ECG Quiz 38

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

ANSWER

(1): OVERVIEW OF THE ECG

There is a regular bradycardia, around 54bpm. The most striking feature is deep T-wave inversion in leads V1 to V4. There are many possible causes for this pattern.

MORE DETAILED ANALYSIS OF THE ECG

The rhythm is regular at 54bpm. The P-waves are flat and inconspicuous, but appear to be present before each QRS. The P-wave axis is about +30°, so they are probably of sinus origin. There is no clear evidence of AV block. The PR interval is around 180ms (measured in lead III).

The QRS is just under 120ms with no septal q in V5 or V6, but the pattern is not typical of LBBB. There is terminal slurring of the QRS in V5-6 consistent with an early repolarisation pattern. The QRS axis is normal, +50°, and there are no pathological Q-waves. The QRS voltage is normal.

The ST segments are not deviated. The frontal plane T-wave axis is +90°, so the QRS-T angle is borderline (60° is the upper limit of normal). The T-waves are inverted from V1 to V4. The QT interval is markedly prolonged at 700ms, giving a QTc of 661ms (Bazett).

This pattern of deep anterior T-wave inversion, with or without QT prolongation, can occur in a large number of different conditions, including all of those mentioned in the question.

The correct answer is therefore (e): All of the above.

(2): ELUCIDATING THE CAUSE

The key to the diagnosis is the clinical context. A second ECG within a short time is unlikely to be significantly different. A stress ECG may be impractical, uninterpretable or dangerous. Investigations, such as coronary angiography or cerebral imaging, should be reserved for the appropriate clinical circumstances.

The answer to (Question 2) is therefore (a): A detailed history and physical examination.

This ECG was recorded from a 35-year-old woman admitted in a coma due to subarachnoid haemorrhage. While the sinus bradycardia and markedly prolonged QT interval are typical of intracranial pathology with raised intracranial pressure due to Cushing's reflex, they are not unique (Figures 1 and 2).

T-WAVE ABNORMALITIES

The T-wave recorded on the surface ECG reflects total ventricular repolarisation, a complex process influenced by the transmural dispersion of repolarisation times from endocardium to epicardium. M cells, residing deep in the myocardium, differ from both endocardial and epicardial cells in their repolarisation times and in their response to changes in rate and to drugs or other agents which prolong the action potential.[1]

In most ECG leads, T-wave polarity is the same as that of the QRS, indicating that ventricular depolarisation and repolarisa-

Primary anterior T-wave inversion: (examples – Figure 1)
• Normal variant
• Athlete's heart
• Wellens' syndrome (acute anterior wall ischaemia)
• Prinzmetal's variant angina (after relief of coronary spasm)
• Takotsubo cardiomyopathy
• Pulmonary embolus
• Complete heart block
• Hypokalaemia
• Hypertrophic cardiomyopathy
• Arrhythmogenic right ventricular cardiomyopathy (ARVC)
• Class III antiarrhythmic drug
• Acute intracranial pathology (e.g. subarachnoid haemorrhage, trauma)
• Phaeochromocytoma
• Pulmonary oedema
• Myocarditis
• Electroconvulsive therapy
• Cardiac sarcoidosis
Secondary anterior T-wave inversion: (examples – Figure 2)
• Right bundle branch block
• Right ventricular hypertrophy
• Pre-excitation
• Left ventricular pacing
• Post-pacing
• Post tachycardia

tion waves travel in opposite directions. However, this relationship is easily upset, resulting in the ubiquity and lack of specificity of T-wave abnormalities.

While some degree of T-wave negativity in V1 is common in normal adults, particularly younger adults, the T-waves in V2 and V3 are usually positive by the age of 14 years, despite the predominantly negative QRS complexes in these leads. It is common to have negative T-wave inversion in children <14 years – so called "Juvenile pattern". Negative T-waves, often very deep, can occur in many conditions[2] (see box – not an exhaustive list), of which some will be discussed here. Abnormal T-waves may be primary (related to pathology or primary electrical changes) or secondary to abnormal depolarisation (e.g. bundle branch block or pre-excitation), although this distinction is sometimes artificial.

We will discuss a few of the many conditions causing anterior T-wave inversion in more detail.

