Acute Myocardial Infarction and STEMI-equivalent Patterns

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Abstract

The electrocardiogram (ECG) is the first-line investigation in patients presenting with an acute coronary syndrome (ACS). Recent research has identified several limitations in the use of current ECG criteria in diagnosing which ACSs require primary (i.e. urgent) coronary angiography. Two independent studies have shown approximately a quarter of NSTEMI patients have complete occlusion at early angiography. These patients had larger infarcts and worse clinical outcomes compared to NSTEMI patients with incomplete occlusion. Koyama and colleagues (2002) found a completely occluded culprit artery in 47% of NSTEMI ECGs (regardless of risk stratification) versus 57% of patients with STEMI. This suggests there is a significant subgroup of NSTEMI patients who would benefit from primary coronary angiography, rather than medical therapy alone. These STEMI-equivalents include: posterior myocardial infarction (MI) (ST-depression in V1-V4, tall R/S ratio >1 in V1 or V2), de-Winter ST/T wave complex, delayed activation wave (notch in terminal QRS complex II, III, aVF, or I, aVL), and T-wave precordial instability (T-wave height in V1 >V6). These novel ECG changes are not indications for urgent coronary angiography in current guidelines, although recent evidence suggests they may have a role in the future of ACS risk stratification.

Key words: Arterial cannulation, Art line, Radial Artery

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1. Introduction

The electrocardiogram (ECG) continues to be the first-line investigation in patients presenting with an acute coronary syndrome (ACS). Recent research has identified several limitations in the use of current ECG criteria in diagnosing ACS in need of primary (i.e. urgent) coronary angiography. Recognition of patients meeting ST-elevation criteria for activation of primary coronary angiography is consistently poor, with only 70-78% of clearly eligible patients receiving guideline recommended therapy [1,2]. Other studies have shown that up to 57% of non-ST elevation acute coronary syndromes (NSTEMI), and 20% of non-ST elevation myocardial infarction (NSTEMI) patients were lacking classic ischaemic ECG changes [3]. In addition, two independent studies have shown that approximately a quarter of NSTEMI patients have complete occlusion at early angiography [4,5]. These patients had larger infarcts, and worse clinical outcomes compared to NSTEMI patients with incomplete occlusion. Koyama and colleagues (2002), found a completely occluded culprit artery in 47% of NSTEMI ECGs (regardless of risk stratification) versus 57% of patients with STEMI. This suggests there is a significant subgroup of NSTEMI patients who would benefit from primary coronary angiography, rather than medical therapy alone [6].

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Currently, guidelines suggest NSTEMI patients should receive angiography within 24hrs if they meet high-risk criteria, detailed in Table 1. This may result in patients with complete occlusion being placed in the elective (non-urgent) category, and waiting up to 24hrs for angiography. Hence, there is a need for better identification, and triaging of acute coronary occlusions lacking ‘classic’ ST-elevation, which are otherwise defined by international consensus committees as ‘STEMI-equivalents’ [7].

Table 1. High-risk features in NSTEMI

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<th>Presentation with clinical features consistent with acute coronary syndromes (ACS) and any of the following high-risk features:</th>
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<td>Elevated level of at least one cardiac biomarker (troponin or creatine kinase-MB isoenzyme);</td>
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<td>Persistent or dynamic electrocardiographic changes of ST-segment depression 0.5 mm or new T-wave inversion 2mm;</td>
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<td>Transient ST-segment elevation (0.5 mm) in more than two contiguous leads;</td>
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<tr>
<td>Haemodynamic compromise — systolic blood pressure &lt; 90 mmHg, cool peripheries, diaphoresis, Killip Class &gt; I, and/or new-onset mitral regurgitation;</td>
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<td>Sustained ventricular tachycardia; Syncope;</td>
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<td>Left ventricular systolic dysfunction (left ventricular ejection fraction &lt; 0.40);</td>
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<td>Prior percutaneous coronary intervention within 6 months or prior coronary artery bypass surgery;</td>
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<tr>
<td>Presence of known diabetes (with typical symptoms of ACS); or Chronic kidney disease (estimated glomerular filtration rate &lt; 60 mL/minute) (with typical symptoms of ACS).</td>
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Adjusted from Guidelines for the Management of Acute Coronary Syndromes 2006 (MJA, 2006) [8].

Recently, there has been a shift in the literature towards finding novel ECG changes, which are highly suggestive of total coronary artery occlusion but lack ST-elevation in contiguous leads. These STEMI-equivalents include:

- posterior myocardial infarction (MI) (ST-depression in V1-V4, tall R/S ratio >1 in V1 or V2)
- de-Winter ST/T wave complex
- delayed activation wave (notch in terminal QRS complex II, III, aVF, or I, aVL) and
- T-wave precordial instability (T-wave height in V1 >V6).

