Electrocardiogram patterns in non-ST segment myocardial infarction - from theory to practical challenges

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Abstract: Patients presenting with non ST-segment elevation myocardial infarction (NSTEMI) represent a wide spectrum of coronary artery disease severity and, therefore, have major differences in the outcome. Rapid risk stratification in patients with NSTEMI is crucial for appropriate management, especially for very high risk patients, who benefit from an early invasive approach. The ECG remains the most accessible used diagnostic tool for guiding emergent treatment strategies. The ECG recorded during acute myocardial ischemia has diagnostic, therapeutic and prognostic significance. When confronted with ST - T abnormalities, the main challenge is to differentiate between a primary and secondary ST depression due to other pathologies. If there is a clinical high index of myocardial infarction and the ECG is not diagnostic, serial ECGs at 5-10 min intervals are needed.

Keywords: non ST-segment elevation myocardial infarction, ECG patterns, risk stratification

The Third Universal Definition of myocardial infarction (MI) consensus document defines MI by the evidence of myocardial necrosis in a clinical setting suggestive for acute myocardial ischemia, proved by a rise and/or fall of cTn, with at least one value above the 99th percentile of a normal reference population in the presence of at least one of the following: a) symptoms consistent with myocardial ischemia; b) new or presumed new significant ST-segment or T-wave changes or new left bundle-branch block; c) development of pathological Q waves on the electrocardiogram; d) imaging evidence of new loss of viable myocardium such as new regional wall motion abnormalities; or e) identification of an intracoronary thrombus either by angiography or autopsy¹.

Myocardial ischemia may occur during two pathophysiologic processes: decreased blood supply, in which a coronary artery has been acutely occluded by a thrombus or vasospasm, or a mismatch between increased demands due to excess cardiac work (caused by exercise or other stress) and inadequate blood supply in the presence of nonoclusive coronary artery disease (CAD).

On the basis of the above criteria, MI is diagnosed in instances in which a supply/demand imbalance leads to myocardial injury with necrosis that is not caused by acute coronary syndrome (ACS), including arrhythmias, aortic dissection, severe aortic valve disease, hypertrophic cardiomyopathy, shock, respiratory failure, severe anemia, hypertension with or without left ventricular hypertrophy, coronary spasm, coronary embolism or vasculitis, and coronary endothelial dysfunction without significant CAD²-⁴.

Patients with myocardial ischemia secondary to a decreased supply typically present with two types of electrocardiogram (ECG) patterns: a) predominant ST-

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segment elevation acute coronary syndrome, and are classified as having either “aborted myocardial infarction (MI)” or ST-elevation MI (STEMI) based on the presence or absence of biomarkers of myocardial necrosis; and b) patients without predominant ST segment elevation on the 12-lead ECG - non-ST elevation ACS.

Patients presenting with NSTEMI represent a wide spectrum of CAD severity and, therefore, have major differences in the outcome. Urgent reperfusion with thrombolytic therapy has been proven to be beneficial only in patients presenting with ST-segment elevation, whereas in the general group without ST-segment elevation, including those with ST-segment depression (Figure 1,2), flat or negative T wave and even normal or unchanged ECG, thrombolytic therapy may even be harmful, while an early invasive strategy (percutaneous coronary intervention/urgent CABG) is superior to a conservative approach in high-risk patients with NSTEMI. Rapid risk stratification in patients with NSTEMI is crucial for appropriate management, especially for higher-risk patients, who benefit from an early invasive approach. Therefore, the timing of angiography and revascularization should be based on patient risk profile.

Patients at very high risk (haemodynamic instability or cardiogenic shock, recurrent or ongoing chest pain refractory to medical treatment, life-threatening arrhythmias including conduction disturbances or cardiac arrest, mechanical complications of MI, acute heart failure, recurrent dynamic ST-T wave changes (Figure 3).
current symptoms (none of the above criteria met), a noninvasive assessment of inducible ischaemia should be performed before hospital discharge3.

