ECG Patterns of Occlusion Myocardial Infarction: A Narrative Review

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The traditional management of acute coronary syndrome has relied on the identification of ST-segment elevation myocardial infarction (STEMI) as a proxy of acute coronary occlusion. This conflation of STEMI with acute coronary occlusion has historically overshadowed non–ST-segment elevation myocardial infarction (NSTEMI), despite evidence suggesting 25% to 34% of NSTEMI cases may also include acute coronary occlusion. Current limitations in the STEMI/NSTEMI binary framework underscore the need for a revised approach to chest pain and acute coronary syndrome management. The emerging paradigm distinguishing occlusion myocardial infarction from nonocclusion myocardial infarction (NOMI) seeks to enhance diagnostic accuracy and prognostic effect in acute coronary syndrome care. This approach not only emphasizes the urgency of reperfusion therapy for high-risk ECG patterns not covered by current STEMI criteria, but also emphasizes the broader transition from viewing acute coronary syndrome as a disease defined by the ECG to a disease defined by its underlying pathology, for which the ECG is an important but insufficient surrogate test. This report outlines the emerging occlusion myocardial infarction paradigm, detailing specific ECG patterns linked to acute coronary occlusion, and proposes a new framework that could enhance triage accuracy and treatment strategies for acute coronary syndrome. Although further validation is required, the occlusion myocardial infarction pathway holds promise for earlier acute coronary occlusion detection, timely cath lab activation, and improved myocardial salvage—offering potentially significant implications for both clinical practice and future research in acute coronary syndrome management. [Ann Emerg Med. 2025;85:330-340.]

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INTRODUCTION

The primary treatment of atherothrombotic acute myocardial infarction involves urgent myocardial reperfusion and has traditionally prioritized the treatment of ST-segment elevation myocardial infarction (STEMI) with rapid reperfusion within less than 120 minutes of acute coronary occlusion to improve prognosis. According to the Fourth Universal Definition of Myocardial Infarction, the STEMI criteria consist of (1) 1 mm ST-segment elevation in any 2 consecutive leads at the J point, excluding V2 and V3; and (2) age- and sex-specific ST-segment elevation thresholds in V2 and V3 ($\geq$1.5 mm elevation in women regardless of age, $\geq$2.5 mm in men $<$40 years, and $\geq$2 mm in men aged $\geq$40 years), in the absence of left ventricular hypertrophy and bundle branch block.[1] These criteria have been endorsed in clinical practice guidelines.[2] STEMI criteria were initially established to separate myocardial infarction from nonmyocardial infarction individuals using patients with CK-MB and not to identify acute coronary occlusion or to stratify patients for reperfusion benefits, but the scientific community treats STEMI and non–ST-segment elevation myocardial infarction (NSTEMI) as surrogates of acute coronary occlusion myocardial infarction and nonocclusion myocardial infarction, respectively.[3] Unfortunately, approximately half of high-risk patients with occlusion myocardial infarction fail to meet STEMI criteria, resulting in delays in cath laboratory activation and depriving them of the potential benefits of urgent revascularization.[4,5] An increasing body of evidence identifies distinct ECG patterns that signify occlusion myocardial infarction, even in the absence of traditional ST-segment elevation criteria, challenging the prevailing STEMI-focused paradigm. As these insights evolve, the traditional STEMI/NSTEMI framework may ultimately transition to a more precise occlusion myocardial infarction/nonocclusion myocardial infarction model.

ACUTE CORONARY OCCLUSION MYOCARDIAL INFARCTION: BEYOND THE STEMI/NSTEMI PARADIGM

Occlusion myocardial infarction represents an ongoing ischemia resulting in irreversible infarction caused by

complete or near-complete occlusion of a culprit epicardial coronary artery, with inadequate collateral circulation, thus necessitating immediate reperfusion.[6] Occlusion myocardial infarction is a clinical diagnosis that involves a continuous, vigilant assessment of dynamic patient data, rather than reliance on isolated ECG, echocardiographic, or angiographic findings. Importantly, some cases of occlusion myocardial infarction may lack clear ECG changes initially, with the diagnosis only becoming evident as the ischemic process unfolds. Hence, clinicians should remain acutely aware that even a nondiagnostic ECG may conceal an urgent need for reperfusion in the evolving context of occlusion myocardial infarction.[6] Confusion arises when patients with ECG features of occlusion myocardial infarction before the angiogram later have an open artery at angiogram. This is because of the dynamic nature of coronary thrombosis, with spontaneous reperfusion occurring between the time of ECG recording and the angiogram. Among all cases of definite "STEMI," one-third have an open artery by the time of the angiogram.[7-9] The diagnosis of occlusion myocardial infarction extends beyond a single ECG reading and requires dynamic assessment of ECG changes in serial tracings relative to symptom evolution, particularly when chest pain exhibits a crescendo pattern.[10-12] However, even in the presence of a significant, persistent occlusion proven by TIMI-0 flow at angiography, STEMI criteria often do not ever develop. A more flexible approach, recognizing the evolving nature of ECG patterns in acute coronary syndrome, can improve diagnostic accuracy and ensure timely reperfusion therapy in patients with occlusion myocardial infarction.[2,12-14]

