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Pitfalls of Posterior Leads for Posterior Occlusion MI

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The ST Elevation Myocardial Infarction (STEMI) paradigm has guided the diagnosis and treatment of acute MI, restricting emergent reperfusion for patients whose ECGs meet STEMI criteria. This performs poorly especially for posterior MI [1], which has been called the "dark side of the moon" [2] because the standard 12-lead ECG does not directly record posterior wall activity. While posterior STEMI is recognized as a STEMI equivalent that manifests on the 12-lead ECG as anterior ST depression, STEMI guidelines advise posterior leads to confirm posterior STE [3], but these may be underutilized. However, STEMI criteria have high rates of false positives (STE without occlusion) leading to unnecessary cath lab activation [4], and false negatives (Non-STEMI with occlusion) leading to delayed reperfusion and higher mortality [5]. This has led to a proposed paradigm shift from STEMI to Occlusion MI (OMI) [6]. While not yet universally adopted in guidelines, this approach is gaining traction and supported by growing evidence, including a recent study showing ischemic STD maximal in V1-4 (STDmaxV1-4) is 97% specific for OMI (and does not require tall R waves or upright T waves) [7].

That study, however, did not include posterior leads, so the role of posterior leads in diagnosing OMI remains unclear; and this finding from the high-risk population study may not generalize to all ED patients with chest pain. We reviewed two years of emergency department (ED) patients with chest pain and who had posterior leads performed. Our goal was to compare STEMI criteria with OMI criteria, review patient management and outcomes, and identify advantages and limitations of posterior leads.

This retrospective cohort study followed STROBE guidelines. We reviewed charts from July 2022 to June 2024 from two urban academic EDs. Research Ethics Board exemption (Quality Improvement Review Committee exemption #24-0901) was obtained as part of ongoing quality improvement for ED patients with acute coronary occlusion.

The hospital data center provided a list of all ED patients who presented with chest pain and had an order for both 12-lead and 15-lead ECG (which includes V4R and V8-9) in the online medical records. We excluded duplicate orders and those without both a 12-lead and 15-lead ECG recorded while in the ED, and excluded those admitted via emergency medical services directly to the cardiology service. Abstractors were trained and used standardized abstraction forms, with outcomes defined a priori. Patients were classified by outcome based on prior studies [8, 9]:(1) OMI (acute culprit lesion with either TIMI 0–2 flow or peak high-sensitivity troponin I > 10,000 ng/L, or if no angiogram then peak high-sensitivity troponin I > 10,000 ng/L plus new regional wall motion abnormality on echocardiogram), (2) NOMI (Non-OMI: type 1 MI not meeting OMI criteria, or type 2 MI), or MIRO (MI ruled out, with no rise in troponin). Patients were excluded if there was insufficient information to classify as OMI versus NOMI.

The first author (TN) reviewed the initial 12-lead ECGs and the first 15-lead ECGs to record the time stamp and whether the final interpretation by the over-reading cardiologist (who is blinded to clinical presentation and outcome) was "STEMI" or not. The other authors (JTTM and MEB, an emergency physician

and senior emergency resident) then reviewed all 12-lead ECGs, blinded to patient demographics, clinical information, cardiology interpretation and outcome, to determine whether they showed signs of OMI or not—including subtle STE and reciprocal STD, hyperacute T waves and reciprocal T wave inversion, suspected acute Q waves, modified Sgarbossa criteria for left bundle branch block or ventricular paced rhythm, and ischemic STDmaxV1-4 from posterior OMI [7]. After finalizing interpretations, they then reviewed all 15-lead ECGs for OMI, to see if this would change interpretation. JTTM then reviewed posterior leads for STE ≥ 0.5 mm in either V8 or V9, and compared with its reciprocal anterior STD. Patient charts were then reviewed for management (admission as code STEMI or not, and door-to-cath time), admission diagnosis (STEMI, Non-STEMI, or other), and final outcome (OMI, Non-OMI or MIRO).

There were 245 ED patients with chest pain who had both 12-lead and 15-lead ECGs recorded in the ED. There were 19

TABLE 1 | ECGs with either ischemic STDmaxV1-4 on 12-lead or posterior STE on 15 lead, including interpretation of STEMI by cardiologist and OMI by emergency physicians.

