Country cardiograms case 61: Answer

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The electrocardiogram (ECG) obtained on admission (Fig. 1) shows sinus arrhythmia, with a mean rate of 69 per minute. The PR interval is 0.20 seconds, QRS duration 0.10 seconds and QT interval 0.39 seconds. There is abnormal R wave progression from lead V1 to lead V2, and abnormal Q waves are present in leads V2–V4. ST-segment elevation of 1 mm is present in lead aVL. Reciprocal ST-segment depression is present in leads II, III and aVF. Slight ST-segment elevation is present in lead aVR. The ST segments in leads V2–V5 slope steeply upward, from an initial depressed J point of –2 mm in leads V3–V5. Very tall, symmetric (but not narrow) T waves are present in leads V2–V4.

The patient was assessed as having an acute coronary syndrome. Oxygen, acetylsalicylic acid, nitroglycerine, morphine, clopidogrel, atorvastatin and enoxaparin were administered in accordance with standard protocols. The question that then needed to be addressed was, "Does the patient meet the criteria for thrombolysis?" In such a remote setting, every ST-elevation myocardial infarction (STEMI) presentation will far exceed the recommended 120-minute door-to-needle time for percutaneous coronary intervention (PCI).

STEMI is defined as myocardial ischemia requiring immediate reperfusion therapy. The specifics, which continue to evolve, are currently defined as:[1]

- Appropriate symptoms *and*
- ST-segment elevation of 1.0 mm or

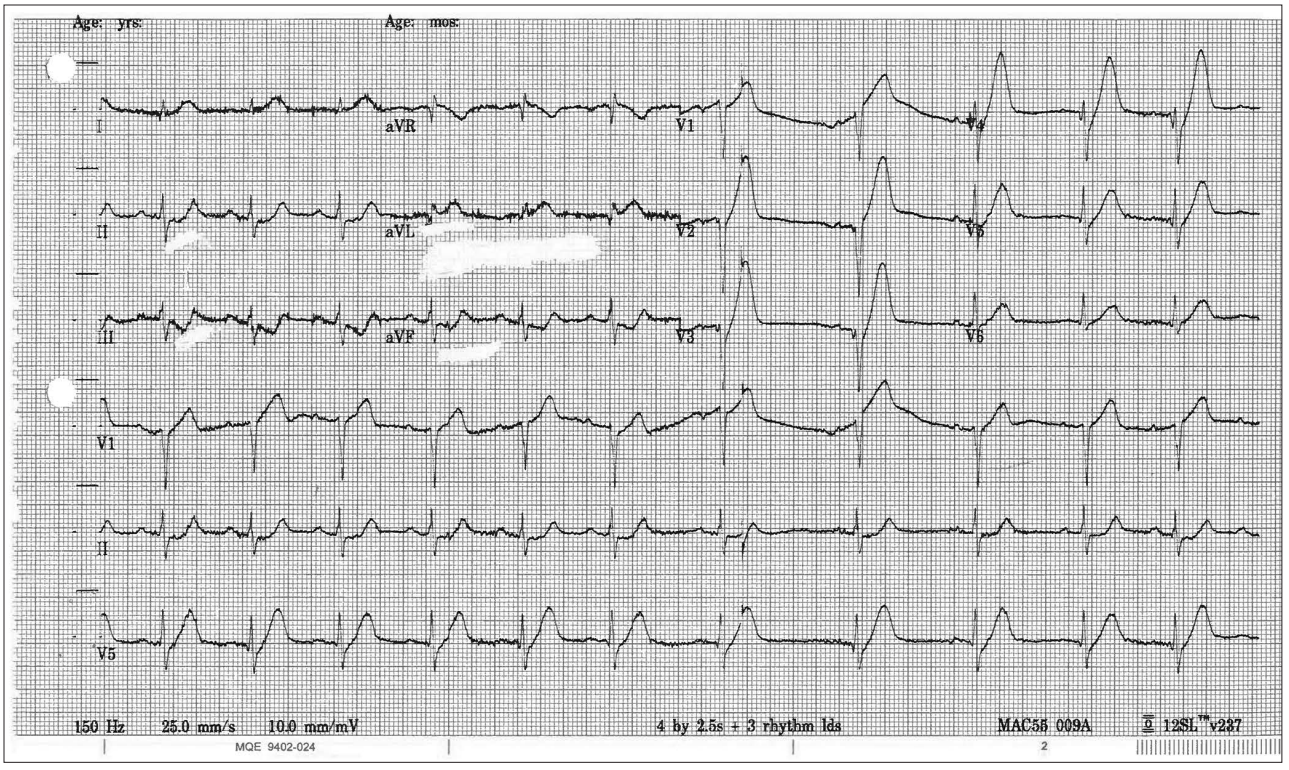

Fig. 1: Electrocardiogram of a 55-year-old man brought to a rural emergency department with retrosternal pain, shortness of breath and sweating.

greater in 2 contiguous leads, aside from leads V2–V3 (≥ 2.0 mm is required in men aged 40 years or more, ≥ 2.5 mm in men younger than 40 years and ≥ 1.5 mm in women).

