Country cardiograms case 52: Answer

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The electrocardiogram (ECG) shown in Figure 1 (on page 151) shows sinus bradycardia at a rate of 52 beats/min. There are prominent T waves in precordial leads V2 to V6 and ST-segment depression in limb leads III and aVF. There is no evidence of ST-segment elevation that would meet diagnostic significance. No previous ECGs were available for comparison. The prominent nature of the anterior T waves was suggestive of hyperacute T waves. This interpretation was supported by the presence of inferior ST-segment depression, which was interpreted as representing reciprocal changes. Based on these findings and the clinical history, acute myocardial infarction (MI) in the anterior distribution was diagnosed. The patient was sent for emergent coronary angiography.

Hyperacute T waves refer to positive-deflection, tall-amplitude, primary T wave abnormalities associated with acute MI. A T wave amplitude that is less than two-thirds of the R wave amplitude is generally accepted as normal. More specifically, the T waves should be less than 0.5 mV in limb leads and less than 1.5 mV in the precordial leads. However, factors such as body habitus and lead placement can make these criteria unreliable. In practice, making the correct diagnosis requires a high index of suspicion while placing the ECG findings in the clinical context.

The modern classification of MI makes a distinction between those with ST-segment elevation (i.e., representing transmural ischemia) and those without. This classification has important diagnostic, treatment and prognostic utility.[1] In the setting of an acute ST-segment elevation MI (STEMI), the most recognizable ECG feature is elevation of the ST segment. However, hyperacute T waves are often the first ECG sign of complete coronary artery occlusion and may be present within minutes of symptom onset.[2] In most

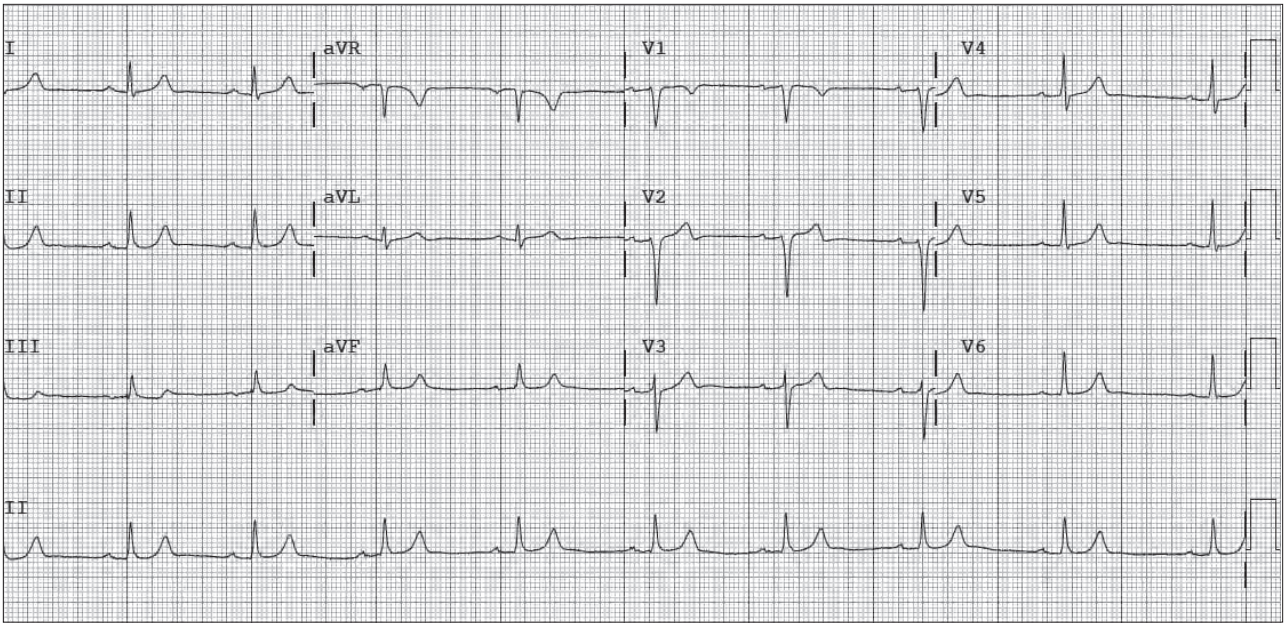

Fig. 2. Repeat electrocardiogram after percutaneous coronary intervention.

patients with hyperacute T waves, these changes are transient, and subsequent ECG analysis shows evolution of typical ST-segment elevation. However, in 2%–8% of patients with a transmural anterior MI, hyperacute T waves persist without the evolution of ST-segment elevation.[3,4] Previous evidence suggested that patients with only hyperacute T waves on ECG may have collateral circulation and, as a result, have less cardiac necrosis and lower risk of mortality.[3,5,6] However, more recent evidence from this patient population using cardiac magnetic resonance imaging has shown typical transmural necrosis — the same changes observed in patients with STEMI.[7] As a result, this ECG pattern requires prompt recognition and revascularization.

