



Reevaluating STEMI: The Utility of the Occlusive Myocardial Infarction Classification to Enhance Management of Acute Coronary Syndromes

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Accepted: 7 February 2025
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Abstract

Background The current classification of acute myocardial infarction (AMI) into ST-segment elevation myocardial infarction (STEMI) and non-ST-segment elevation myocardial infarction (NSTEMI) has limitations in identifying patients with acute coronary occlusion (ACO) who do not exhibit classic ST-elevation. Emerging evidence suggests that a reclassification to "Occlusive Myocardial Infarction" (OMI) may enhance diagnostic accuracy and therapeutic interventions.

Methods A comprehensive review of the literature was conducted, focusing on the pathophysiology, electrocardiographic (EKG) patterns, and management of ACO. The utility of the OMI paradigm was evaluated against the traditional STEMI/NSTEMI framework, with a particular emphasis on atypical EKG findings and their role in guiding early intervention.

Results Traditional STEMI criteria fail to identify ACO in approximately 30% of NSTEMI patients, leading to delayed reperfusion and increased mortality. The OMI framework demonstrates improved sensitivity (78.1% vs. 43.6% for STEMI criteria) for detecting ACO by incorporating subtle EKG changes, including hyperacute T-waves, de Winter T-waves, and posterior infarction patterns. OMI-guided management facilitates timely diagnosis and intervention, potentially reducing adverse outcomes. Emerging artificial intelligence (AI) tools further enhance EKG interpretation and clinical decision-making.

Conclusions Transitioning to the OMI paradigm addresses critical gaps in the STEMI/NSTEMI framework by emphasizing the identification of ACO irrespective of ST-segment elevation. This approach could significantly improve patient outcomes by reducing delays in reperfusion therapy. Future randomized trials are needed to validate the OMI paradigm and optimize its implementation in clinical practice.

Keywords Occlusive myocardial infarction · Acute coronary syndrome · Electrocardiography · ST-segment elevation · Coronary occlusion

Abbreviations

ACO	Acute Coronary Occlusion
AI	Artificial Intelligence
AMI	Acute Myocardial Infarction
ACS	Acute Coronary Syndrome
AVR	Augmented Vector Right (EKG lead)
CCTA	Coronary Computed Tomography Angiography
D1	First Diagonal Branch of the Left Anterior Descending Artery
EKG	Electrocardiogram
GRACE	Global Registry of Acute Coronary Events
LAD	Left Anterior Descending Artery
LMCA	Left Main Coronary Artery
LV	Left Ventricle
LBBS	Left Bundle Branch Block
MACE	Major Adverse Cardiovascular Events

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MI	Myocardial Infarction
NSTEMI	Non-ST-Segment Elevation Myocardial Infarction
NSTEACS	Non-ST-Segment Elevation Acute Coronary Syndrome
OMI	Occlusive Myocardial Infarction
OCA	Occluded Culprit Artery
PCI	Percutaneous Coronary Intervention
QRS	A specific wave pattern in an EKG
REF	Reference (used for studies)
STE	ST-Segment Elevation
STD	ST-Segment Depression
STEMI	ST-Segment Elevation Myocardial Infarction
TTE	Transthoracic Echocardiogram
TIMI	Thrombolysis in Myocardial Infarction

Introduction

The classification of myocardial infarction (MI) has evolved significantly over the past few decades, driven by advances in understanding of coronary pathophysiology. Historically, MI was divided into two major categories based on electrocardiogram (EKG) findings; Q-wave and non-Q-wave infarctions [1]. Q-wave MI was associated with more extensive myocardial damage and believed to be a harbinger of worse clinical outcomes, while non-Q-wave MI, was thought to represent a less severe form [2–4]. However, this simple classification method proved insufficient for guiding therapeutic decisions as understanding of ischemic heart disease progressed. This historical framework was then replaced by the current paradigm, using the nomenclature ST-segment elevation myocardial infarction (STEMI) and non-ST-segment elevation myocardial infarction (NSTEMI) [5–7]. The shift was based on the understanding that ST-segment elevation is a key marker of acute and complete coronary artery occlusion signaling an urgent need for revascularization to restore blood flow and limit myocardial muscle damage [8]. NSTEMI is thought to be typically caused by partial occlusion or reduced blood flow, at times permitting more conservative approach to treatment [9, 10]. This evolution in MI classification has helped define appropriate timing of treatment strategies in patients with acute coronary syndrome (ACS).

The mainstay of treatment is reperfusion of acute coronary occlusion (ACO) which has been shown to significantly reduce mortality and morbidity in patients who present with acute myocardial infarction (AMI) [11]. The only placebo-controlled trials of reperfusion therapy were conducted in the era of thrombolytic therapy, whereby EKGs were classified as having ST-segment Elevation (STE), ST-segment Depression (STD), or neither. A large meta-analysis of these trials revealed that STE was associated

with reduced mortality from thrombolytic therapy [11]. However, these trials lacked angiographic confirmation and STE was also poorly characterized. Despite these limitations, STE became a surrogate marker for ACO, leading to the classification of STEMI for cases requiring emergent reperfusion in patients with AMI. No further interventional trials have examined the relationship between STE—or other EKG findings and reperfusion for ACO. Nevertheless, the STEMI vs. NSTEMI paradigm has persisted, and international guidelines continue to use "STEMI" as a surrogate for acute coronary occlusion myocardial infarction. Unfortunately, under this classification, approximately 25–30% of NSTEMI cases involve unrecognized ACO which are only discovered on delayed angiography, typically performed around 24 h after presentation. These patients have approximately double the risk of short and long term mortality rates compared to NSTEMI patients without occlusion [12]. Conversely, 15–35% of catheterization laboratory activations due to perceived STEMI criteria are later found to be false positives, with no identifiable blockages [13, 14]. Another critical consideration is that identifying a coronary occlusion long after the event and subsequent intervention provides no clinical benefit to the patient. Therefore, there is a narrow therapeutic window, making timely diagnosis essential for appropriate intervention, as demonstrated by the OAT trial [15].

Emerging evidence suggests that the current classification may miss patients with ACO who do not exhibit classic STE but still benefit from urgent intervention. Studies have shown that many patients with OMI have subtle or atypical EKG patterns, such as hyperacute T-waves, terminal QRS distortion, and low QRS amplitude, which are not recognized under traditional STEMI criteria [16]. In a recent study, Aslanger et al. reclassified 28% of NSTEMIs as OMI by using structured EKG criteria, identifying a subset of patients with coronary lesions and outcomes similar to those seen in STEMI [17]. Some OMI cases may present with no EKG abnormalities at all, requiring a combination of clinical suspicion, ongoing symptoms, biomarker elevation, echocardiography, or coronary computed tomography angiography (CCTA) to establish the diagnosis [18, 19]. Opposition to the OMI/NOMI classification often references studies suggesting that early angiography in NSTEMI patients does not improve outcomes. However, many of these studies excluded patients with persistent symptoms or did not implement very early intervention [20–24]. For instance, in one large study, patients with persistent symptoms were excluded, and "early" angiography occurred at an average of 16 h from presentation; despite this, patients with high-risk features, such as GRACE (Global Registry of Acute Coronary Events) score greater than 140, did benefit from earlier reperfusion [25]. In studies that did not exclude symptomatic patients and

involved truly early intervention, outcomes were better for those randomized to early angiography.

Consequently, international guidelines for NSTEMI recognize the limitations of the current randomized clinical trials and recommend emergent angiography for patients with symptoms highly suspicious for ACS and ongoing instability, even in the absence of definitive EKG or biomarker evidence of AMI [26, 27]. These recommendations reflect the understanding that acute coronary occlusion, rather than specific EKG millimeter criteria, is the underlying pathology warranting emergent reperfusion. This is further supported by a retrospective cohort study by Smith et al., which found that STEMI (-) OMI patients experienced significant delays in diagnosis and reperfusion yet had outcomes similar to those of STEMI (+) OMI patients [27]. Despite the significantly higher mortality of missed occlusions in NSTEMI patients, no randomized trial has directly addressed this issue due to ethical concerns regarding delayed treatment.

Hence, we propose moving toward the nomenclature "Occlusive Myocardial Infarction" (OMI) to more accurately reflect the pathophysiology of coronary occlusion and better guide therapeutic decisions. Herein we evaluate the potential benefits of transitioning to the OMI classification system. Specifically, we will focus on identifying the EKG patterns associated with OMI and assess whether this approach could improve the timely recognition and management of acute coronary syndromes compared to the current STEMI/NSTEMI distinction. By doing so, we aim to shed light on a paradigm that could potentially enhance clinical outcomes for patients who may benefit from expedited reperfusion therapy and are currently being missed under the STEMI/NSTEMI classification.

Strengths and Limitations of the Current STEMI/NSTEMI Paradigm

The current STEMI/NSTEMI paradigm has several strengths that have led to an improved patient care in acute myocardial infarction. One of its primary advantages is the ability to differentiate between patients requiring immediate reperfusion therapy and those who may benefit from more conservative or delayed management. This distinction ensures that patients with a STEMI with ACO are rapidly identified and treated with percutaneous coronary intervention (PCI) or fibrinolytic therapy, reducing time to reperfusion, and improving survival outcomes [28–30]. This framework has also facilitated efficient decision-making in emergency settings, enabling clinicians to triage patients based on EKG findings and deliver timely interventions, thereby standardizing care across healthcare systems [5, 31].

Despite its strengths, the STEMI/NSTEMI classification has notable limitations. Its reliance on ST-segment elevation as a marker for ACO excludes many patients with coronary occlusion but without this EKG finding, resulting in

misclassification as NSTEMI [32–34]. These patients, who may require urgent revascularization, often receive more conservative management, leading to delays in reperfusion and increased myocardial damage. Additionally, the current STEMI/NSTEMI paradigm may overlook a subset of high-risk patients, highlighting the need for a more refined classification system to better capture these cases.

The refinement of an OMI Framework

The proposed OMI framework addresses several of the limitations of the current STEMI/NSTEMI paradigm. One of its key advantages is the improved sensitivity and specificity for detecting acute coronary occlusion [35, 36]. By focusing on the presence of total coronary obstruction rather than the strict adherence to ST-segment elevation, the OMI classification can capture a broader spectrum of patients who might otherwise be misclassified as NSTEMI. This approach is especially beneficial in recognizing high-risk patients who present with non-traditional or subtle EKG findings such as isolated ST-segment depressions, deep T-wave inversions, or hyperacute T-waves which may still signify significant coronary occlusion [37].

Emerging evidence suggests that the OMI framework could lead to better patient outcomes. Studies have demonstrated that patients with acute coronary occlusion who do not exhibit ST-segment elevation still face substantial risks of myocardial damage if timely intervention is delayed. Early recognition and intervention in these patients, made possible by the OMI, could reduce reperfusion delays and consequently improve clinical outcomes [38].

EKG Patterns Suggestive of OMI

Acute coronary syndrome is divided into two main categories: STEMI and non-ST segment elevation acute coronary syndrome (NSTEMI), as determined by initial 12-lead EKG. Current guidelines recommend early reperfusion therapy for STEMI patients as a class I indication [5]. In contrast, NSTEMI encompasses a range of conditions, including unstable angina (without cardiomyocyte damage) and NSTEMI (with cardiomyocyte necrosis). Management of NSTEMI relies on risk stratification, favoring an early invasive approach for high-risk patients, particularly those with elevated cardiac necrosis biomarkers [27, 39].

While STEMI typically results from acute total or near-total occlusion of a coronary artery, instances of complete occlusion of the culprit artery have also been reported in NSTEMI patients. Large retrospective studies indicate that up to 30% of patients with NSTEMI have a totally occluded culprit artery (OCA) [40–42]. One explanation

for the presentation of OCA as NSTEMI rather than STEMI is the limited sensitivity of the EKG in detecting acute ischemia or infarction, particularly in the posterior or lateral walls, where the left circumflex artery is often implicated as the culprit [42]. Furthermore, large meta-analyses of non-STEMI patients have revealed that approximately 25% exhibit a completely occluded artery, while 34% show a Thrombolysis in Myocardial Infarction (TIMI) flow grade of 0 to 1 [12, 34]. Notably, these patients experience nearly double the mortality rate compared to non-STEMI patients without OMI, despite being younger and having fewer comorbid conditions [34].

A report addressing the diagnosis of STEMI in the emergency department highlighted the difficulties associated with interpreting EKGs, particularly in cases of borderline ST elevation. It emphasized the need to distinguish STEMI from other conditions such as early repolarization, pericarditis, left bundle branch block, and old myocardial infarction characterized by persistent ST elevation [43]. Additionally, dichotomizing EKGs based solely on the presence or absence of ST elevation limits the ability of STEMI criteria to differentiate between various types of ST elevation. This approach also fails to recognize ST elevation that may be secondary to an abnormal QRS complex combined with underlying primary ischemic elevation [34].

Atypical Electrocardiographic Patterns in Acute Coronary Occlusion

Within the STEMI framework, cardiologists and emergency physicians have identified occlusion and reperfusion patterns that do not conform to traditional STEMI classifications [44]. These include Wellens' syndrome, characterized by T-wave inversion associated with a risk of reocclusion; de Winter T waves, indicative of left anterior descending (LAD) artery occlusion; the "South African flag sign," which helps identify first diagonal artery occlusion; and the Aslanger pattern, which represents inferior occlusion with concurrent high-grade stenosis. Additionally, ischemic ST depression, particularly in leads V1 to V4, can signal posterior occlusion myocardial infarction and aid in distinguishing it from subendocardial ischemia [34]. Table 1 showcases Essential EKG patterns in the OMI paradigm in more details.

Wellen's Syndrome

Wellen's syndrome is characterized by distinct EKG findings that emergency physicians must recognize, as a significant proportion of patients may progress to anterior wall myocardial infarctions if timely intervention is not initiated. Urgent coronary angiography is warranted for patients with this EKG finding [45]. Quick recognition of these classic T-wave changes can help prevent acute myocardial

infarction, particularly in patients without a known history of cardiovascular disease or unmitigated risk factors.

De Winter T Waves

Similarly, the de Winter EKG pattern is recognized as an anterior STEMI equivalent, primarily characterized by upsloping ST-segment depression at the J point in leads V1–V6, accompanied by tall, symmetrical T waves. This pattern is observed in approximately 2% of patients with subtotal or total occlusion of the proximal LAD artery [46]. Patients presenting with this pattern are experiencing OMI and require immediate reperfusion therapy [47]. Nikus et al. later reported a case of acute myocardial infarction resulting from sub-occlusion of the left circumflex artery, which presented with a similar EKG pattern of upsloping ST-segment depression [48]. It has been noted that patients exhibiting this EKG pattern may progress to typical ST elevation myocardial infarction [49]. A consensus document advised that evidence of this pattern in a patient with suggestive ongoing symptoms and without tachycardia should prompt urgent reperfusion therapy via PCI [49]. The findings discussed above suggest a correlation between upsloping ST-segment depression and myocardial ischemia, but it is essential to recognize that this association is valid only within the appropriate clinical context [50].

ST Depression in Anterior Precordial Leads as an Indicator of Posterior AMI

Acute posterior wall MI accounts for approximately 15% to 21% of all acute MIs, predominantly occurring alongside acute infarction of the inferior or lateral walls of the left ventricle. Detecting acute posterior wall MI presents challenges, as the standard 12-lead EKG inadequately visualizes the posterior wall of the left ventricle [51]. Furthermore, the electrocardiographic criteria indicative of acute posterior wall MI are not well-known among practitioners. The lack of evident ST elevation often leads to missed diagnoses, making posterior wall MI one of the most commonly overlooked patterns of acute infarction in electrocardiographic assessments [52].

Several electrocardiographic findings indicative of acute posterior wall MI can be observed in the 12-lead EKG, particularly in leads V1, V2, or V3. These findings include: 1) horizontal ST segment depression, 2) a tall, upright T wave, 3) a tall, wide R wave, and 4) an R/S wave ratio greater than 1.0 in lead V2 [53]. Additionally, the presence of both horizontal ST segment depression and an upright T wave enhances the diagnostic accuracy of these electrocardiographic findings [53]. Therefore, adopting the OMI paradigm, which focuses on the presence or absence of acute coronary occlusion rather than solely relying on ST elevation

Table 1 Essential EKG patterns in the OMI paradigm, detailing characteristic findings, implicated coronary occlusions, and recommended clinical actions

EKG Pattern	Characteristics	Indicative of	Clinical Implications
STEMI (+) OMI			
ST Elevation	STE in two contiguous leads	Classical STEMI diagnosis	Urgent reperfusion therapy, high-risk ACS
aVR STE with Multi-lead Depression	STE in aVR with ST depression in other leads	Severe left main or LAD disease or multi-vessel ischemia	Indicates high-risk patients; urgent intervention required
Shark Fin Sign	Large, wide, and rounded ST-segment elevations that appear as a 'shark fin' pattern	Severe coronary occlusion, often with extensive infarct area	Seen in severe ischemia. Requires urgent reperfusion
Transient STEMI	Brief episodes of ST elevation that resolve spontaneously or with minimal intervention; may lack clear obstructive findings	Transient occlusion or severe spasm without permanent damage	Observation may be needed; recurrent episodes increase risk
South African Flag Sign	STE in noncontiguous leads V2 and aVL with inferior reciprocal ST depression	First diagonal artery occlusion	Requires prompt recognition and intervention
Aslanger Pattern	STE in III with reciprocal STD in I/aVL, with V1 > V2 and ST depression in V5–V6	Inferior OMI with concurrent critical stenosis	Suggests high risk inferior territory occlusion, warranting urgent intervention
Smith-Modified Sgarbossa Criteria	STE concordance or discordant STE/S ratio > 25% in LBBB or ventricular pacing	OMI in cases of LBBB or ventricular paced rhythm	Sensitive marker for occlusion in complex EKG cases, improving diagnostic accuracy
Right Ventricular Infarction	STE in V1, STE in V1 with ST depression in V2, or ST elevation in III > II; confirmed by ST elevation in right-sided leads (V3R–V6R)	Complicates up to 40% of inferior STEMIs; isolated RV infarction is rare	Patients are preload-sensitive and may develop severe hypotension with nitrates. Treated with fluids; nitrates contraindicated
STEMI (-) OMI			
Wellens' Syndrome	Deep, symmetric T-wave inversion in V2–V3; occurs without STE	LAD artery occlusion, often before infarction	Requires early angiography
de Winter Pattern	Upsloping ST depression at the J point with tall, symmetrical T-waves in precordial leads	Anterior STEMI equivalent, usually LAD occlusion	Urgent reperfusion therapy
Posterior MI	Horizontal ST depression, upright T wave, and tall, wide R wave in V1–V3; R/S ratio > 1 in V2	Occlusion affecting posterior wall, often with inferior or lateral MI	Urgent reperfusion therapy necessitated. Posterior EKG leads may assist in diagnosis
Hyperacute T Waves	Broad, tall T waves that precede STE in early occlusion stages	Early occlusive event in any coronary artery	Prompt identification needed to initiate early intervention
Fragmented QRS Complex	Multiple small notches within QRS complex, typically in ≥ 2 contiguous leads	Marker of ischemic damage, often linked to multivessel disease	Higher risk of ventricular arrhythmias, MACE, and heart failure
Primary STD in aVL	Primary STD in aVL reciprocal to inferior hyperacute T waves	RCA occlusion with inferior ischemia	High specificity for RCA occlusion, useful for differentiating inferior OMI from pericarditis
Early Q Waves	Development of early Q waves, often with hyperacute T waves in the same region	LAD occlusion, often anterior OMI	Early indicator of transmural myocardial infarction, requiring urgent intervention

STE ST Segment Elevation, LAD Left Anterior Descending artery, ACS Acute Coronary Syndrome, MI Myocardial Infarction, aVR Augmented Vector Right lead on EKG, MACE Major Adverse Cardiovascular Event

on EKG, could enhance the detection of cases requiring interventions for acute coronary occlusion, even when ST elevation is absent.

ST Elevation in AVR Lead (LMCA/Proximal LAD Occlusion or Multivessel Disease)

The American Heart Association guidelines maintain that STE must be present in at least two contiguous leads for a STEMI diagnosis. However, the 2013 update introduced the recognition of isolated STE in the augmented vector right (aVR) lead, accompanied by multi-lead ST depression, as a potential indicator of STEMI due to left main coronary artery (LMCA) or proximal LAD occlusion [5]. Previous studies have identified two primary clinical scenarios associated with ST elevation in lead aVR. The first scenario involves total occlusion of the LMCA or the proximal LAD, where STE in lead aVR indicates ischemia in the basal interventricular septum and is not considered reciprocal [54]. Timely reperfusion in these cases is essential for a favorable outcome. The second scenario occurs in patients with severe multivessel disease without acute occlusion, resulting in subendocardial ischemia that produces ST segment depression in the inferolateral leads alongside reciprocal elevation in aVR [55–57]. This group is considered high-risk within the acute coronary syndrome population. However, it is important to note that a study found only 10% of patients with ST segment elevation in lead aVR have an acutely occluded coronary artery [53].

Fragmented QRS Complexes and Hyperacute T Waves

Other EKG findings indicative of coronary occlusion that do not present as ST elevation include hyperacute T waves and fragmented QRS complexes. Fragmented QRS complexes are multiple small notches within QRS complex, typically in ≥ 2 contiguous leads. They are significantly associated with both in-hospital and long-term mortality, as well as major adverse cardiovascular events (MACE) in patients with acute myocardial infarction. They are also positively correlated with a higher incidence of triple vessel coronary artery lesions [58]. Additionally, they are linked to an increased risk of ventricular arrhythmias and heart failure, potentially serving as a marker for mortality and MACE risk [58]. Hyperacute T waves typically appear shortly after the onset of coronary occlusion and transmural infarction, evolving rapidly into STE [59].