Timely reperfusion is crucial in acute atherothrombotic myocardial infarction. Although a meta-analysis showed that the overall sensitivity of STEMI criteria for occlusion myocardial infarction is 43% (95% confidence interval 34.7% to 52.9%), another meta-analysis involving 40,777 patients with NSTEMI showed that 25.5% had total coronary occlusion (TIMI-0 flow) at next day angiogram.[4,15] Additionally, in a larger meta-analysis of 25 studies, comprising 60,898 patients with NSTEMI, 17,212 (28%) were found to have an occluded culprit artery (TIMI 0/1 flow) at delayed angiogram.[16] Because many coronary arteries will reperfuse spontaneously in the time period between presentation and next day angiogram, the percent that were occluded at presentation is almost certainly higher, and this explains the difference between the NSTEMI studies and the meta-analysis of sensitivity of STEMI criteria for occlusion myocardial infarction. Notably, in this group, patients with NSTEMI occlusion myocardial infarction—despite being younger and healthier at baseline—had a nearly 2-fold increase in 1-year mortality compared with patients with NSTEMI nonocclusion myocardial infarction. The DIFOCCULT study, which evaluated 1,000 patients each from STEMI, NSTEMI, and control groups, found that 28% of patients with NSTEMI were reclassified as having acute coronary occlusion based on blinded ECG interpretation by 2 cardiologists.[17] This subgroup showed a high-risk clinical profile comparable with patients with STEMI, with increased prevalence of acute coronary occlusion, larger infarct sizes, and higher mortality. In a retrospective case-control study of 808 patients with suspected acute coronary syndrome, 2 ECG experts, blinded to all patient information except for age and sex, used predetermined occlusion myocardial infarction criteria and achieved greater diagnostic accuracy in identifying acute coronary occlusion than the traditional STEMI criteria.[6] These findings highlight the importance of incorporating detailed ECG analysis to identify a subgroup of patients with high-risk acute coronary syndrome who could benefit from timely emergent reperfusion.[18,19]

ELECTROCARDIOGRAPHIC PATTERNS OF ACUTE CORONARY OCCLUSION MYOCARDIAL INFARCTION

In the last decade, there have been descriptions of ECG patterns requiring urgent reperfusion therapy, even if they do not meet the conventional STEMI criteria.[20,21] Martí et al[22] found that 20% of 504 suspected patients with STEMI treated by systematic primary percutaneous coronary intervention displayed subtle ST-segment elevation (0.1 to 1 mm), often indicating acute coronary occlusion. Three quarters of occlusion myocardial infarctions initially overlooked can indeed be identified by subtle ST changes suggesting dynamic or evolving ST-segment elevation.[17] On a detailed analysis of 146 patients who were diagnosed with acute coronary occlusion earlier by occlusion myocardial infarction criteria than by STEMI criteria, several diagnostic patterns were more frequently identified (Table 1).[6] We detail here high-risk ECG occlusion myocardial infarction patterns (Table 2, Figure 1) indicative of acute myocardial ischemia resulting from active occlusion or reperfused/reperfusing occlusion of a coronary artery with residual critical stenosis.

Wellens Syndrome

Wellens syndrome is characterized by biphasic (pattern A) or deeply inverted (pattern B) T wave in leads V2-V3 and the absence of Q waves (Figure 1, Table 2) in a patient with recently resolved chest pain, indicating critical and proximal stenosis of the left anterior descending coronary artery.[23] Wellens' pattern represents acute reperfusion of occlusion

Table 1. Frequently observed occlusive myocardial infarction ECG patterns.

ECG Pattern	%
Subtle STE, that did not fulfill the typical STEMI criteria	83%
Reciprocal ST-depression and/or T-wave inversion	82%
Any STE in inferior leads coupled with ST-depression or T-wave inversion in lead aVL	50%
Terminal QRS distortion in leads V2-V3	50%
Subtle STE associated with pathologic Q waves, which cannot be attributed to old MI	47%
Posterior OMI, ST-depression predominantly in the V2-V4 leads	45%

From Meyers et al[6]
MI, Myocardial infarction; *OMI*, occlusion myocardial infarction; *STE*, ST-segment elevation.

myocardial infarction that went unrecorded by the ECG during the episode of pain. Cardiac biomarker levels almost always have some amount of elevation above the upper reference limit. During the active symptoms, the culprit vessel is functionally occluded (but neither ECG nor angiogram was recorded or recognized during that time), but then there is spontaneous or medication-induced partial thrombolysis with the restoration of some myocardial blood flow, causing resolution of active occlusion myocardial infarction findings and symptoms. This is usually immediately followed by the ECG pattern of coronary reperfusion, which is characterized by terminal T-wave inversion that enlarges progressively to full T-wave inversion over the course of days to weeks and eventually resolves to baseline weeks or even months after resolution of the event.[24] The reperfusion pattern occurs in all coronary distributions (Figure E1, available at http://www.annemergmed.com) but has been best named and described in the left anterior descending coronary artery distribution as Wellens syndrome.