NORMAL VARIANT AND "ATHLETE'S HEART"

T-wave inversion in V1-3, without QT prolongation, occasionally occurs in people with no clinical or other detectable evidence of structural heart disease. This includes the "African pattern" described by Grusin[3] from Baragwanath Hospital in 1954 (Grusin Type I) and subsequently shown to be more common in black than white Americans. Elite athletes have a high prevalence of ECG patterns that would be considered abnormal, including anterior T-wave inversion. In a recent study, T-wave inversion was associated with cardiac pathology (most commonly hypertrophic cardiomyopathy) in 45% of athletes with T-wave inversion. The authors concluded that cardiac pathology needs to be excluded in all athletes with anterior T-wave inversion.[4]

Wellens' syndrome

The ECG pattern of deep anterior T inversion, with or without QT prolongation, may be present at admission or thereafter in patients presenting with acute ischaemic chest pain. Wellens'

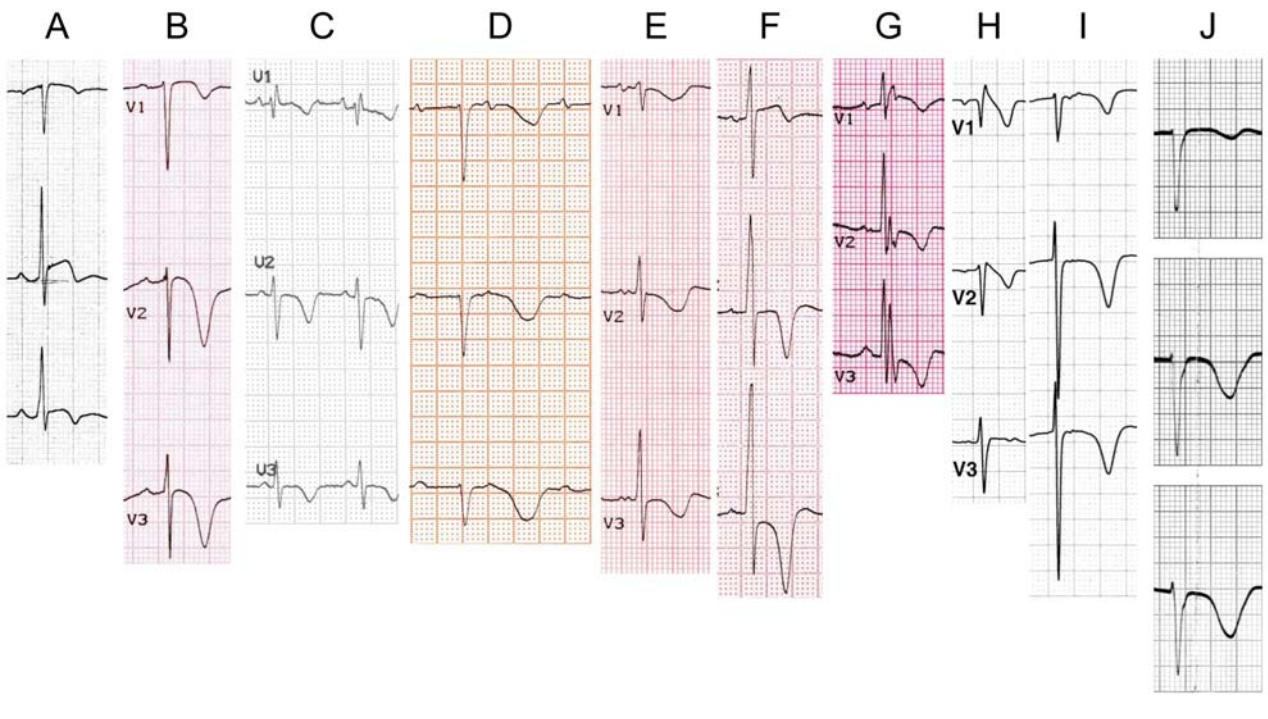

FIGURE 1: Primary T-wave inversion V1-V3.

A: no structural heart disease, B: Wellens' syndrome, C: pulmonary embolus, D: complete heart block, E: hypokalaemia, F: apical hypertrophic cardiomyopathy, G: ARVC, H: Brugada, I: sotalol, J: amiodarone.