Patients with ST-segment depression or abnormal T wave changes with elevation of myocardial enzyme levels are considered to have non-STEMI and are not candidates for emergent thrombolysis or PCI. ECG changes in the absence of enzyme level elevation represent unstable angina. New, or suspected new, left bundle branch block was removed from the 2012 criteria, given its documented low accuracy as a stand-alone ECG finding.[1]

In Figure 1, strict thrombolysis criteria, as defined above, are not met, yet the chest pain presentation, the ST-segment elevation in lead aVL, the reciprocal ST-segment depression in the inferior leads, the anterior Q waves and the very tall, "hyperacute" T waves in leads V2–V4 combine to suggest a significant event that probably involves the left anterior descending artery and may represent an evolving STEMI. Are there any so-called STEMI equivalents to be found in Figure 1 that might indicate the need for thrombolysis?

The earliest sign of myocardial ischemia has long been recognized as hyperacute T waves.[2] These have no formal definition but are generally symmetric, are abnormally large relative to the QRS complex and have broad peaks. This is in contrast to the T waves of hyperkalemia, which are typically more narrow and sharply peaked. (In this case, we rapidly obtained a serum potassium level of 3.2 mmol/L; the normal reference range is 3.5–5.1 mmol/L).

Can hyperacute T waves be used as an ancillary criterion for thrombolysis when other criteria are not met, provided other causes for tall T waves (hyperkalemia, early repolarization pattern) have been excluded? Hyperacute T waves by themselves generally indicate impending STEMI, but most authors recommend repeating ECG and waiting for traditional STEMI criteria to evolve before proceeding to thrombolysis.[1,3]

However, hyperacute T waves may be seen in association with other findings that, according to some authors, call for reperfusion therapy.[3] These are known as the de Winter ST–T wave complex. The combination of J point depression, steeply upsloping ST segments leading into symmetric, tall, relatively wide T waves and the possibility of ST-segment elevation in aVR is consistent with this pattern, which was described in 2008.[4] It represents significant ischemia in the territory of the left anterior descending artery, although arterial occlusion may be incomplete. This pattern sometimes persists on the ECG until PCI is performed or it may evolve into a typical STEMI pattern.[5] The pathophysiologic features of the ECG waveform most likely lie in significant endocardial ischemia with epicardial sparing.[6]

The de Winter ST–T wave complex, which was identified retrospectively based on angiographic findings, has been postulated as a STEMI equivalent and may be present in up to 2% of anterior myocardial infarctions.[4] Other so-called STEMI equivalents include ST-segment elevation of 0.5 mm or greater in posterior leads (V7–9) with anterior depression,[1,3] ST-segment elevation of 0.5 mm or greater in right-sided precordial leads (V4R) with inferior ST-segment elevation[1] and meeting the Sgarbossa criteria in left bundle branch block.[3]

Pathologic Q waves are classically thought to represent completed transmural myocardial infarction. However, more than 50% of STEMI presentations have Q waves on the initial ECG, and these are potentially reversible with reperfusion.[7] Q waves per se are not recognized as criteria for reperfusion therapy, although they are an ominous prognostic sign in acute ischemia.

Thrombolytic therapy in a rural or remote setting can confer major benefits but can cause serious, potentially life-threatening complications. Expanding the criteria for thrombolysis therefore needs to be based on sound evidence that its benefits outweigh its risks. This would include information on the sensitivity and specificity of the changes seen in the de Winter ST–T wave complex. Until such high-quality evidence is available, it seems premature to advise thrombolysis in such cases.

Applying such considerations in this case, the de Winter ST–T wave complex is a cause for significant concern. While considering this, we repeated ECG (Fig. 2). Based on these recordings, the standard criteria for thrombolysis are now clearly met, with ST-segment elevation present in contiguous leads I and aVL, and leads V2–V4. Abnormal Q waves remain in leads V2–V3. The ST-segment elevation previously noted in lead aVR has resolved.