Shark Fin Sign

Also referred to as triangular QRS-ST-T waveform, lambda-wave, or giant R waves. It appears as a giant R wave with an

amplitude of > 1 mV and a QRS complex fused with the ST-segment and T-wave. This pattern is linked to massive myocardial ischemia, particularly resulting from LAD or LMCA occlusion [60, 61]. Due to its rarity, occurring in only 1.7% of STEMI cases, the shark fin pattern demands high clinical suspicion and prompt intervention. It is also associated with a significant risk of atrial and ventricular fibrillation, contributing to higher in-hospital mortality rates [62].

South African-Flag Pattern

This sign signifies occlusion of the first diagonal branch (D1) of LAD. It manifests as ST-elevation in leads I, aVL and V2 along with ST-depression in lead III and inferior leads resembling the south African flag shape [63, 64].

Left Bundle Branch Block (LBBB) with Smith-Modified Sgarbossa Criteria

A diagnosis of ACO can be difficult to make in patients with LBBB due to appropriate discordance in which an abnormal depolarization (i.e., ST-segment and T-wave deviation) is followed by an abnormal repolarization which are not necessarily secondary to coronary artery occlusion. However, serial ECG can be helpful in such situation while taking into account Smith-Modified Sgarbossa Criteria which have been developed to improve the sensitivity and specificity of ECG in the detection of ACO in LBBB [65]. They diagnose ACO in patients LBBB depending on the presence of one of the following: (1) concordant ST-elevation > 1 mm in one lead or more (5 points), (2) concordant ST-depression > 1 mm in one lead or more of V1-V3 (3 points) or (3) Proportionally excessive discordant ST-elevation in any lead with ≥ 1 mm STE as defined by $\geq 25\%$ of the depth of the preceding S-wave (2 points) [60]. Those criteria have relatively low sensitivity but are highly specific (with 90% specificity for a score of 3 or more); making them a very useful tool when encountering patients with LBBB in appropriate clinical scenarios with high suspicion for ACO.

Transient STEMI

While presenting with typical symptoms for ACO and having ST-changes typical for STEMI, patients with this clinical entity have spontaneous resolution of symptoms and electrocardiographic changes prior to reperfusion therapy and represent up to 24% of STEMI cases [66]. In a study by Meisel et al., patients with transient STEMI were found to have resolution of symptoms and electrocardiographic changes in 1.2 ± 0.8 h after presentation. While the exact approach for managing this condition is not yet standardized, some evidence suggest that early medical treatment followed by invasive reperfusion therapy is recommended

[67]. Fortunately, patients with transient STEMI generally exhibit a more favorable prognosis compared to those with persistent STEMI, potentially due to factors such as spontaneous recanalization of the infarct-related artery or transient epicardial coronary artery spasm [66].

ST-Segment Depression in Lead aVL

Differentiating inferior STEMI from pericarditis represents a common and significant clinical dilemma for physicians. A retrospective cohort study involving 154 patients with inferior STEMI and 49 patients with pericarditis examined the diagnostic value of ST-depression in lead aVL in distinguishing between these two conditions. The study found that ST-depression in lead aVL is 100% specific for diagnosing ACO due to inferior STEMI over pericarditis [68].

Old Q waves versus New STEMI

Another conundrum that is faced in clinical practice is the presence of Q waves with concurrent ST-elevation in the anterior leads which can muddy the distinction between acute anterior STEMI and left ventricular (LV) aneurysm [69]. A useful tool was developed by Smith et al. in which the ratio of highest T/QRS amplitudes in leads (V1-V4) is measured. If this ratio is shown to be more than 0.36, a diagnosis of STEMI can be made with 90% accuracy [70].

Clinical Implications

In the STEMI/NSTEMI framework, relying solely on the standard 12-lead EKG may lead to the omission of patients with ACO, as STEMI criteria alone are a poor surrogate for ACO [71, 72]. One major challenge is the limited sensitivity of 12-lead EKGs in detecting ACO. A systematic review and meta-analysis found the sensitivity of EKG for detecting ACO in STEMI patients was only 43.6%, implying more than half of cases of ACO had no ST-elevation [73].

Occlusive MI Framework Implementation: Clinician Training and EKG Interpretation

Implementing the OMI paradigm shift requires enhancing clinician education, awareness, and training in EKG interpretation. Educational programs are essential to increase EKG determination accuracy. A systematic review and meta-analysis highlighted that physicians' accuracy in EKG interpretation varies widely, with a median accuracy of 54% pre-training and 67% post-training, leaving substantial room for enhancement [74]. Furthermore, EKG pattern interpretation is largely individual dependent and thus prone

to misinterpretation and human error. The American Heart Association emphasizes the importance of ongoing training for physicians and nurses in EKG interpretation [75]. In addition, it is essential to properly educate residents on identifying specific EKG changes that can suggest ACO (including hyperacute T waves, de Winter's sign, or Wellens' syndrome). The American College of Cardiology cautions clinicians to pay attention to EKG changes that are harder for the human eye to detect, which may signal early vessel occlusion and necessitate emergent coronary angiography [71].

McLaren et al. stress the significance of quality improvement programs using standardized feedback and case discussions that will ultimately accelerate ACO identification and treatment without increasing false activation [76]. Furthermore, a new artificial intelligence tool (i.e., Queen of Hearts) has been developed to detect coronary artery occlusion regardless of the presence of ST-elevation with promising performance at 94% specificity and 81% sensitivity [77].

Changes in Sensitivity and Specificity for MI detection

The effective application of OMI criteria to patients with acute MI has important consequences on both the sensitivity and specificity for detecting MI as well as long-term patient events. Patients who meet OMI criteria, especially those with NSTEMI, but have coronary occlusion that needs to be treated urgently, otherwise portend poor long-term results. Studies have shown that these patients are at similar risk of morbidity and mortality if catheterization is delayed as those with STEMI [35].

The OMI criteria enhance the ability to detect acute coronary occlusions compared to the traditional STEMI criteria. A systematic review and meta-analysis by de Alencar Neto et al. found that while STE alone has a sensitivity of about 43.6% for identifying acute coronary occlusion, the OMI criteria raise this sensitivity to 78.1% without a notable impact on specificity. This improved sensitivity decreases false negative data and is crucial for promptly identifying patients who need immediate reperfusion therapy [73]. Furthermore, the OMI paradigm enhances the detection of coronary occlusion in patients who do not show the typical ST elevation. For example, significant ST-segment depression in leads V1-V4 has a high specificity of 97% for identifying OMI, even without ST elevation. This approach helps reduce the number of cases that might go undetected by traditional STEMI/NSTEMI criteria [38].

The OMI criteria are designed to enable earlier and more accurate diagnosis of ACO. Clinicians can recognize coronary occlusion earlier by detecting minor changes in EKG as

well as utilizing sophisticated diagnostics such as machine learning models that improve patient health prospects through faster initiation of treatment [78, 79].