Hyperacute T Waves

Hyperacute T waves is a term used to describe a distinctive T-wave change seen in the early to middle stages of occlusion myocardial infarction, in which the T waves in the affected leads are both more symmetric and enlarged in terms of overall area under the curve relative to both their baseline size and to their QRS complex size. The increase in area under the curve and symmetry involves components of increased T-wave breadth, increased T-wave height, diminished concavity, or even evolving to a straight or convex slope of both sides of the T wave, and increased QT interval.[25] Hyperacute T waves are often the earliest specific ECG feature of occlusion myocardial infarction that can be recognized by trained and qualified ECG readers, exceeding the diagnostic accuracy of STEMI criteria in blinded studies. In the absence of specific guidelines defining hyperacute T waves, the ratio of the T-wave amplitude, and especially the total area under the curve to the preceding QRS complex, is more relevant than isolated T-wave amplitude. Hyperacute T waves should be only assessed in proportion to QRS amplitude, and that QRS analysis is as important as ST-T analysis in the diagnosis of occlusion myocardial infarction (Figure 1, Table 2, Figure E2, available at http://www.annemergmed.com).[26] Hyperacute T waves are now a formal "STEMI-Equivalent" according to the American College of Cardiology.[14] Differential diagnoses of hyperacute T waves include hyperkalemia, left ventricular hypertrophy, early repolarization, bundle branch block, pericarditis, valvular heart disease, and left ventricular aneurysm.[27] Smith et al[28] further developed and validated an ECG rule to distinguish between anterior acute STEMI and left ventricular aneurysm; the rule uses higher T-wave amplitude compared with the QRS to identify acute occlusion myocardial infarction.[29]

De Winter Pattern

De Winter et al[30] identified an ECG pattern without ST-segment elevation, characterized by a 1- to 3-mm upward-sloping ST-depression at the J point in precordial leads (V1-V6), succeeded by tall, positive, symmetrical T wave, and 1- to 2-mm ST-segment elevation in lead aVR (Figure 1, Table 2, Figure E3, available at http://www.annemergmed.com). De Winter T waves are considered an anterior STEMI-equivalent indicative of proximal left anterior descending coronary artery occlusion myocardial infarction, necessitating emergency coronary angiography; however, they are in fact just a small but more easily recognized subset of hyperacute T waves. Cao et al[31] observed cases where de Winter's T wave evolved into hyperacute T waves without ST-depression, ST-segment elevation, or manifest after an initial ST-segment elevation or hyperacute T-waves phase. Most patients with hyperacute T waves do not exhibit the additional de Winter feature of depressed ST takeoff. The pattern was described first in the left anterior descending coronary artery distribution, but it can occur in any coronary distribution.

Aslanger Pattern

The Aslanger pattern has emerged as a distinctive ECG signature associated with acute inferior occlusion myocardial infarction, more often circumflex occlusion than right coronary artery, in individuals with concurrent multivessel disease. It is distinguished by ST-segment elevation in lead III only (often <1 mm) in the context of widespread ST-depression. Noteworthy is the absence of ST-segment elevation in a contiguous lead, so this pattern

Table 2. High-risk ECG patterns indicative of acute coronary occlusion.

OMI Patterns	ECG Features	Coronary Anatomy
Hyperacute T Waves[25,26]	• Broad-based T waves, disproportionally large by area (both height and width) compared with their QRS complex, bulky, increased convexity, and abnormally symmetric • T-wave amplitude/QRS amplitude ratio holds more clinical significance than T-wave amplitude alone	Any artery, in accordance with localization of ischemia
De Winter pattern[30]	• ST-depression at the J point in precordial leads, 1-3 mm upward-sloping, succeeded by tall, positive, symmetrical T wave • 1-2 mm STE in lead aVR	Proximal LAD occlusion
Aslanger pattern[32]	• STE isolated to lead III but not in any other inferior lead • ST-depression in leads V4-V6 without involvement of V2, with a positive/terminally positive T wave • ST segment in lead V1 > V2	LCX or RCA occlusion in multivessel disease
South African flag sign[33]	• STE in leads I, aVL, and V2 • ST-depression in lead III	Occlusion of the first diagonal branch of LAD
New-onset RBBB and LAFB[36,37]	• New-onset RBBB • New-onset LAFB • Concordant STE in any of the anterior and/or lateral leads; concordant ST-depression in the inferior leads	Proximal LAD occlusion
Posterior OMI[39]	• ST-depression max in V1-V4, not due to an abnormal QRS (eg, RBBB)	Occlusion of variable arteries, usually LCX, RCA, or their branches
Terminal QRS distortion[42]	• Absence of an S wave and J wave in either leads V2 or V3	LAD occlusion
Modified Sgarbossa-Smith criteria[44,45]	• Concordant STE of ≥1 mm in ≥ 1lead • Concordant ST-depression of ≥1 mm in any one of leads V1-V3 • STE/S-wave amplitude ≥0.25 (excessively discordant)	In accordance with localization of ischemia
Precordial Swirl[47]	• STE in aVR and V1-V2 • ST-depression in V5-V6	LAD occlusion
Northern OMI	• Any STE in aVR and aVL with negative T waves • Any ST-depression in inferior and lateral precordial leads with positive or biphasic T waves	LAD and first diagonal bifurcation occlusion in multivessel disease
Reperfusion Patterns (Wellens syndrome)[23]	• Terminal T-wave inversion (eg, Wellens pattern A) that progresses to full T-wave inversion (eg, Wellens pattern B) over time because the myocardium is reperfused	Any artery (proximal LAD in Wellens syndrome)

LAD, Left anterior descending coronary artery; *LAFB*, left anterior fascicular block; *LCX*, left circumflex artery; *OMI*, occlusion myocardial infarction; *RBBB*, right bundle branch block; *RCA*, right coronary artery; *STE*, ST-segment elevation.

does not meet STEMI criteria.[32] Patients with the Aslanger pattern had a larger infarct size, as evidenced by higher 24-hour troponin levels, a higher frequency of angiographic culprit lesions, and a greater frequency of composite acute coronary occlusion endpoints than their non-STEMI counterparts. In contrast, they exhibited similar inhospital and 1-year mortality rates compared with patients with inferior STEMI (Figure 1, Table 2, Figure E4, available at http://www.annemergmed.com).[32]

South African Flag Sign

The South African flag sign is a unique ECG pattern that recognizes high lateral infarction. It is characterized by ST-segment elevation, often subtle, in leads I, aVL, and V2, and ST-depression in lead III (Figure 1, Table 2, Figure E5, available at http://www.annemergmed.com). Although ST-segment elevation in leads I and aVL is indeed continuous ST-segment elevation, it may not be recognized as such because ECG formats frequently do not display I and aVL as contiguous, and especially because in this pattern, ST-segment elevation manifests in only one precordial lead (V2). Using a 3×4 lead display format of 12-lead ECG, it has a visually striking manifestation that reminds the design elements of the South African national flag.[33] It is typically associated with acute coronary occlusion of the first diagonal branch of the left anterior descending coronary artery.

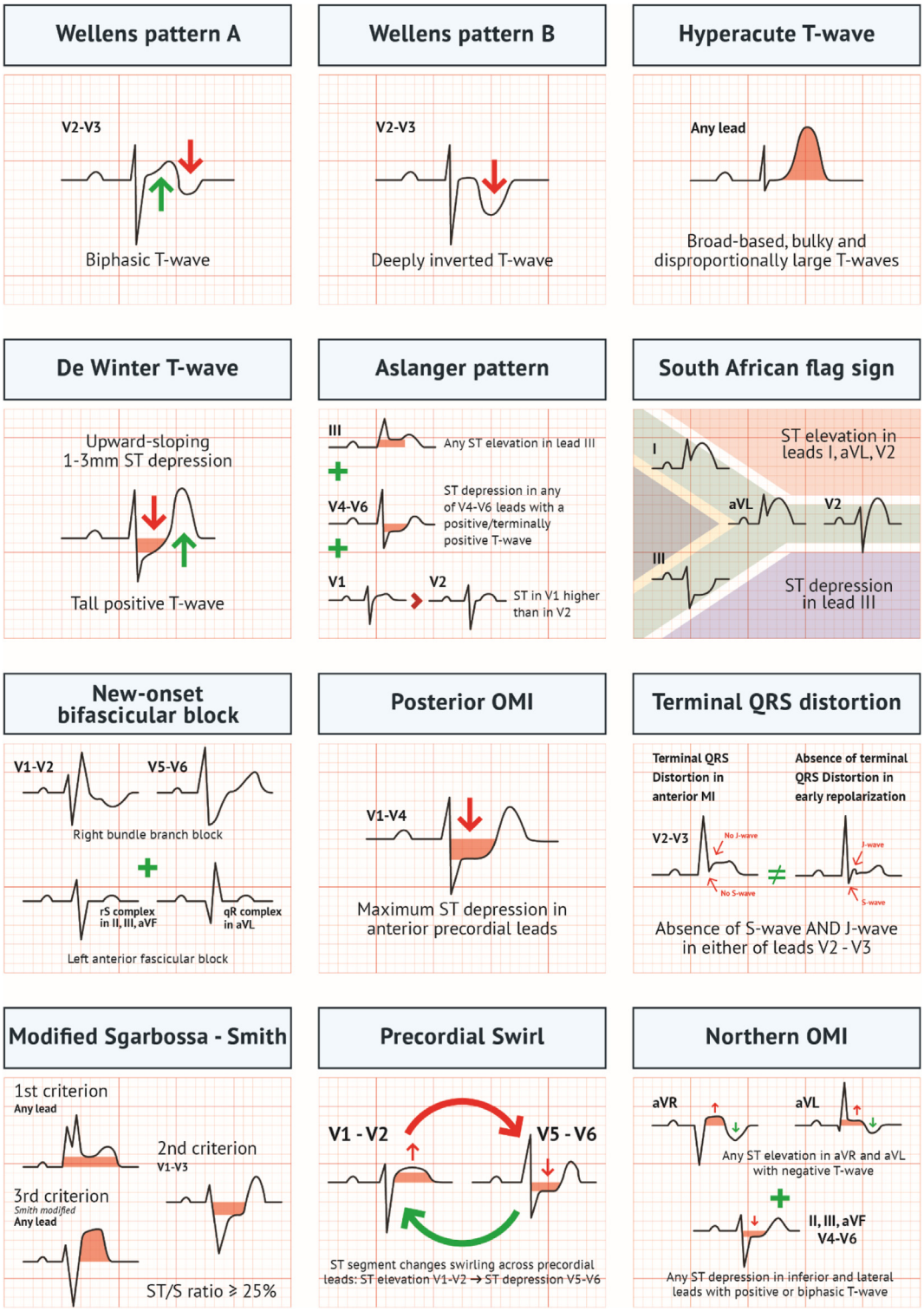

Figure 1. Electrocardiographic occlusion myocardial infarction (OMI) patterns.

New-Onset Bifascicular Block

Previous research has linked the new onset of left bundle branch block with proximal left anterior descending coronary artery occlusion and high-risk myocardial infarction.[34] However, the traditional clinical belief that left bundle branch block is caused by extensive septal infarction has been challenged. A necropsy study disclosed that blood supply to the right bundle branch and left anterior fascicle

is predominantly provided by septal perforators of the proximal left anterior descending coronary artery in 90% of the cases examined.[35] Similarly, the right coronary artery supplies the left posterior fascicle in the same percentage of cases. Notably, a dual blood supply to these fascicles occurs in 40% to 50% of instances. Thus, obstruction of the proximal left anterior descending coronary artery may result in a right bundle branch block and/or left anterior fascicular block. However, for myocardial infarction to cause left bundle branch block, typically both proximal left anterior descending coronary artery and right coronary artery occlusions are required. Acute myocardial infarctions presenting with right bundle branch block are often due to acute coronary occlusion and are more frequently managed with primary percutaneous coronary intervention than those presenting with left bundle branch block.[36] The 2023 European Society of Cardiology guidelines recommend prompt reperfusion therapy in patients exhibiting signs or symptoms indicative of ongoing myocardial ischemia accompanied by right bundle branch block.[2] However, the STEMI quantitative millimeter criteria have been shown to be only 40% sensitive for occlusion myocardial infarction in the setting of right bundle branch block (Figure 1, Table 2, Figure E6, available at http://www.annemergmed.com).[37]

Posterior Occlusion Myocardial Infarction

Posterior infarction is a frequently missed challenging diagnosis with the standard 12-lead ECG, which inadequately captures the left ventricular segments opposite the anterior wall. The Fourth Universal Definition of Myocardial Infarction suggests that isolated ST-depression 0.5 mm and greater in V1-V3 leads may indicate left circumflex artery occlusion, best detected using posterior leads (ST-segment elevation $\geq$0.5 mm in V7-V9), with increased specificity at ST-segment elevation cutoff 1 mm and greater.[1] Isolated posterior occlusion myocardial infarctions are infrequently identified by ST-segment elevation in the standard 12 leads, and even when ST-segment elevation is present, it may not meet STEMI criteria. A TRITON-TIMI-38 trial subanalysis revealed around one third of patients with "isolated precordial ST-depression" had acute coronary occlusion, leading to worse outcomes.[38] The study suggests that patients with patent arteries may have been occluded during ECG recording, supporting the idea that two thirds of ST-depression in V1-V4 indicate acute coronary occlusion. A subanalysis of the DOMI-ARIGATO study documented that in individuals with high-risk acute coronary syndrome, the specificity of any amount of ischemic ST-depression (even $<$1 mm) in leads V1-V4 was found to be 97% for occlusion myocardial infarction and 96% for occlusion myocardial infarction requiring urgent percutaneous coronary intervention (Figure 1, Table 2, Figure E7, available at http://www.annemergmed.com).[39] Posterior occlusion myocardial infarction is now a formal "STEMI-Equivalent" according to the American College of Cardiology.[14]

Differentiating the ST-Segment Elevation of Normal Variant in V2-V4 From Subtle Occlusion Myocardial Infarction