Tenecteplase was administered. Within minutes, there was evidence of effective reperfusion: the patient's pain resolved, reperfusion arrhythmia developed (accelerated idioventricular rhythm),

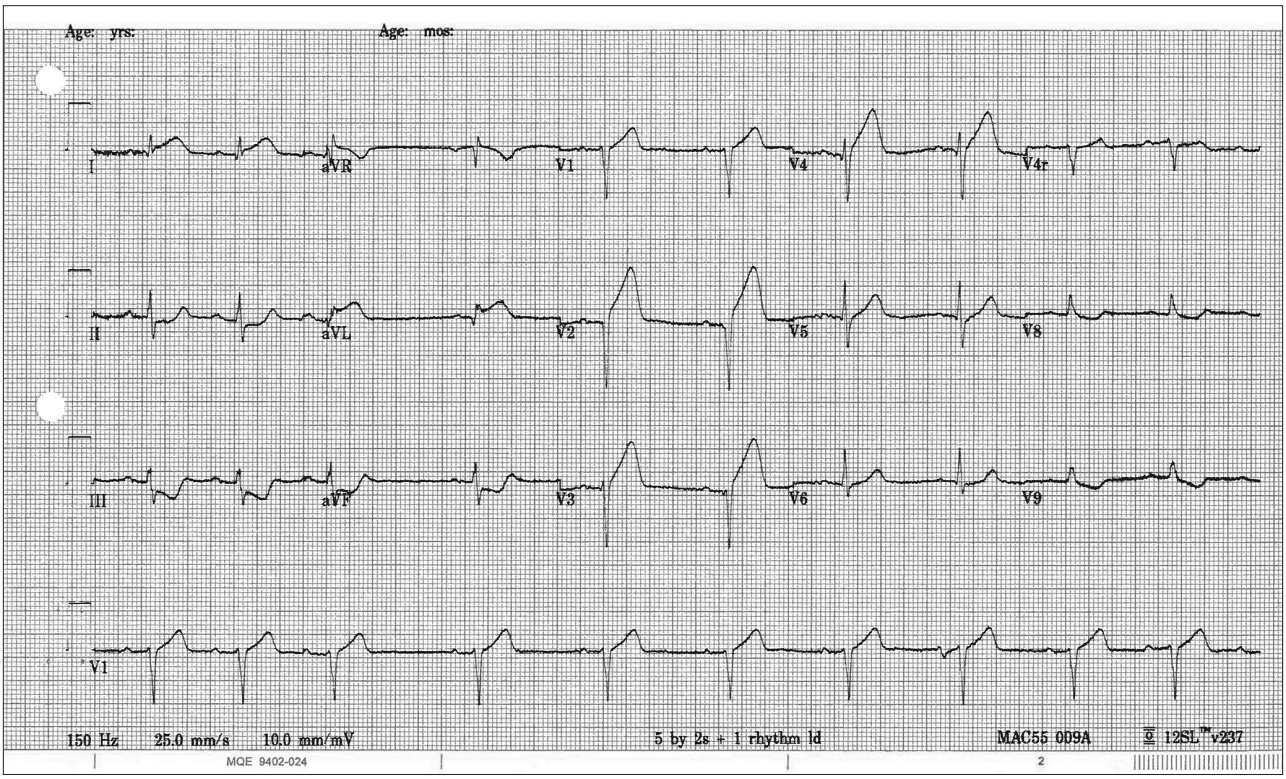

Fig. 2: Repeat electrocardiogram, showing ST-segment elevation in contiguous leads I and aVL, and leads V2–V4. Abnormal Q waves remain in leads V2–V3. The ST-segment elevation previously noted in lead aVR has resolved.

and the ECG returned to normal, with resolution of all ST segment and T wave changes and reappearance of normal R wave progression in leads V1–V4.

The troponin level was initially normal (< 50 ng/L); a level of 2192 ng/L was later obtained. On arrival at a tertiary centre several hours later, the patient underwent immediate PCI, and a single drug-eluting stent was placed in the left anterior descending artery. He was discharged 3 days later.

Criteria for immediate reperfusion continue to evolve. Hyperacute T waves by themselves generally indicate impending STEMI, but ECG should be repeated to look for traditional STEMI criteria before proceeding to thrombolysis.

Angiographic evidence suggests that reperfusion therapy may be indicated when de Winter ST–T wave complexes are seen. However, this opinion is dependent on having PCI capabilities, which the rural emergency department does not have access to. A cautious approach would require the publication of high-quality evidence showing the benefits of thrombolysis before expansion of its criteria is accepted. Given this uncertainty, frequent repeat ECG is advisable to look for the development of traditional STEMI criteria for thrombolysis, along with consultation with a cardiologist.

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For the question, see page 21.

Competing interests: None declared.