Impact of the OMI Paradigm Implementation on Management and Treatment

The OMI paradigm simply asserts all patients with ACO need immediate activation/intervention, irrespective of classic ST-segment elevation. The idea of this approach is that patients with high-risk EKG findings (subtle ST-segment changes or hyperacute T waves) be sent for early coronary angiography, to receive timely and appropriate reperfusion therapy [35]. Thus, the implementation of the OMI paradigm requires updating clinical protocols and guidelines. This requires robust revision of the recommendations provided by the American College of Cardiology and other societies to include OMI-specific criteria that would facilitate rapid invasive revascularization for patients with acute coronary occlusions as is already recommended in those with STEMI.

Even with the development of high-sensitivity troponin tests, EKG interpretation remains essential. Proper EKG interpretation, particularly noticing subtle changes that suggest OMI, is key to making a timely diagnosis and initiating treatment [80].

Patients with acute coronary occlusion who fail to meet conventional STEMI criteria yet are identified by OMI criteria share a similar adverse outcome as their “STEMI” cohorts when revascularization is delayed. It may be required to reduce mortality and morbidity because they could ensure that no ACO patient is admitted to a hospital without being treated with reperfusion therapy [80]. All that being said, the lack of randomized clinical trial validation of the OMI paradigm in comparison to the STEMI paradigm represents a significant limitation for the applicability of such paradigm.

Future Directions

The literature review uncovers important details and pitfalls in the STEMI/NSTEMI approach to treating ACO. Although traditional STEMI/NSTEMI criteria are widely used, they often fail to identify patients with ACO who lack the classic ST-elevation pattern. This highlights the potential value of detecting occlusions through more subtle EKG changes, as captured by the OMI rule, which may be missed by traditional criteria in real-world clinical practice [81].

Existing evidence suggests that dependence on the STEMI/NSTEMI dichotomy alone to characterize ACS will fail to diagnose and treat many patients in an expedited fashion, resulting in worse outcomes associated with delayed treatment.

The OMI criteria could aid clinicians to recognize the non-classical EKG patterns which are often under-recognized by current clinical guidelines. Incorporating these subtle EKG findings into daily practice may result in more accurate and timely anterior wall AMI diagnoses [82]. A promising advancement in this area is the use of AI to augment clinical decision-making. Software platforms like PowerfulMD, Queen of Hearts, and similar AI-driven tools are increasingly capable of detecting subtle EKG patterns that may be challenging for even the most experienced clinicians to recognize. These AI systems can assist in identifying non-classical signs of ACO, potentially improving early diagnosis. As AI technology evolves, it holds the potential to significantly enhance diagnostic accuracy and streamline clinical workflows, ensuring more timely and precise interventions for patients at risk [35].

There is considerable potential for further research and education on non-classical EKG patterns suggesting the presence of ACO. Patients recognized through OMI criteria, especially those with NSTEMI-ACO, appear to be at high risk of adverse events similar to STEMI and should not experience delays in reperfusion. Research has shown that this group faces increased odds of mortality and delays in catheterization procedures. Early recognition and intervention according to the OMI criteria could substantially improve long-term patient outcomes by guaranteeing timely reperfusion therapy [12, 35]. Randomized controlled trials comparing the STEMI and OMI paradigms are mandatory to validate the benefits of the latter approach for earlier recognition and management of ACO.

Key References

- Kola M, Shuka N, Meyers HP, Zaimi (Petrela) E, Smith SW. OMI/NOMI: Time for a New Classification of Acute Myocardial Infarction. *J Clin Med.* 2024;13(17):5201. <https://doi.org/10.3390/jcm13175201>.
 - This article proposes a paradigm shift from STEMI/NSTEMI classification to Occlusion MI (OMI) and Non-Occlusion MI (NOMI), addressing the limitations of traditional EKG interpretations in acute coronary syndrome.
- McLaren J, De Alencar JN, Aslanger EK, Meyers HP, Smith SW. From ST-Segment Elevation MI to Occlusion MI. *JACC Adv.* 2024;3(11):101,314. <https://doi.org/10.1016/j.jacadv.2024.101314>.
 - This paper highlights the clinical implications of misclassifying occlusive myocardial infarctions under the STEMI paradigm, advocating for the adoption of OMI to improve diagnostic accuracy and patient outcomes.

- de Alencar Neto JN, Scheffer MK, Correia BP, Franchini KG, Felicioni SP, De Marchi MFN. Systematic review and meta-analysis of diagnostic test accuracy of ST-segment elevation for acute coronary occlusion. *Int J Cardiol.* 2024; 402:131,889. <https://doi.org/10.1016/j.ijcard.2024.131889>.

○ This meta-analysis comprehensively evaluates the diagnostic accuracy of ST-segment elevation in identifying acute coronary occlusion, underscoring the need for more refined criteria to enhance clinical decision-making.

Acknowledgments The authors declare no specific acknowledgments for this manuscript.

Author Contributions MA1 contributed to the design of the study, data analysis, data interpretation, and final drafting of the manuscript. MA2, DG, BF, MB, and JP contributed to design of the study, data collection, data entry, and initial manuscript drafting. JA contributed to the design of the study, data interpretation, drafting of the manuscript, and supervision of the work. All authors have reviewed and given their approval for the final version of the manuscript. Each author has been actively involved in the work and is willing to take full responsibility for its content.

Funding The authors have not received any funds for this manuscript, and thus have none to declare.

Data Availability No datasets were generated or analysed during the current study.

Compliance with Ethical Standards

Competing Interests The authors declare no competing interests.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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References

