ECG Quiz 36

CASSA
CARDIAC ARRHYTHMIA SOCIETY OF SOUTHERN AFRICA
A SPECIAL INTEREST GROUP OF SA HEART ASSOCIATION

ANSWER

(1) OVERVIEW OF THE ECG

The rhythm is regular, around 90bpm. The most obvious feature is widespread ST segment elevation with Q-waves in the inferior and anterolateral leads. There is a dominant R-wave in V1.

MORE DETAILED ANALYSIS OF THE ECG

The P-waves are probably of sinus origin, being positive in II and negative in aVR. The PR interval is about 150ms.

The QRS complexes are wide (about 140ms), but measurement is made difficult by the marked ST segment elevation. The rsR pattern in V1 is compatible with complete right bundle branch block (RBBB).

There is right axis deviation, +120°. The main causes of right axis deviation are:

- Right ventricular hypertrophy (RVH)
- Lateral myocardial infarction
- Left posterior fascicular block (LPFB)
- WPW pattern

The combination of right axis deviation and a dominant R in V1 suggests RVH, but bifascicular block due to a combination of LPFB and RBBB can produce a similar pattern. The terminal R in V1 and wide QRS in this case are more suggestive of RBBB, but the differentiation of right axis due to LPFB from that due to RVH cannot be made with certainty from the ECG alone. Further clinical data are essential.

The right axis deviation is not solely due to lateral infarction on this ECG, as aVL has an rS pattern, rather than Qr.

WPW pattern is excluded by the normal PR interval, absence of delta waves and predominantly terminal QRS delay.

Small, but broad Q-waves are present in the inferior leads as well as V2 and V3. The complexes are mainly negative from V4 to V6, but start with tiny R-waves.

There is ST segment elevation of 6-7mm in the inferior leads as well as V2, V3, and to a lesser extent, V4.

The above combination of features suggests acute transmural infarction of both the inferior and anterior walls of the left ventricle. In addition, there is RBBB and right axis deviation due either to LPFB (bifascicular block) or RVH.

While acute pericarditis could explain ST segment elevation in more than one coronary artery territory, it would not account for the Q-waves and lack of R-wave progression. In addition, the PR segments are not depressed in II or elevated in aVR, which are usually seen in acute pericarditis.

The best ECG diagnosis would therefore appear to be (c), Acute anterior and inferior STEMI with RBBB. However, simultaneous infarction in 2 separate coronary territories is a very unusual clinical situation (see below), so it is worth considering an alternative explanation.

Shortly after the above ECG, another recording was made (Figure 1B), which showed an identical pattern in the chest leads, but a very different one in the limb leads. The right axis deviation has been replaced by left axis deviation of -60°, a shift of 180°! There is now ST elevation in I and aVL and marked reciprocal ST depression in III and aVF. This is much more consistent with the anterior infarction which is apparent in the chest leads. All subsequent ECGs follow the same pattern.

How, then, can we explain this?

Figure 1 shows how it happened. In the first ECG (A), the left arm and left leg electrodes were transposed. The effect of this is to transform lead I to lead II and lead aVL to aVF. At the same time, the polarity of lead III is reversed, giving rise to the apparent dual territory infarction (B).[1] Note that, while the P-wave axis changed from +60° in the correctly recorded tracing (A), to 0° when the leads were transposed (B), this is not nearly as noticeable as it would be in the more usual lead error in which the left and right arm leads are transposed.

The correct answer is therefore (g): Other.

The answer to (2) is (b): Repeat the ECG first before thrombolytic therapy or primary PCI (if available).

This only takes a few minutes to do and it resolved the question of infarction in 2 territories immediately.

Ischaemia or infarction in 2 non-adjacent vascular territories does, however, occasionally occur.

The ECG in Figure 2 was recorded on admission of a 42-year-old man with severe chest pain accompanied by sweating. The pattern is similar to that of the subject of this quiz, but shows even more dramatic ST elevation, reaching 6mm in II and 20mm in V4. The QRS complexes are narrow and no Q-waves have developed, as yet. The frontal plane QRS axis is normal

(+45°). Two subsequent ECGs, taken over a short period, were identical.

At angiography, there was a severe lesion in the mid left anterior descending artery (LAD) and a stent was successfully inserted. The LAD was seen to wrap right around the apex ("wrap-around LAD"), such that it supplied a large part of the inferior wall in addition to the anterior wall (Figure 3).

The ECG after the procedure showed resolution of the ST segments with anterior T-wave inversion, but no pathological Q-waves.

The most important difference between the ECG of the second case compared to the first, is the lack of ST depression in aVL and the presence of a raised ST segment in lead I. If the left arm and left leg leads had been transposed, ST elevation would have still been present in both leads I and II.