The differential diagnosis for prominent T waves commonly includes acute MI, hyperkalemia, left ventricular hypertrophy and benign early repolarization. The term hyperacute specifically refers to the T wave associated with acute MI. This T wave is often symmetric and broad-based. Importantly, it may be associated with ST depression in reciprocal ECG leads in much the same manner as might be seen with ST-segment elevation. In contrast, the T wave of hyperkalemia is symmetric, tall and narrow, and peaked at the apex. In left ventricular hypertrophy, the T wave has a concave upslope with a strain pattern, and the voltage criteria of left ventricular hypertrophy are also observed. In benign early repolarization, the T wave has a concave upslope, is asymmetric, appears similarly in all leads and has associated ST elevation.[2,8]

Hyperacute T waves on ECG have been found to be associated with complete occlusion of the proximal left anterior descending coronary artery.[4–6,9] Besides hyperacute T waves, biphasic T wave changes have also been associated with disease of the proximal left anterior descending coronary artery.[10] Reciprocal ST-segment depression in the inferior leads may not always be present but, when present, can aid in making the correct diagnosis. However, if the ST-segment depression is misinterpreted, this could lead to a misdiagnosis of a non-STEMI, particularly if the T wave changes go unrecognized. Current guidelines from the Canadian Cardiovascular Society do not recommend fibrinolytic therapy in the absence of ST-segment elevation.[11] From a rural medicine perspective, it is therefore critical to seek expert consultation if this pattern is identified on ECG in a patient with chest pain. Subsequent ECGs should be obtained to see if frank ST elevation develops, which is an indication of fibrinolysis. However, one must consider that ST-segment elevation may not occur, and timely transport to a site equipped with cardiac catheterization facilities must be arranged. Urgent referral to a centre equipped for catheterization should be considered for patients with refractory cardiogenic shock or ventricular arrhythmias, in addition to patients with acute coronary syndrome.

Our patient underwent emergent cardiac catheterization, which revealed total occlusion of the proximal left anterior descending coronary artery. He received stenting, with excellent results. An ECG taken after percutaneous coronary intervention showed resolution of the hyperacute T waves and normalization of the inferior ST segments (Fig. 2). The patient did well and had only minimal elevation of cardiac enzymes. Echocardiography revealed only a mild reduction in left ventricular systolic function, with an ejection fraction of 51%. This good clinical outcome was likely directly related to prompt diagnosis and treatment.

For the question, see page 151.

Competing interests: None declared.

REFERENCES

1. Fuchs RM, Achuff SC, Grunwald L, et al. Electrocardiographic localization of coronary artery narrowings: studies during myocardial ischemia and infarction in patients with one-vessel disease. *Circulation* 1982;66:1168-76.
2. Somers MP, Brady WJ, Perron AD, et al. The prominant T wave: electrocardiographic differential diagnosis. *Am J Emerg Med* 2002; 20:243-51.
3. Bimbaum Y, Sclarovsky S, Blum A, et al. Prognostic significance of the initial electrocardiographic pattern in a first acute anterior wall myocardial infarction. *Chest* 1993;103:1681-7.
4. Verouden N, Koch K, Peters R, et al. Persistent precordial "hyperacute" T-waves signify proximal left anterior descending artery occlusion. *Heart* 2009;95:1701-6.
5. Sagie A, Sclarovsky S, Strasberg B, et al. Acute anterior wall myocardial infarction presenting with positive T waves and without ST segment shift. *Chest* 1989;95:1211-5.
6. Zhong-qun Z, Nikus KC, Sclarovsky S. Prominent precordial T waves as a sign of acute anterior myocardial infarction: electrocardiographic and angiographic correlations. *J Electrocardiol* 2011;44:533-7.
7. Zorzi A, Perazzolo Marra M, Migliore F, et al. Interpretation of acute myocardial infarction with persistent "hyperacute T waves" by cardiac magnetic resonance. *Eur Heart J Acute Cardiovasc Care* 2012;1:344-8.
8. Genzlinger M, Eberhardt M. Analyzing prominent T waves and ST-segment abnormalities in acute myocardial infarction. *J Emerg Med* 2012;43:e81-5.
9. de Winter RJ, Verouden NJW, Wellens HJJ, et al. A new ECG sign of proximal LAD occlusion. *N Engl J Med* 2008;359:2071-3.
10. de Zwaan C, Bar FWHM, Wellens HJJ. Characteristic electrocardiographic pattern indicating a critical stenosis high in left anterior descending coronary artery in patients admitted because of impending myocardial infarction. *Am Heart J* 1982;103:730-6.
11. Antman EM, Anbe DT, Armstrong PW, et al. ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction — executive summary. *Circulation* 2004;110:588-636.