1. Keller KB, Lemberg L. Q and non-Q wave myocardial infarctions. *Am J Crit Care Off Publ Am Assoc Crit-Care Nurses.* 1994;3(2):158–61.
2. Nicod P, Gilpin E, Dittrich H, et al. Short- and long-term clinical outcome after Q wave and non-Q wave myocardial infarction in a large patient population. *Circulation.* 1989;79(3):528–36. <https://doi.org/10.1161/01.cir.79.3.528>.
3. Kim HW, Klem I, Shah DJ, et al. Unrecognized Non-Q-Wave Myocardial Infarction: Prevalence and Prognostic Significance in Patients with Suspected Coronary Disease. *PLoS Med.* 2009;6(4):e1000057. <https://doi.org/10.1371/journal.pmed.1000057>.
4. Saleh M, Ambrose JA. Understanding myocardial infarction. *F1000Research.* 2018;7:F1000 Faculty Rev. <https://doi.org/10.12688/f1000research.15096.1>.
5. O'Gara PT, Kushner FG, Ascheim DD, et al. 2013 ACCF/AHA guideline for the management of ST-elevation myocardial infarction: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. *Circulation.* 2013;127(4):e362–425. <https://doi.org/10.1161/CIR.0b013e3182742cf6>.
6. Macfarlane PW. Age, sex, and the ST amplitude in health and disease. *J Electrocardiol.* 2001;34(Suppl):235–41. <https://doi.org/10.1054/jelc.2001.28906>.
7. Menown IB, Mackenzie G, Adgey AA. Optimizing the initial 12-lead electrocardiographic diagnosis of acute myocardial infarction. *Eur Heart J.* 2000;21(4):275–83. <https://doi.org/10.1053/ehj.1999.1748>.
8. Carville SF, Henderson R, Gray H. The acute management of ST-segment-elevation myocardial infarction. *Clin Med.* 2015;15(4):362. <https://doi.org/10.7861/clinmedicine.15-4-362>.
9. Cohen M, Visveswaran G. Defining and managing patients with non-ST-elevation myocardial infarction: Sorting through type 1 vs other types. *Clin Cardiol.* 2020;43(3):242. <https://doi.org/10.1002/clc.23308>.
10. Basit H, Malik A, Huecker MR. Non-ST-Segment Elevation Myocardial Infarction. In: StatPearls. StatPearls Publishing; 2024. Accessed October 20, 2024. <http://www.ncbi.nlm.nih.gov/books/NBK513228/>
11. Indications for fibrinolytic therapy in suspected acute myocardial infarction: collaborative overview of early mortality and major morbidity results from all randomised trials of more than 1000 patients. Fibrinolytic Therapy Trialists' (FTT) Collaborative Group. *Lancet Lond Engl.* 1994;343(8893):311–322.
12. Khan AR, Golwala H, Tripathi A, et al. Impact of total occlusion of culprit artery in acute non-ST elevation myocardial infarction: a systematic review and meta-analysis. *Eur Heart J.* 2017;38(41):3082–9. <https://doi.org/10.1093/eurheartj/ehx418>.
13. Kontos MC, Kurz MC, Roberts CS, et al. An Evaluation of the Accuracy of Emergency Physician Activation of the Cardiac Catheterization Laboratory for Patients With Suspected ST-Segment Elevation Myocardial Infarction. *Ann Emerg Med.* 2010;55(5):423–30. <https://doi.org/10.1016/j.annemergmed.2009.08.011>.
14. Larson DM, Menssen KM, Sharkey SW, et al. "False-Positive" Cardiac Catheterization Laboratory Activation Among Patients With Suspected ST-Segment Elevation Myocardial Infarction. *JAMA.* 2007;298(23):2754–60. <https://doi.org/10.1001/jama.298.23.2754>.
15. Hochman JS, Lamas GA, Buller CE, et al. Coronary intervention for persistent occlusion after myocardial infarction. *N Engl J Med.* 2006;355(23):2395–407. <https://doi.org/10.1056/NEJMoA066139>.
16. Miranda DF, Lobo AS, Walsh B, Sandoval Y, Smith SW. New Insights Into the Use of the 12-Lead Electrocardiogram for Diagnosing Acute Myocardial Infarction in the Emergency Department. *Can J Cardiol.* 2018;34(2):132–45. <https://doi.org/10.1016/j.cjca.2017.11.011>.
17. Aslanger EK, Yıldırım Ö, Şimşek B, et al. Diagnostic accuracy of electrocardiogram for acute coronary OCCLUSION resulting in myocardial infarction (DIFOCCULT Study). *IJC Heart Vasc.* 2020;30:100603. <https://doi.org/10.1016/j.ijcha.2020.100603>.

18. Linde JJ, Kelbæk H, Hansen TF, et al. Coronary CT Angiography in Patients With Non-ST-Segment Elevation Acute Coronary Syndrome. *J Am Coll Cardiol*. 2020;75(5):453–63. <https://doi.org/10.1016/j.jacc.2019.12.012>.
19. Eek C, Grenne B, Brunvand H, et al. Strain echocardiography predicts acute coronary occlusion in patients with non-ST-segment elevation acute coronary syndrome. *Eur J Echocardiogr*. 2010;11(6):501–8. <https://doi.org/10.1093/ejehocardiography/jeq008>.
20. Thiele H, Rach J, Klein N, et al. Optimal timing of invasive angiography in stable non-ST-elevation myocardial infarction: the Leipzig Immediate versus early and late Percutaneous coronary Intervention trial in NSTEMI (LIPSIA-NSTEMI Trial). *Eur Heart J*. 2012;33(16):2035–43. <https://doi.org/10.1093/eurheartj/ehr418>.
21. Milosevic A, Vasiljevic-Pokrajic Z, Milasinovic D, et al. Immediate Versus Delayed Invasive Intervention for Non-STEMI Patients: The RIDDLE-NSTEMI Study. *JACC Cardiovasc Interv*. 2016;9(6):541–9. <https://doi.org/10.1016/j.jcin.2015.11.018>.
22. Montalescot G, Cayla G, Collet JP, et al. Immediate vs Delayed Intervention for Acute Coronary Syndromes: A Randomized Clinical Trial. *JAMA*. 2009;302(9):947–54. <https://doi.org/10.1001/jama.2009.1267>.
23. van't Hof AWJ, de Vries ST, Dambrink JHE, et al. A comparison of two invasive strategies in patients with non-ST elevation acute coronary syndromes: results of the Early or Late Intervention in unstable Angina (ELISA) pilot study: 2b/3a upstream therapy and acute coronary syndromes. *Eur Heart J*. 2003;24(15):1401–5. [https://doi.org/10.1016/S0195-668X\(03\)00259-8](https://doi.org/10.1016/S0195-668X(03)00259-8).
24. Hoedemaker NPG, Damman P, Woudstra P, et al. Early Invasive Versus Selective Strategy for Non-ST-Segment Elevation Acute Coronary Syndrome: The ICTUS Trial. *J Am Coll Cardiol*. 2017;69(15):1883–93. <https://doi.org/10.1016/j.jacc.2017.02.023>.