In patients with ST-segment elevation in V2-V4 and ischemic symptoms, differentiating a patient's normal baseline ST-segment elevation ("early repolarization") from left anterior descending coronary artery occlusion myocardial infarction can be challenging. Smith et al[26] provided criteria to differentiate early repolarization from subtle occlusion myocardial infarction, emphasizing the utility of R-wave and QRS amplitude as well as the QT interval. In this study, 143 patients with nonobvious left anterior descending coronary artery occlusion myocardial infarction were compared with 171 early repolarization cases, with derivation and validation groups. An ECG formula was derived and validated to distinguish between occlusion myocardial infarction and early repolarization. The variables were R-wave amplitude in lead V4, QTc duration, and ST-segment elevation measurement at 60 ms after the J point in lead V3, with a high sensitivity (86%), specificity (91%), and diagnostic accuracy (88%). According to this formula, a calculated value exceeding 23.4 indicates occlusion myocardial infarction, whereas values less than or equal to 23.4 suggest early repolarization. Driver et al[40] introduced a 4-variable formula, including V2 QRS amplitude, to better differentiate between early repolarization and left anterior descending coronary artery occlusion myocardial infarction. At a cutoff point of 18.2 and greater, this formula achieved better sensitivity (89%), specificity (95%), and diagnostic accuracy (92.0%). Bozbeyoglu et al[41] externally validated both formulas.

Terminal QRS Distortion

Terminal QRS distortion, as defined by absence of both an S-wave and J-point notching in both leads V2 and V3, is virtually always absent in the setting of normal precordial ST-segment elevation ("early repolarization"). Thus, when there is any precordial ST-segment elevation, if terminal QRS distortion is present, it is highly specific for left anterior descending coronary artery occlusion myocardial infarction (Figure 1, Table 2, Figure E5).[22,42] Lee et al[42] found that all 171 cases of early repolarization had S waves

in V2; 90% had S waves in V3, and all ECGs without S waves in V3 had prominent J waves (J-point notching). Thus, the absence of both an S wave and a J wave in V2 and V3 effectively ruled out normal ST-segment elevation.

Smith-Modified Sgarbossa Criteria

Diagnosing occlusion myocardial infarction without meeting STEMI criteria in patients with left bundle branch block or ventricular paced rhythm can be challenging due to the secondary alterations in ventricular depolarization and repolarization. In the GUSTO-1 trial, Sgarbossa et al[43] proposed acute myocardial infarction criteria for left bundle branch block, requiring 3 points and greater (specificity >90%): (1) concordant ST-segment elevation 1 mm and greater in ≥1 lead (5 points) and greater; (2) concordant ST-depression 1 mm and greater in any one of leads V1-V3 (3 points); (3) excessively discordant ST-segment elevation, defined as 5 mm and greater in 1 or more leads with negative QRS (2 points) (Figure 1, Table 2, Figure E8, available at http://www.annemergmed.com). This study has limitations, such as using any acute myocardial infarction endpoint, specifically patients who are CK-MB positive, a small cohort size, and use of an absolute ST-segment elevation cutoff of 5 mm rather than ST-segment elevation proportional to S-wave depth. Smith et al[44] revised Sgarbossa criteria (Table 2) by replacing the third absolute criterion (excessively discordant ST-segment elevation) with a proportional one. These criteria were validated by Meyers et al[45] on 49 occlusion myocardial infarction, and 249 controls, with left bundle branch block. The Smith-modified Sgarbossa criteria now require concordant ST-segment elevation 1 mm and greater in 1 lead and greater, concordant ST-depression 1 mm and greater in 1 lead and greater of V1-V3, and proportionally excessive discordant ST-segment elevation in 1 lead and greater anywhere with 1 mm and greater ST-segment elevation, as defined by 25% and greater of the depth of the preceding S wave (ST/S ratio ≥0.25). A cutoff of ST-segment elevation to S-wave ratio of 20%, rather than 25%, was more sensitive and almost as specific. The Smith-modified Sgarbossa criteria are more specific and significantly more sensitive than the original Sgarbossa criteria for diagnosing occlusion myocardial infarction in the presence of ventricular paced rhythm.[46]

Precordial Swirl

The "precordial swirl" occlusion myocardial infarction pattern features marked ST-segment elevation and/or hyperacute T waves in V1-V2 and ST-depression and/or T-wave inversion in V5-V6, creating a distinctive clockwise vortex appearance of the ST-T waves through precordial leads. This pattern exhibits a rightward ST-segment elevation vector with elevation in V1 and aVR and reciprocal ST-depression in V5-V6, indicating left anterior descending coronary artery occlusion myocardial infarction, typically proximal to the first septal perforator (Figure 1, Table 2, Figure E9, available at http://www.annemergmed.com).[47,48] The *precordial swirl* occlusion myocardial infarction pattern results from transmural ischemia affecting the anterior wall, apex, and septum. Clinicians must differentiate it from left ventricular hypertrophy, left bundle branch block, and subendocardial ischemia pattern, because these may present similar ST-segment deviations. Prospective studies are needed to establish its specificity and sensitivity.