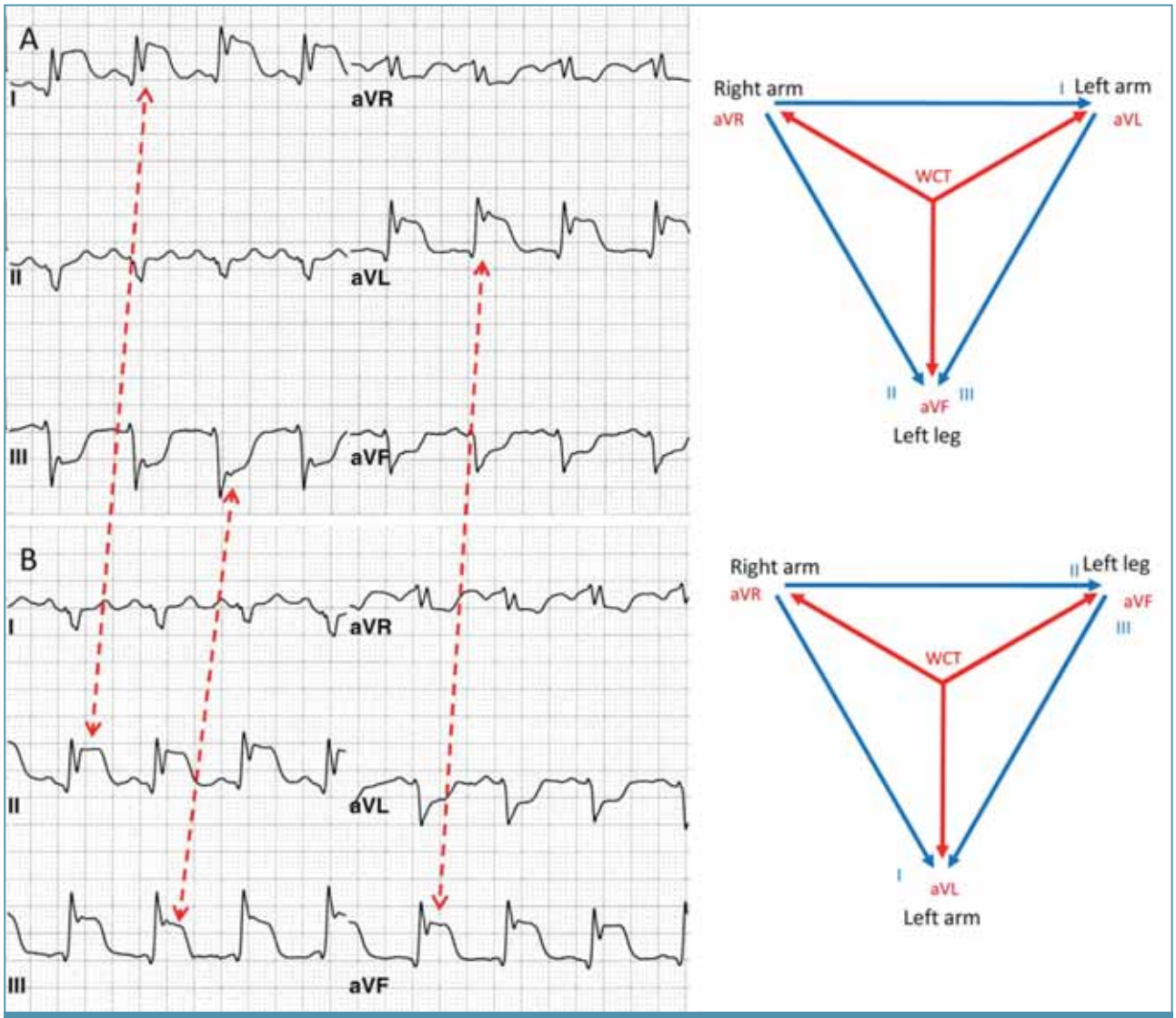

FIGURE 1: A: ECG with correctly placed electrodes.
B: Admission ECG. The left arm and left leg electrodes are transposed. Lead I becomes lead II, aVL becomes aVF and the polarity of lead III is reversed.

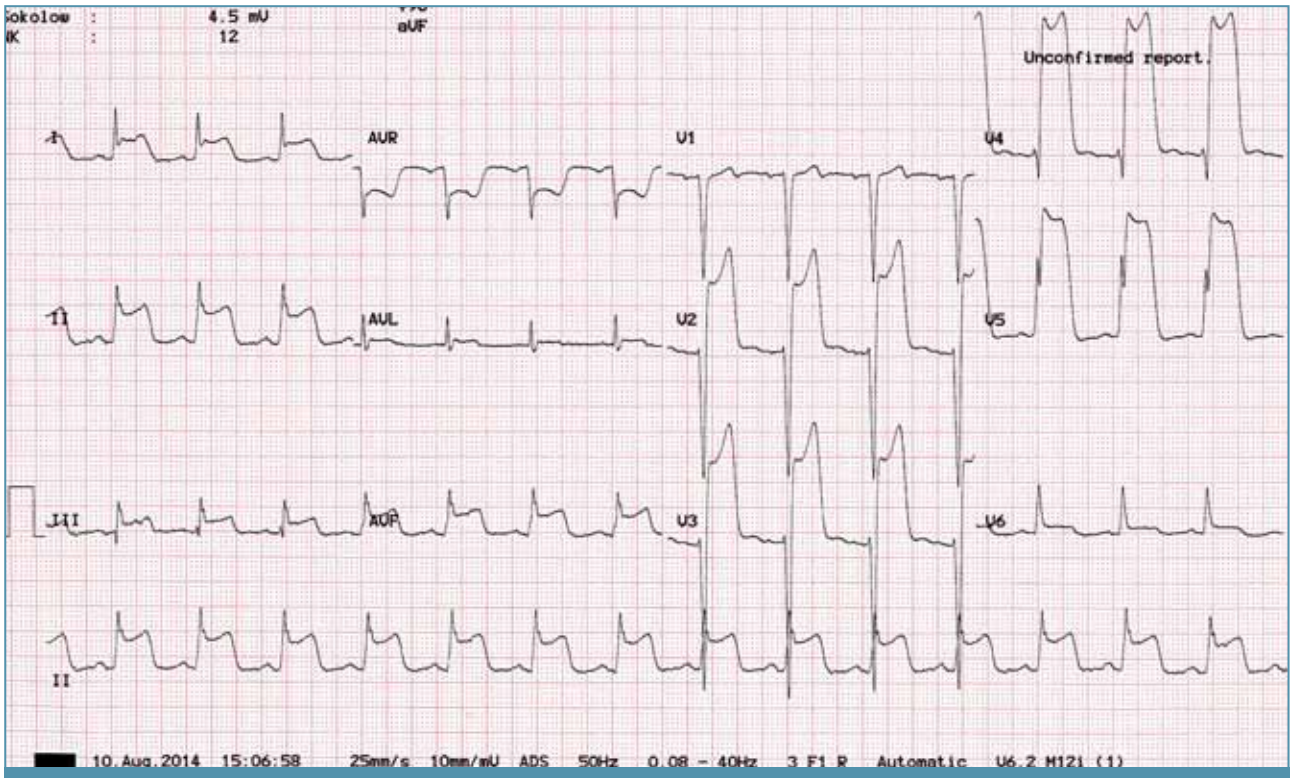

FIGURE 2: ECG of the second patient, with ischaemia genuinely affecting both the anterior and inferior walls.

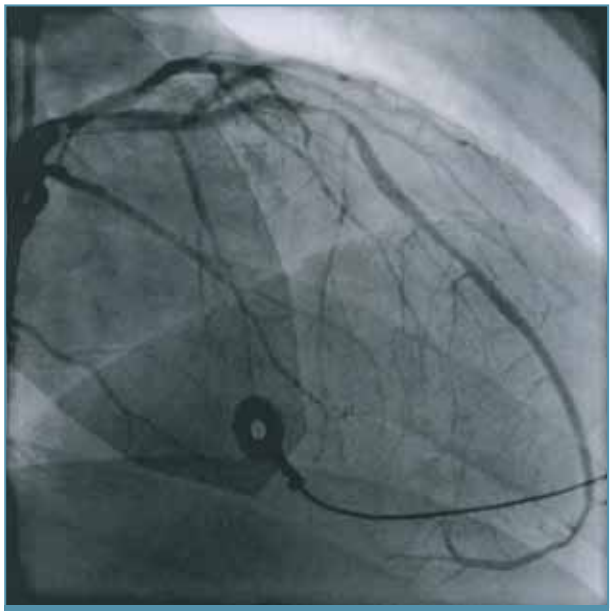

FIGURE 3: Left coronary angiogram which shows a "wrap-around" LAD supplying the anterior and inferior walls of the left ventricle. The culprit lesion is seen in the mid-LAD.

LESSONS AND CONCLUSIONS

- Acute STEMI rarely affects 2 coronary artery territories simultaneously.
- Consider incorrect lead placement when confronted with unusual ECG abnormalities.

REFERENCES

1. Scott Millar RN, Makanjee B. Masking inferior infarction by anterior myocardial injury – alternative explanation. Circulation 2007;115:e202.

Conflict of interest: none declared.

ECG and **QUESTION** on page 208