25. Mehta SR, Granger CB, Boden WE, et al. Early versus Delayed Invasive Intervention in Acute Coronary Syndromes. *N Engl J Med*. 2009;360(21):2165–75. <https://doi.org/10.1056/NEJMoa0807986>.
26. Ibanez B, James S, Agewall S, et al. 2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation: The Task Force for the management of acute myocardial infarction in patients presenting with ST-segment elevation of the European Society of Cardiology (ESC). *Eur Heart J*. 2018;39(2):119–77. <https://doi.org/10.1093/eurheartj/ehx393>.
27. Amsterdam EA, Wenger NK, Brindis RG, et al. 2014 AHA/ACC Guideline for the Management of Patients With Non-ST-Elevation Acute Coronary Syndromes. *Circulation*. 2014;130(25):e344–426. <https://doi.org/10.1161/CIR.0000000000000134>.
28. Akbar H, Foth C, Kahloon RA, Mountfort S. Acute ST-Segment Elevation Myocardial Infarction (STEMI). In: *StatPearls*. StatPearls Publishing; 2024. Accessed October 20, 2024. <http://www.ncbi.nlm.nih.gov/books/NBK532281/>
29. Scholz KH, Meyer T, Lengenfelder B, et al. Patient delay and benefit of timely reperfusion in ST-segment elevation myocardial infarction. *Open Heart*. 2021;8(1): e001650. <https://doi.org/10.1136/openhrt-2021-001650>.
30. Lambert L, Brown K, Segal E, Brophy J, Rodas-Cabau J, Bogaty P. Association between timeliness of reperfusion therapy and clinical outcomes in ST-elevation myocardial infarction. *JAMA*. 2010;303(21):2148–55. <https://doi.org/10.1001/jama.2010.712>.
31. Writing Committee Members, Antman EM, Anbe DT, et al. ACC/AHA Guidelines for the Management of Patients With ST-Elevation Myocardial Infarction—Executive Summary. *Circulation*. 2004;110(5):588–636. <https://doi.org/10.1161/01.CIR.0000134791.68010.FA>.
32. Ayad SW, Zawawy THE, Lotfy MI, Naguib AM, Amrawy AME. Incidence and impact of totally occluded culprit coronary artery in patients with non-ST segment elevation myocardial infarction acute coronary syndrome. *Egypt Heart J*. 2021;73:36. <https://doi.org/10.1186/s43044-021-00160-x>.
33. Spirito A, Vaisnora L, Papadis A, et al. Acute Coronary Occlusion in Patients With Non-ST-Segment Elevation Out-of-Hospital Cardiac Arrest. *J Am Coll Cardiol*. 2023;81(5):446–56. <https://doi.org/10.1016/j.jacc.2022.10.039>.
34. Hung CS, Chen YH, Huang CC, et al. Prevalence and outcome of patients with non-ST segment elevation myocardial infarction with occluded “culprit” artery – a systemic review and meta-analysis. *Crit Care*. 2018;22:34. <https://doi.org/10.1186/s13054-018-1944-x>.
35. Meyers HP, Bracey A, Lee D, et al. Comparison of the ST-Elevation Myocardial Infarction (STEMI) vs. NSTEMI and Occlusion MI (OMI) vs. NOMI Paradigms of Acute MI. *J Emerg Med*. 2021;60(3):273–84. <https://doi.org/10.1016/j.jemermed.2020.10.026>.
36. Aslanger EK. Beyond the ST-segment in Occlusion Myocardial Infarction (OMI): Diagnosing the OMI-nous. *Turk J Emerg Med*. 2022;23(1):1. <https://doi.org/10.4103/2452-2473.357333>.
37. Kola M, Shuka N, Meyers HP, ZaimiPetrela E, Smith SW. OMI/ NOMI: Time for a New Classification of Acute Myocardial Infarction. *J Clin Med*. 2024;13(17):5201. <https://doi.org/10.3390/jcm13175201>.
38. Meyers HP, Bracey A, Lee D, et al. Accuracy of OMI ECG findings versus STEMI criteria for diagnosis of acute coronary occlusion myocardial infarction. *Int J Cardiol Heart Vasc*. 2021;33:100767. <https://doi.org/10.1016/j.ijcha.2021.100767>.
39. O'Donoghue M, Boden WE, Braunwald E, et al. Early invasive vs conservative treatment strategies in women and men with unstable angina and non-ST-segment elevation myocardial infarction: a meta-analysis. *JAMA*. 2008;300(1):71–80. <https://doi.org/10.1001/jama.300.1.71>.
40. DeWood MA, Spores J, Notske R, et al. Prevalence of total coronary occlusion during the early hours of transmural myocardial infarction. *N Engl J Med*. 1980;303(16):897–902. <https://doi.org/10.1056/NEJM198010163031601>.
41. Dixon WC, Wang TY, Dai D, Shunk KA, Peterson ED, Roe MT. Anatomic Distribution of the Culprit Lesion in Patients With Non-ST-Segment Elevation Myocardial Infarction Undergoing Percutaneous Coronary Intervention. *J Am Coll Cardiol*. 2008;52(16):1347–8. <https://doi.org/10.1016/j.jacc.2008.07.029>.
42. Wang TY, Zhang M, Fu Y, et al. Incidence, distribution, and prognostic impact of occluded culprit arteries among patients with non-ST-elevation acute coronary syndromes undergoing diagnostic angiography. *Am Heart J*. 2009;157(4):716–23. <https://doi.org/10.1016/j.ahj.2009.01.004>.
43. Emergency department: rapid identification and treatment of patients with acute myocardial infarction. National Heart Attack Alert Program Coordinating Committee, 60 Minutes to Treatment Working Group. *Ann Emerg Med*. 1994;23(2):311–329.
44. Aslanger EK, Meyers HP, Smith SW. Recognizing electrocardiographically subtle occlusion myocardial infarction and differentiating it from mimics: Ten steps to or away from cath lab. *Turk Kardiyol Dernegi Arsivi Turk Kardiyol Derneginin Yayin Organidir*. 2021;49(6):488–500. <https://doi.org/10.5543/tkda.2021.21026>.
45. Mead NE, O'Keefe KP. Wellen's syndrome: An ominous EKG pattern. *J Emerg Trauma Shock*. 2009;2(3):206–8. <https://doi.org/10.4103/0974-2700.55347>.
46. de Winter RJ, Verouden NJW, Wellens HJJ, Wilde AAM, Interventional Cardiology Group of the Academic Medical Center. A new ECG sign of proximal LAD occlusion. *N Engl J Med*. 2008;359(19):2071–3. <https://doi.org/10.1056/NEJMc0804737>.
47. Wang S, Shen L. de Winter syndrome or inferior STEMI? *BMC Cardiovasc Disord*. 2021;21(1):614. <https://doi.org/10.1186/s12872-021-02441-4>.
48. Nikus K, Pahlm O, Wagner G, et al. Electrocardiographic classification of acute coronary syndromes: a review by a committee