These novel ECG changes are not indications for urgent coronary angiography in current guidelines, although recent evidence suggests they may have a role.

2. Wellens’ Syndrome

First described as an ECG feature of subgroup of patients admitted to hospital for unstable angina [9], Wellens’ syndrome is considered to herald an extensive anterior wall MI. The pattern, also referred to as ‘LAD coronary T wave syndrome’, indicates critical stenosis of the proximal left anterior descending artery, and has a natural history of AMI. Rinehart et al. [10] described the diagnostic criteria of Wellens’ Syndrome on ECG as:

- Deeply-inverted or biphasic T waves in V2-3 (may extend to V1-6)
- Isoelectric or minimally-elevated ST segment (< 1mm)
- Absence of precordial Q waves
- Normal precordial R wave progression

These features are well appreciated in Figure 4 [11]. Rinehart et al. [10] outlined that the patient may have a recent history of angina, but the ECG pattern is present in the pain-free state. The T wave changes may persist for hours to weeks but will normalise with definitive management of the stenosed vessel. Serum cardiac markers may be slightly elevated.

3. Posterior Myocardial Infarction

The posterior aspect of the myocardium is not well visualised by standard 12-lead ECG. It is often only through indirect evidence via ST-depression in the antero-septal (V1-V4) leads and evolving R-waves in V1 or V2 with R/S ratios >1 that a diagnosis of posterior infarct is suspected [12]. These R-waves are thought to represent inverted Q-waves [13] (see Figure 2 for an example).

Isolated ST-depression in V1-V4 has been shown to appear in 10% of all ACS presentations [15]. In another more recent study of 13,604 patients, ~9% of patients had isolated ST-depression in V1-V4 and of these, 26% had an occluded culprit artery, and 56% had positive troponins [16]. Furthermore, other authors have suggested that the ST-
depression can extend past V4 into V5 and perhaps other leads, but posterior occlusion can still be suspected because the ST-depression in V1-V4 is of greater amplitude [17]. Hence, an improvement to this methodology would be to include ECG traces with ST-depression extending past V4 provided that this depression is relatively minimal compared to V1-V4. In this way we aim to provide evidence of a more sensitive way of identifying posterior MI.

Figure 1. Wellens’ Syndrome, characterised by inverted, biphasic T waves most prominent in V2-V3, minor ST elevation in the precordium with normal R wave progression (V3 R wave >3mm) [11].

Figure 2. Example of posterior MI, suggested by ST-depression in V2-V3, tall broad R-waves in V2-V3, dominant R-wave (R/S ratio>1) in V2, upright terminal portions of T-waves in V2-V3 [14].

4. De-Winter ST/T Wave Complex

In 2009, Verouden et al. were the first to systematically examine a previously identified pattern of acute ECG change suggestive of proximal left anterior descending (LAD) artery occlusion, the de-Winter ST/T wave complex (de-Winters) [18]. They found that 2% of 1890 patients had a complete proximal LAD occlusion without any ST-elevation in V1-V6, but a distinct pattern consisting of 1mm ST-depression at the J-point, with up-sloping ST-segments continuing into tall T-waves in the precordial leads (see Figure 2). Minimal (<0.5mm) ST-elevation in aVR was present in all patients with this finding. These represented static changes, lasting from first contact until the pre-procedural ECG. Hence, this finding was unlike the long described transient hyper-acute T-waves, which subsequently evolves into ST-elevation. This finding has only been studied retrospectively in regards to proven LAD occlusion. There is currently no data regarding its presence in other occlusions. This led Verouden et al. (2009) to suggest that their figure of 2% was perhaps an underestimation.
5. The delayed activation wave

Recently, Niu et al. [20] found 77% of patients with acute occlusion of their left circumflex artery (LCX) had a notch/deflection in the terminal QRS complex in leads II, III, and aVF or I, aVL. This change was present in patients without ST-elevation, and hence represents a new tool in which to identify coronary artery occlusion in ACS. The authors did not define occlusion, nor include troponin analysis.

6. T-wave precordial instability

The normal T-wave in lead V1 is inverted. There has been a suggestion in the literature that an upright T-wave in V1, particularly if it is tall and V1 > V6 is a type of hyper acute T-wave and suggestive of acute ischaemia [21]. Smith et al. [21] found upright T-waves in V1 and V1 > V6 occurred at greater frequencies in acute myocardial infarction (AMI) (39%), as opposed to benign early repolarisation (15%). The authors made no comment as to whether this was statistically significant, nor did they detail the arteries most commonly involved.

7. Conclusion

There is growing evidence that patients may benefit from urgent PCI when presenting with STEMI-equivalent ECG changes in the context of ACS. However, further research is needed to establish whether there would be a morbidity and mortality benefit derived from the incorporation of some or all of these STEMI-equivalent changes in urgent PCI activation guidelines.

Reference:


