

Country cardiograms case #67: Answer

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The electrocardiogram (ECG) shown in Figure 1 displays marked sinus arrhythmia, with a mean rate of 63/min. PR interval is 0.16 s, QRS duration is 0.08 s, QT interval is normal and QRS axis is 35°. P-wave morphology is within the normal limits.

The obvious abnormality lies in the ST–T configuration. Coved ST-segment elevation is evident in the inferior leads, with as much as 2 mm of elevation in leads III and aVF, and about 1 mm of elevation in lead II. Reciprocal ST-segment depression is evident in leads I and aVL. In a patient presenting with severe chest pain, this provides strong evidence of inferior STEMI. In a rural setting, with an anticipated long transfer time to a tertiary centre, a rapid assessment is required to establish that there are no contraindications to thrombolysis, aiming for as short a ‘door-to-drug time’ as possible.

With any inferior myocardial infarction, there are a number of ST–T patterns that should be looked for, which can provide helpful information. ST-segment elevation is often seen in leads V5 and V6 and indicates the involvement of the lateral myocardial wall. ST-segment depression in leads V1–V3 (especially if associated with tall R waves and tall T waves) indicates posterior wall involvement. These patterns are consistent with the anatomy of the right coronary artery, for example, the posterior descending artery supplies the posterior wall of the left ventricle. Another arterial branch supplies the atrioventricular (AV) node and the Bundle of His, and evidence for AV

block should therefore also be sought on the ECG.

In addition, elevation in V1, although not as frequent a finding, can be the only suggestion on the 12-lead ECG of right ventricular myocardial infarction (RVMI), as this lead has the capacity to ‘face’ the right ventricle. In Figure 1, while there is unfortunately some artefact in V1 following the first two QRS complexes, there is nonetheless an impression of ST-segment elevation, and this is clearly evident after the third QRS complex. In all cases of inferior myocardial infarction, a 15-lead ECG is required, mostly to assess the possibility of RVMI in right-sided lead V4R (V8 and V9 are also used and can corroborate the presence of posterior STEMI, although these leads do not usually provide information not already available on the 12-channel ECG).

Attributing this pattern to RVMI in the context of inferior STEMI is much more plausible than suggesting a septal STEMI, which also causes ST-segment elevation in V1: branches of the right coronary artery supply the right ventricle, whereas the intraventricular septum is supplied by the left coronary artery. The presence of simultaneous acute pathology in both left right and left coronary arteries is extremely unlikely.

Figure 2 shows a 15-lead ECG that includes leads V4R, V8 and V9. Lead V4R shows a remarkable degree of ST-segment elevation, as much as 5 mm, providing unequivocal evidence of right ventricular STEMI. Slight ST-segment depression is evident in leads V8 and V9, which is

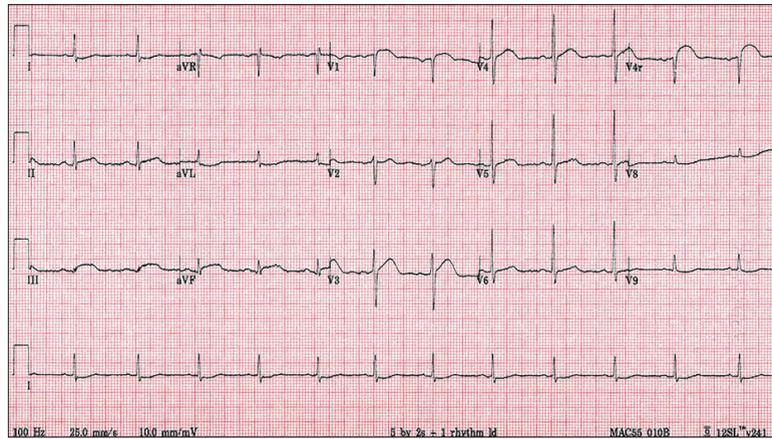


Figure 2: 15-Lead electrocardiogram, with leads V4R, V8 and V9.

essentially a reciprocal change of the ST-segment elevation which is clearly present in V1.

RVMI is an important condition to identify if it is present. In addition to the ECG findings, clinical features should be sought, including the triad of distended neck veins, clear lung fields and hypotension.¹ Its haemodynamic effects can readily be exacerbated by medications which are commonly used in the treatment of ischaemic chest pain, including nitroglycerin, morphine and beta blockers. Sublingual nitroglycerine, in particular, rapidly delivers a large drug dose, and its administration can lead to precipitous hypotension, bradycardia and syncope. The risk of this scenario provides a good rationale for first ensuring a functioning intravenous line. These patients are extremely 'volume sensitive' and respond well to boluses of intravenous fluid; inotropes may be required to treat hypotension.

RVMI occurs in up to 50% of cases of inferior myocardial infarction, although the ECG findings are seldom as dramatic as those shown in Figures 1 and 2.² Compared with cases of inferior myocardial infarction without RVMI, there is a 2.6-fold increase in mortality.³ Every effort should therefore be made to provide definitive management (e.g., percutaneous coronary intervention) as soon as possible. In a rural or remote setting, thrombolysis remains an effective tool before emergency transfer to a facility capable of performing such intervention.

In this case, following prompt diagnosis, thrombolysis was administered. Substantial resolution of the ST-segment changes was observed, and the patient was airlifted to a major tertiary centre. Coronary angiography showed triple-vessel disease, including an 80% occlusion in the right

coronary artery. Three drug-eluting stents were placed. Nicotine replacement therapy was started.

A 15-lead ECG showing ST-segment elevation in V4R is usually considered to be necessary to diagnose RVMI and treat it appropriately, but, as shown in Figure 1, in some cases, RVMI can be diagnosed or strongly suspected on a 12-lead ECG. Depending on which type of ECG machine is used, the extra time taken to record a 15-lead ECG can vary. A rapid diagnosis of RVMI on a 12-channel ECG can therefore aid in avoiding inappropriate treatment and in preparing for the treatment with intravenous fluid boluses. Health care providers in the emergency room need to be familiar with the correct placement of leads for a 15-lead ECG.⁴

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