- of the International Society for Holter and Non-Invasive Electrocardiology. *J Electrocardiol.* 2010;43(2):91–103. <https://doi.org/10.1016/j.jelectrocard.2009.07.009>.
49. Birnbaum Y, Nikus K, Kligfield P, et al. The Role of the ECG in Diagnosis, Risk Estimation, and Catheterization Laboratory Activation in Patients with Acute Coronary Syndromes: A Consensus Document. *Ann Noninvasive Electrocardiol.* 2014;19(5):412–25. <https://doi.org/10.1111/anec.12196>.
50. Khalid U, Birnbaum Y. Clinical Significance of Upsloping ST Depression on Resting Electrocardiogram. *Ann Noninvasive Electrocardiol.* 2016;21(2):202–5. <https://doi.org/10.1111/anec.12273>.
51. Rich MW, Imburgia M, King TR, Fischer KC, Kovach KL. Electrocardiographic Diagnosis of Remote Posterior Wall Myocardial Infarction Using Unipolar Posterior Lead V9. *Chest.* 1989;96(3):489–93. <https://doi.org/10.1378/chest.96.3.489>.
52. Perloff JK. The Recognition of Strictly Posterior Myocardial Infarction by Conventional Scalar Electrocardiography. *Circulation.* 1964;30(5):706–18. <https://doi.org/10.1161/01.CIR.30.5.706>.
53. Brady WJ. Acute posterior wall myocardial infarction: Electrocardiographic manifestations. *Am J Emerg Med.* 1998;16(4):409–13. [https://doi.org/10.1016/S0735-6757\(98\)90143-7](https://doi.org/10.1016/S0735-6757(98)90143-7).
54. Engelen DJ, Gorgels AP, Cheriex EC, et al. Value of the electrocardiogram in localizing the occlusion site in the left anterior descending coronary artery in acute anterior myocardial infarction. *J Am Coll Cardiol.* 1999;34(2):389–95. [https://doi.org/10.1016/S0735-1097\(99\)00197-7](https://doi.org/10.1016/S0735-1097(99)00197-7).
55. Yamaji H, Iwasaki K, Kusachi S, et al. Prediction of acute left main coronary artery obstruction by 12-lead electrocardiography. *J Am Coll Cardiol.* 2001;38(5):1348–54. [https://doi.org/10.1016/S0735-1097\(01\)01563-7](https://doi.org/10.1016/S0735-1097(01)01563-7).
56. Zimetbaum PJ, Josephson ME. Use of the Electrocardiogram in Acute Myocardial Infarction. *N Engl J Med.* 2003;348(10):933–40. <https://doi.org/10.1056/NEJMr022700>.
57. Tamura A. Significance of lead aVR in acute coronary syndrome. *World J Cardiol.* 2014;6(7):630–7. <https://doi.org/10.4330/wjc.v6.i7.630>.
58. Luo G, Li Q, Duan J, Peng Y, Zhang Z. The Predictive Value of Fragmented QRS for Cardiovascular Events in Acute Myocardial Infarction: A Systematic Review and Meta-Analysis. *Front Physiol.* 2020;11:1027. <https://doi.org/10.3389/fphys.2020.01027>.
59. Morris F. ABC of clinical electrocardiography: Acute myocardial infarction—Part I. *BMJ.* 2002;324(7341):831–4. <https://doi.org/10.1136/bmj.324.7341.831>.
60. Meyers HP, Limkakeng AT, Jaffa EJ, et al. Validation of the modified Sgarbossa criteria for acute coronary occlusion in the setting of left bundle branch block: A retrospective case-control study. *Am Heart J.* 2015;170(6):1255–64. <https://doi.org/10.1016/j.ahj.2015.09.005>.
61. Jaiswal AK, Shah S. Shark Fin Electrocardiogram: A Deadly Electrocardiogram Pattern in ST-Elevation Myocardial Infarction (STEMI). *Cureus.* 2021;13(6):e15989. <https://doi.org/10.7759/cureus.15989>.
62. Bracey A, Meyers HP, Smith SW. Post-arrest wide complex rhythm: What is the cause of death? *Am J Emerg Med.* 2021;45:683.e5–683.e7. <https://doi.org/10.1016/j.ajem.2020.12.028>.
63. Littmann L. South African flag sign: a teaching tool for easier ECG recognition of high lateral infarct. *Am J Emerg Med.* 2016;34(1):107–9. <https://doi.org/10.1016/j.ajem.2015.10.022>.
64. Durant E, Singh A. Acute first diagonal artery occlusion: a characteristic pattern of ST elevation in noncontiguous leads. *Am J Emerg Med.* 2015;33(9):1326.e3–5. <https://doi.org/10.1016/j.ajem.2015.02.008>.
65. Smith SW, Dodd KW, Henry TD, Dvorak DM, Pearce LA. Diagnosis of ST-elevation myocardial infarction in the presence of left bundle branch block with the ST-elevation to S-wave ratio in a modified Sgarbossa rule. *Ann Emerg Med.* 2012;60(6):766–76. <https://doi.org/10.1016/j.annemergmed.2012.07.119>.
66. Janssens GN, Lemkes JS, van der Hoeven NW, et al. Transient ST-elevation myocardial infarction versus persistent ST-elevation myocardial infarction. An appraisal of patient characteristics and functional outcome. *Int J Cardiol.* 2021;336:22–8. <https://doi.org/10.1016/j.ijcard.2021.05.018>.
67. Meisel SR, Dagan Y, Blondheim DS, et al. Transient ST-elevation myocardial infarction: clinical course with intense medical therapy and early invasive approach, and comparison with persistent ST-elevation myocardial infarction. *Am Heart J.* 2008;155(5):848–54. <https://doi.org/10.1016/j.ahj.2007.12.010>.
68. Bischof JE, Worrall C, Thompson P, Marti D, Smith SW. ST depression in lead aVL differentiates inferior ST-elevation myocardial infarction from pericarditis. *Am J Emerg Med.* 2016;34(2):149–54. <https://doi.org/10.1016/j.ajem.2015.09.035>.
69. Beeman WW, Smith SW, Shroff GR. 369 T/QRS Amplitude Ratio Is Significantly Higher in Acute Anterior ST-Elevation Myocardial Infarction Than in Previous Myocardial Infarction With Persistent ST Elevation (Left Ventricular Aneurysm Morphology): A Validation. *Ann Emerg Med.* 2011;58(4):S302. <https://doi.org/10.1016/j.annemergmed.2011.06.401>.
70. Smith SW. T/QRS ratio best distinguishes ventricular aneurysm from anterior myocardial infarction. *Am J Emerg Med.* 2005;23(3):279–87. <https://doi.org/10.1016/j.ajem.2005.01.003>.
71. Kontos MC, De Lemos JA, Deitelzweig SB, et al. 2022 ACC Expert Consensus Decision Pathway on the Evaluation and Disposition of Acute Chest Pain in the Emergency Department. *J Am Coll Cardiol.* 2022;80(20):1925–60. <https://doi.org/10.1016/j.jacc.2022.08.750>.
72. McLaren J, De Alencar JN, Aslanger EK, Meyers HP, Smith SW. From ST-Segment Elevation MI to Occlusion MI. *JACC Adv.* 2024;3(11):101314. <https://doi.org/10.1016/j.jacadv.2024.101314>.
73. de Alencar Neto JN, Scheffer MK, Correia BP, Franchini KG, Felicioni SP, De Marchi MFN. Systematic review and meta-analysis of diagnostic test accuracy of ST-segment elevation for acute coronary occlusion. *Int J Cardiol.* 2024;402:131889. <https://doi.org/10.1016/j.ijcard.2024.131889>.
74. Cook DA, Oh SY, Pusic MV. Accuracy of Physicians' Electrocardiogram Interpretations: A Systematic Review and Meta-analysis. *JAMA Intern Med.* 2020;180(11):1461–71. <https://doi.org/10.1001/jamainternmed.2020.3989>.
75. Sandau KE, Funk M, Auerbach A, et al. Update to Practice Standards for Electrocardiographic Monitoring in Hospital Settings: A Scientific Statement From the American Heart Association. *Circulation.* 2017;136(19):e273–344. <https://doi.org/10.1161/CIR.0000000000000527>.
76. McLaren JTT, Taher AK, Kapoor M, Yi SL, Chartier LB. Sharing and Teaching Electrocardiograms to Minimize Infarction (STEMI): reducing diagnostic time for acute coronary occlusion in the emergency department. *Am J Emerg Med.* 2021;48:18–32. <https://doi.org/10.1016/j.ajem.2021.03.067>.
77. PMcardio OMI AI ECG Model | Queen of Hearts. November 14, 2023. Accessed November 24, 2024. <https://www.powerfulmedical.com/pmcardio-omi/>.
78. Herman R, Meyers HP, Smith SW, et al. International evaluation of an artificial intelligence-powered electrocardiogram model detecting acute coronary occlusion myocardial infarction. *Eur Heart J Digit Health.* 2024;5(2):123–33. <https://doi.org/10.1093/ehjdh/ztad074>.
79. Al-Zaiti SS, Martin-Gill C, Zègre-Hemsey JK, et al. Machine learning for ECG diagnosis and risk stratification of occlusion myocardial infarction. *Nat Med.* 2023;29(7):1804–13. <https://doi.org/10.1038/s41591-023-02396-3>.
80. Tziakas D, Chalikias G, Al-Lamee R, Kaski JC. Total coronary occlusion in non ST elevation myocardial infarction: Time to change our practice? *Int J Cardiol.* 2021;329:1–8. <https://doi.org/10.1016/j.ijcard.2020.12.082>.

81. Avdikos G, Michas G, Smith SW. From Q/Non-Q Myocardial Infarction to STEMI/NSTEMI: Why It's Time to Consider Another Simplified Dichotomy; a Narrative Literature Review. *Arch Acad Emerg Med*. 2022;10(1):e78. <https://doi.org/10.22037/aaem.v10i1.1783>.
82. Type-1 Myocardial Infarction (OMI & NOMI). EMCrit Project. Accessed October 29, 2024. <https://emcrit.org/ibcc/mi/>

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