Northern Occlusion Myocardial Infarction

We have recently documented a novel occlusion myocardial infarction pattern in patients with concurrent repolarization abnormalities from transmural and subendocardial ischemia in the setting of multivessel coronary artery disease. This pattern, named "northern occlusion myocardial infarction," is defined by any ST-segment elevation in aVR and aVL with negative T wave and any ST-depression in inferior and lateral precordial leads with positive or biphasic T waves (Figure 1, Table 2, Figure E10, available at http://www.annemergmed.com). The terminology "northern occlusion myocardial infarction" encapsulates the unique vector orientation and its diagnostic significance in identifying this peculiar acute coronary occlusion pattern. Prospective studies are needed to establish its specificity and sensitivity.

EFFECT OF AGE, ETHNICITY, AND SEX

Variability in ST-segment elevation significantly influences the diagnostic accuracy of ECG for acute coronary occlusion with substantial effects from ethnicity, age, and sex. It is already known that age and sex affect STEMI classification, but reference values are predominantly derived from populations with a Western European descent.[49-51] The prevalence of ECGs exceeding ST-segment elevation thresholds is notably higher in younger individuals and men, especially sub-Saharan African men who typically show higher J-point amplitudes. Up to 27% of Ghanaian men aged below 40 years exhibit ST-segment elevation, whereas older Turkish women show no occurrences.[52] The J point is essential for diagnosing STEMI, but current diagnostic thresholds overlook ethnic variations in baseline J-point amplitudes.[2,53,54] This oversight can lead to misdiagnosis: higher baseline

amplitudes in sub-Saharan African populations may cause false positives and unnecessary interventions, whereas lower amplitudes in Turkish women can lead to false negatives, delaying treatment and potentially worsening outcomes.[49-51,55,56] The extremely low prevalence of baseline ST-segment elevation in women and certain ethnic subgroups could lead to a high rate of false-negative STEMI diagnosis. Women and certain ethnic groups have lower baseline ST values and must develop more significant ST-segment elevation to exceed thresholds.[52] Adjusting thresholds for specific female and ethnic subgroups could enhance ECG sensitivity for acute coronary occlusion and improve treatment outcomes. Additional research is essential to explore whether ethnicity-dependent differences in ST-segment elevation during acute coronary syndrome augment or mitigate these baseline differences, putatively resulting in adverse outcomes with both overdiagnosis and underdiagnosis of acute coronary occlusion.

THE OCCLUSION MYOCARDIAL INFARCTION PATHWAY: LIMITATIONS AND FUTURE RESEARCH

Implementing the occlusion myocardial infarction pathway (Figure 2) involves addressing current challenges and clarifying the roadmap for future research. Current evidence linking occlusion myocardial infarction to poorer prognosis, such as larger infarct sizes and higher event rates, is primarily drawn from large observational studies and supported by a strong pathophysiological rationale.[57]