The ECG remains the most accessible used diagnostic tool for guiding emergent treatment strategies. The ECG recorded during acute myocardial ischemia has diagnostic, therapeutic and prognostic significance. ECG changes may include transient ST-segment elevation, persistent or transient ST-segment depression, T-wave inversion, flat T waves or pseudo-normalization of T waves or the ECG may be normal. Therefore, it is paramount to indentify the patients having anatomically or functionally severe coronary obstruction based on standard 12-lead ECG interpretation. When ischemia is confined primarily to the subendocardium, the overall ST vector typically faces the inner ventricular layer and the ventricular cavity such that the surface ECG leads show ST-segment depression. This subendocardial ischemic pattern is a frequent finding (3,4), particularly with intermittent ST-elevation) should be considered for urgent coronary angiography (in less than 2 hours). In patients at high risk (at least one of the following criteria: rise or fall in cardiac troponin compatible with MI, dynamic ST- or T-wave changes (symptomatic or silent - Figure 5), GRACE score >140) an early invasive strategy within 24 hours appears to be the reasonable strategy. In intermediate-risk cases (with a GRACE risk score under 140, diabetes mellitus, LVEF<40% or congestive heart failure, the invasive evaluation can be delayed without increased risk but should be performed preferably within 72 hours of admission (Figure 6,7). In low-risk patients without re-
been established as ECG markers of poor outcome in NSTEMI7.

The number of leads showing ST depression and the magnitude of ST depression are indicative of the extent of ischaemia and correlate with worse prognosis, while benefitting from an early invasive treatment strategy.

When confronted with ST-T abnormalities, the main challenge is to differentiate between a primary and secondary ST depression due to other pathologies. With primary ST depression, the T wave abnormalities are accompanied by a normal QRS, and likely reflect myocardial ischemia. In secondary ST depression, the T wave abnormalities are in response to QRS complex abnormalities or ventricular hypertrophy and are not reflecting ischemia (LBBB, RBBB).

An interesting variant of hyperacute T-waves was first described in 2009 by Verouden and colleagues (Figure 8) and was found to represent complete LAD occlusion and therefore a STEMI-equivalent (Figure 9). This pattern of up sloping ST-segment depression paired with a tall, prominent T-wave (de Winter T-waves) is present in about 2% of patients with LAD occlusion and these patients require immediate reperfusion8.

Another pattern suggestive for subepicardial ischemia is the T-wave pseudonormalization (equivalent of hyperacute T waves). This phenomenon may occur when a patient has a re-occlusion of a recently reperfused artery.

Figure 9. Coronary angiogram, occlusions left anterior descending artery (LAD).

Figure 10. Different from the previous case: preserved precordial R wave progression, biphasic T waves in V2-4.

Figure 11. Coronary angiogram - LAD coronary artery subocclusion.
Wellens’ syndrome - a pattern of deeply inverted or biphasic T waves in V2-3 developing after the angina attack, with preserved precordial R wave progression, no precordial Q waves (Figure 10), recent history of angina and normal or slightly elevated cardiac markers - which is highly specific for a critical stenosis of the left anterior descending artery (Figure 11) must be diligently recognized since these patients usually require early invasive therapy, due to the high risk of LAD occlusion.

Acute cerebrovascular events, especially hemorrhage, may present with diffuse precordial T wave inversions due to small vessel ischemia. Although some suggest the QT interval is usually prolonged in these cases in opposition to NSTEMI/ACS, in most patients there is no proven way to differentiate between the two entities solely on the ECG (Figure 12,13).

T wave inversion persisting after right ventricular pacing, LBBB or WPW are called “memory” T waves, caused by an unique phenomenon of electrical remodeling seen after periods of altered ventricular conduction wherein the T-wave direction during ‘memory’, i.e. during periods after altered depolarization, is similar to that of the QRS complex during periods of abnormal depolarization (Figure 14, 15). Although it appears to be a relatively benign pathophysiologic finding, T-wave inversions (TWI) due to cardiac memory may lead to confusion with NSTEMI/ACS and therefore to unnecessary testing and treatment.

CONCLUSIONS

ECG is the mainstay of diagnosing NSTEMI which is a true medical emergency and making the correct diagnosis promptly is life-saving. If there is a clinical high index of MI and the ECG is not diagnostic, serial ECGs at 5-10 min intervals are needed. We should keep in mind the multiple ECG patterns of NSTEMI and always interpret them in clinical context. The ECG is the first clinical tool that allowed assessment of myocardial ischaemia and despite multiple paradigm shifts in the management of ACS, it continues to be the pre-em-
ponent test directing therapeutic management and prognostic stratification.

Conflict of interest: none declared.

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