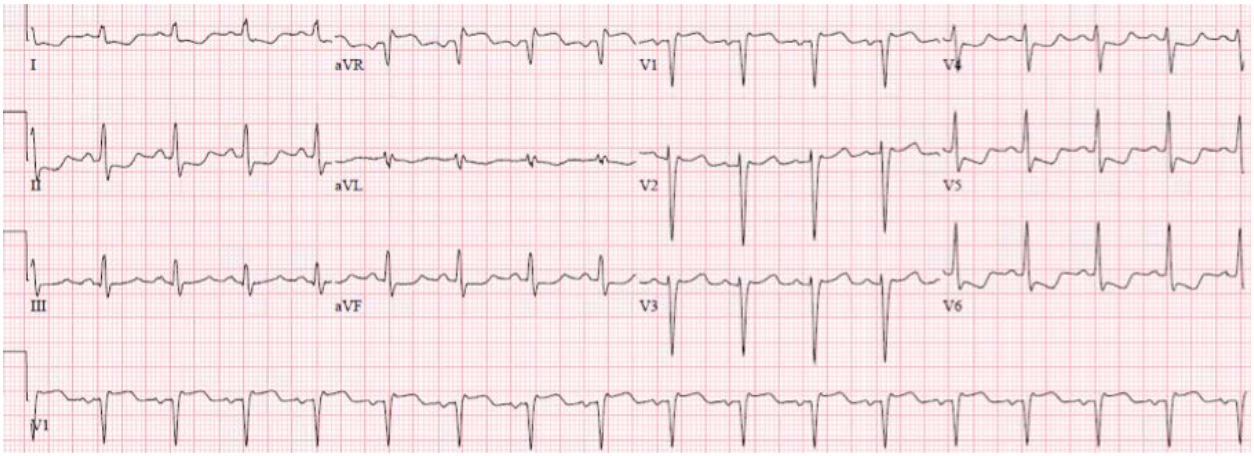

Figure S2. A 47-year-old female with prior history of 4-vessel CABG presented with nearly 24 hours of ongoing chest pain. Her triage ECG (shown above) shows STD in V4-V6, I, II, and aVF, with reciprocal STE in aVR and V1. The STD is maximal in leads V5-V6, as well as lead II. The patient was admitted for delayed cardiac catheterization, which occurred 23.7 hours after arrival and demonstrated known chronic severe 3 vessel disease, patent CABG grafts bypassing the left main and LAD, known chronically occluded CABG graft bypassing the RCA, and an acute 99% lesion (TIMI 3 flow) of the CABG graft bypassing the OM1. No PCI was performed, and the patient was managed medically. Peak troponin T was 0.34 ng/mL. The patient survived to discharge.