	Age	12 lead STEMI/OMI	12–15 time	15 lead: anterior vs. posterior leads	ED diagnosis and outcome, Door-to-cath time (min) sinitial and peak troponin I (ng/L)
1	39	STEMI(+) not OMI	1 min	Both STE: false positive from pericarditis	Pericarditis. Troponin 4 to 5
2	70	STEMI(−) not OMI	36 min	No anterior STE, false posterior STE	Pericarditis. Troponin 2 to 2
3	25	STEMI(−) Not OMI	114 min	Both STE: false positive	Myocarditis. Troponin 11,915 to 22,314
4	78	STEMI(−) Not OMI	10 min	Diffuse STD, posterior STE: false positive posterior	STEMI: No culprit. Troponin 42 to 70. Diagnosis: AF
5	25	STEMI(+) OMI inf-post-lat	7 min	Anterior STD, posterior STE: false positive posterior	STEMI: No culprit. Troponin 5604 to 16,374
6	55	STEMI(−) OMI postero-lat	46 min	Anterior STD, no posterior STE: false negative posterior	STEMI (91 min): 99% RCA. Troponin 333 to 44,938
7	75	STEMI(+) OMI infero-post	15 min	Anterior STD, no posterior STE: false negative posterior	STEMI (34 min): 100% RCA. Troponin 19,049 to 133,792
8	65	STEMI(−) OMI Posterior	39 min	Anterior STD, no posterior STE: false negative poisterior	Non-STEMI (2700 min): 100% LCX. Troponin 2201 to 52,652
9	58	STEMI(+) OMI Infero-post	6 min	Anterior STD > posterior STE	STEMI (44 min): 100% RCA. Troponin 6576 to 39,885
10	61	STEMI(+) OMI Inf-post-lat	10 min	Anterior STD > posterior STE	STEMI (60 min): 100% diagonal. Troponin 1376 to 28,761.
11	53	STEMI(−) OMI Infero-post	1 min	Anterior STD > posterior STE	STEMI (65 min): 100% RCA. Troponin 182 to 79,807
12	59	STEMI(−) OMI Infero-post	12 min	Anterior STD > posterior STE	STEMI (72 min): 100% RCA. Troponin 3306 to 66,554
13	46	STEMI(−) OMI Inf-post-lat	14 min	Anterior STD = posterior STE	Non-STEMI (2700 min): 100% LCX. Troponin 562 to 136,966
14	71	STEMI(+) OMI postero-lat	41 min	Dynamic change from OMI to VT	STEMI (181 min): 100% diagonal. Troponin 53 to 307,016
15	73	STEMI(−) OMI Infero-post	359 min	No posterior STE, resolution of anterior STD: reperfusion	Non-STEMI (912 min): 100% diagonal. Troponin 408 to 49,078
16	75	STEMI(−) Not OMI	280 min	New anterior STD = posterior STE	Non-STEMI (1703 min): 100% LCX. Troponin 5744 to 18,825
17	34	STEMI(−) Not OMI	12 min	No anterior STD posterior STE = false negative anterior	STEMI (58 min): 100% LCX. Troponin 27,960 to 39,574
18	100	STEMI(−) Not OMI	39 min	No anterior STD but posterior STE = false negative anterior	Non-STEMI (1046 min): 90% LAD +100% RCA. Troponin 60 to 13,477

patients admitted as Code STEMI, of whom 8 did not have OMI (42% false positive), and 71 admitted as Non-STEMI, of which 21 had OMI (29.6% false negative); yielding a total of 32 OMI, 51 NOMI, and 162 MIRO.

For the 11 true positive Code STEMI, the initial 12-lead was STEMI(+) OMI in 4 and STEMI(−) OMI in 5, including 3 involving posterior OMI. There were an additional 3 STEMI(−) OMI admitted as Non-STEMI, all involving posterior OMI. OMI signs on 12-lead ECG had twice the sensitivity of STEMI criteria combining 12 and 15 lead ECGs (37.5% vs. 18.8%, $p = 0.0066$) with preserved specificity (99.5 vs. 96.7%, $p > 0.05$). Interpreters agreed on OMI versus not OMI in 98.4% of ECGs (kappa = 0.71). Interpretation only changed from not OMI to OMI in 2 cases, one of which had dynamic change on anterior leads. Comparing ischemic STDmaxV1-4 on the 12-lead to STE ≥ 0.5 mm on V8 or V9 of the subsequent 15-lead found no difference in specificity (99.5 vs. 97.7%, $p > 0.05$) or sensitivity (31.3% vs. 25.0%, $p > 0.05$) for OMI.

Of 11 initial ECGs showing ischemic STDmaxV1-4, only one was false positive (and also had posterior STE): a Code STEMI with peak troponin of 16,374 ng/L and regional wall motion abnormality but no culprit lesion, diagnosed as myocardial infarction with non-obstructive coronary arteries (MINOCA). Of the 10 true positives, the subsequent 15 lead ECG showed: one had similar posterior STE, 4 had less obvious posterior STE, 3 had no posterior STE despite anterior STD, and 2 had a dynamic change and did not reveal either posterior STE or anterior STD. Three patients were admitted as Non-STEMI with delayed reperfusion.