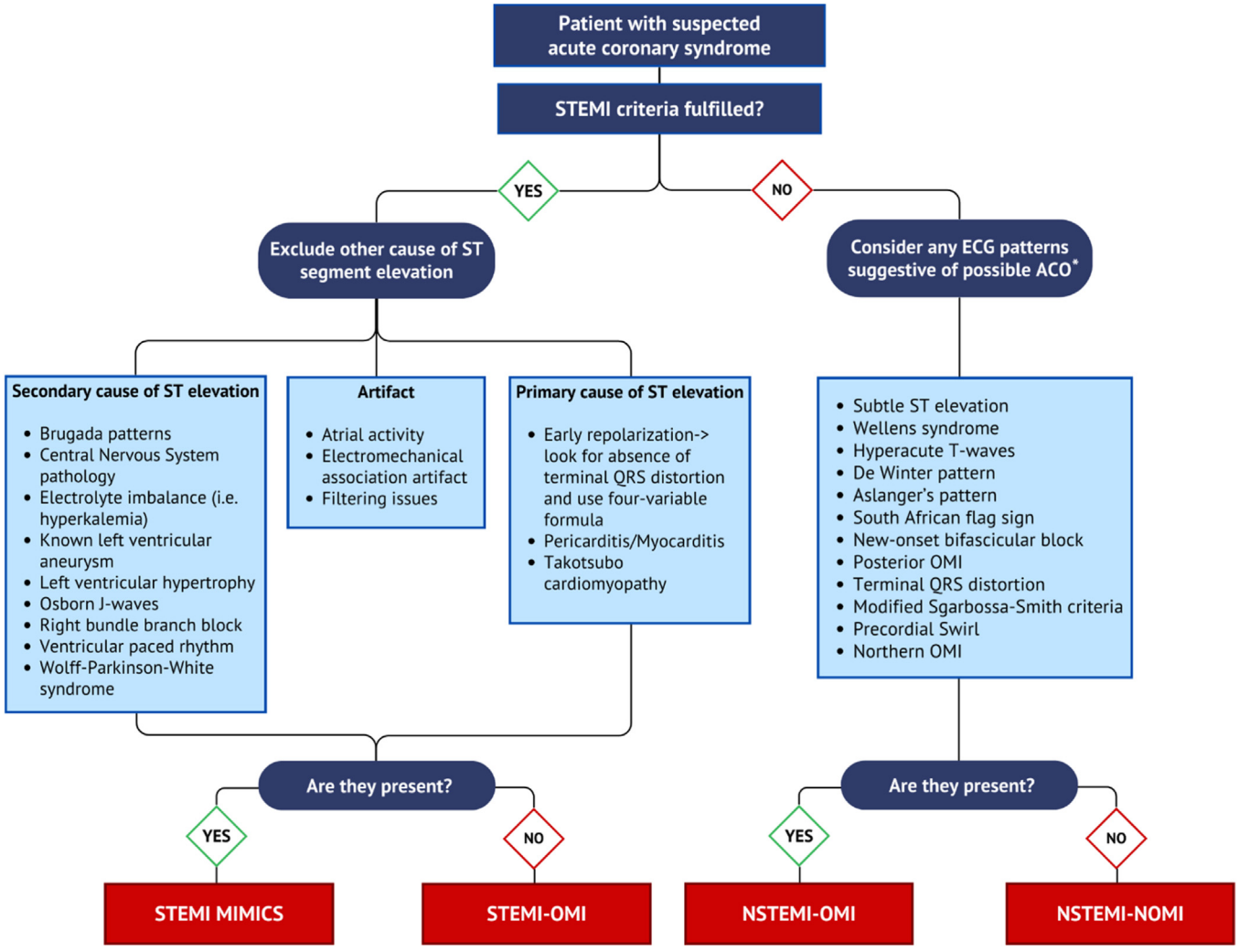

Figure 2. The OMI pathway. *Always consider dynamic ECG assessment, compare with prior ECGs, correlate with symptoms and serial troponin concentrations, rule-out mimics, double-check ST-T changes in lead aVL (Figure E11, available at http://www.annemergmed.com) and differentiate ST elevation of normal variant in V2-V4 (Figure E12, available at http://www.annemergmed.com). Modified from Aslanger et al.[12,70] *ACO*, Acute coronary occlusion; *NOMI*, non-occlusion myocardial infarction; *OMI*, occlusion myocardial infarction.

Although randomized controlled trials substantiate early coronary intervention for STEMI, equivalent evidence is lacking for occlusion myocardial infarction. This reliance on nonrandomized data must be acknowledged, and future research should aim to address this gap. Nevertheless, several randomized trials have demonstrated that patients with NSTEMI with persistent symptoms, which encompass most occlusion myocardial infarction cases, should receive emergent reperfusion, translating into significantly better outcomes.[58-66] Although refractory ischemia was an exclusion criterion in most of these trials, which take place in research settings, less than 10% of patients in the community with very high risk of acute coronary syndrome, which includes persistent chest pain, undergo guideline-recommended less than 2-hour angiography.[19]

Several conditions that present with ST-segment elevation mimic STEMI-occlusion myocardial infarction, thereby reducing the specificity of STEMI millimeter criteria as a robust diagnostic indicator. A significant number of cases showing at least 1 mm of ST-segment elevation in the context of undifferentiated chest pain stem from various other conditions such as early repolarization, left ventricular hypertrophy, acute pericarditis, myopericarditis, Takotsubo cardiomyopathy, left bundle branch block, and ventricular paced rhythm. These ECG presentations can lead to false positives, resulting in unnecessary activations of the catheterization laboratory. Proper identification of these patterns is crucial not only to ensure that patients with actual acute coronary occlusion receive timely coronary angiography but also to minimize instances of unwarranted urgent cath lab activations. Although a plethora of ECG criteria have been proposed to differentiate occlusion myocardial infarction from mimics, their adoption can be limited due to their complexity and unclear interobserver reliability.[18,67]

Implementing the occlusion myocardial infarction pathway requires tackling these diagnostic challenges, fostering accurate recognition of occlusion myocardial infarction pattern mimics, and establishing a clear roadmap for interdisciplinary collaboration focused on timely reperfusion goals. The natural history of occlusion myocardial infarctions is not as well documented as that of STEMI, where extensive data on the evolutionary pattern of ECGs exist. Furthermore, to our knowledge, no studies have systematically collected standard 12-lead ECGs at the exact time of coronary angiography. This emphasizes the need for targeted clinical research to test the correlation between distinct ECG patterns and angiography findings during acute coronary events and after reperfusion to support the recognition of STEMI-equivalent ECG patterns and identifying reliable ECG markers of poor myocardial reperfusion. Finally, the specificity of occlusion myocardial infarction patterns in varied clinical contexts is unclear, which is crucial for their practical application, and operational challenges in using these patterns in emergency settings require further exploration. Future research should prioritize external validation of distinct occlusion myocardial infarction patterns, evaluating their diagnostic accuracy and prognostic value across diverse patient populations who have an ECG recorded. Artificial intelligence shows promise in overcoming these challenges, because it has shown high accuracy in ECG diagnosis of occlusion myocardial infarction.[47,68,69]

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