Of 234 ECGs not showing ischemic STDmaxV1-4, there were 7 that had posterior STE on subsequent 15 lead, of which 4 were false positive (from pericarditis or atrial fibrillation with rapid ventricular response) and one had dynamic change with both posterior STE and new anterior STD. Only two showed posterior STE despite not showing anterior STD, of which one was still admitted as Non-STEMI with delayed reperfusion. The median time from 12-lead to 15-lead ECG was 63.0 min for all patients, 48.5 min for all OMI and 12 min for OMI admitted as STEMI.

Table 1 shows all patients with either 12-lead ECG ischemic STDmaxV1-4 or 15-lead ECG posterior STE to compare STEMI versus OMI interpretations and anterior versus posterior leads. Appendix S1 shows the associated ECGs.

Our study confirms and further clarifies the paradigm shift from STEMI to OMI, and specifically posterior OMI. First, our findings confirm STEMI criteria to be a poor surrogate marker for acute coronary occlusion, and that OMI signs have double the sensitivity with preserved high specificity [5]. We confirmed the study by Meyers et al. showing high specificity for STDmaxV1-4 for posterior OMI [7], and also found similar sensitivity and earlier diagnosis compared with posterior leads. In other words, posterior OMI may be the "dark side of the moon" for STEMI criteria, but OMI criteria shine a light.

Second, by comparing OMI criteria on 12-lead with STEMI criteria on 15-lead, we identified pitfalls of posterior leads. Because of small posterior lead voltage and the principle of proportionality, we found posterior STE was usually less obvious (and even falsely negative) compared with its reciprocal anterior STD. Conversely, posterior leads could be falsely positive from diffuse ST elevation (e.g., pericarditis) or from tachyarrhythmia. Posterior leads were more likely to be falsely positive, leading to unnecessary cath lab activation, or falsely negative, leading to delayed reperfusion. Posterior leads were only helpful in two cases showing posterior STE in the absence of reciprocal anterior STD, but one was still admitted as Non-STEMI.

Third, by comparing the time between 12-lead and 15-lead ECGs, we identified another pitfall and potential advantage of posterior leads. Interval reperfusion can lead to resolution of anterior STD with lack of posterior STE, while ongoing occlusion can lead to posterior STE along with new anterior STD. In other words, the 15-lead ECG functions as a serial ECG, which can show dynamic occlusion/reperfusion on both anterior and posterior leads.

Our study has several limitations. First, we had a relatively small number of patients with STEMI or OMI and excluded patients admitted directly to cardiology via EMS, and larger studies including pre-hospital transfers may provide more details. But this also indicates how often posterior leads are done in the absence of ischemic anterior STD. Secondly, we only examined the first 12-lead and first 15-lead ECG, which could have missed changes on serial 12-lead or serial 15-lead. Third, OMI interpretation was done by two emergency providers with an interest in ECG interpretation, which may not be representative either of the average emergency physician or expert interpretation. The former may limit the generalizability of our study, while the latter may underestimate the capacity to diagnose OMI based on the 12-lead ECG. Artificial intelligence ECG interpretation would help both generalize and standardize this process [10].

In conclusion, we confirmed the high specificity of ischemic STDmaxV1-4 for posterior OMI and found equal sensitivity and earlier diagnosis compared with posterior leads. Furthermore, by comparing ECG characteristics and timing of 12-lead and 15-lead ECG, we highlighted posterior lead pitfalls including less obvious, false negative, or dynamically resolved STE as well as false positive STE. We also identified potential advantages of posterior leads as a serial ECG (for both anterior and posterior leads) and for rare falsely negative anterior leads. This can help clinicians integrate ECG advances as part of the evolving paradigm shift from STEMI to OMI.

Author Contributions

T.N., M.E.-B., and J.T.T.M. contributed to the study concept/design, data acquisition and analysis, and data interpretation. J.T.T.M. drafted the manuscript, and J.T.T.M. and M.E.-B. contributed critical revisions.

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Disclosure

The authors have nothing to report.

Data Availability Statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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Supporting Information

Additional supporting information can be found online in the Supporting Information section. **Appendix S1:** acem70220-sup-0001-AppendixS1.pdf. **Supplement 1:** ECGs with either ischemic STDmaxV1-4 or posterior STE, comparing 12-lead STEMI vs. OMI, and 15 lead anterior STD vs. posterior STE.