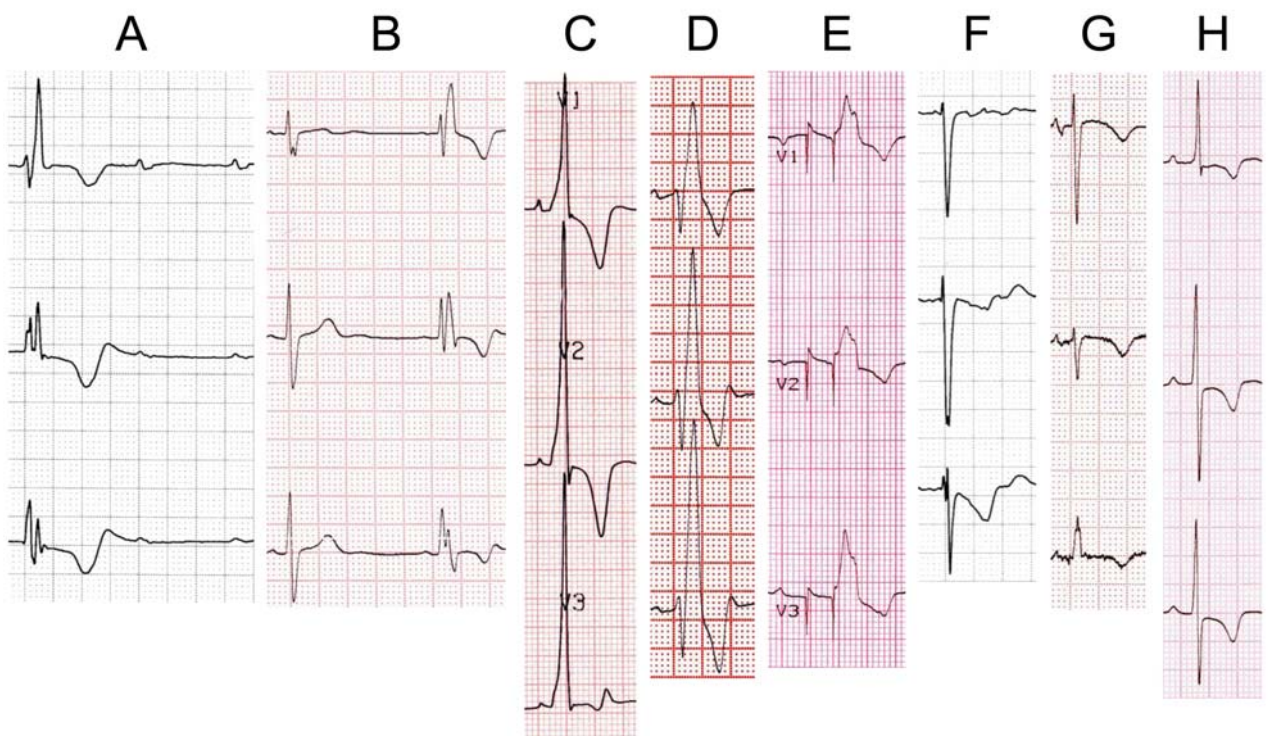

FIGURE 2: Secondary T-wave inversion V1-V3.

A: complete heart block, wide QRS, B: rate-related RBBB, C: WPW pattern, D: Tetralogy of Fallot, RBBB, E: left ventricular pacing, F: post pacing, G: post tachycardia, H: right ventricular hypertrophy.

group from Maastricht described the association of this presentation with a significant proximal stenosis of the left anterior descending coronary artery (LAD) in almost all patients,[5] usually without a significant rise in cardiac enzymes. It is considered an indication for prompt revascularisation because of the high risk of anterior wall infarction within a few weeks after admission.

A similar pattern may occur following relief of proximal LAD spasm in the unusual syndrome of Prinzmetal's variant angina, in the absence of significant fixed coronary stenosis.

Acute intracranial pathology

The ECG of this unfortunate woman shows a pattern commonly seen in severe subarachnoid haemorrhage.[6] It may also occur with head trauma, intracranial tumours and other cerebral insults. While not specific, QT prolongation is almost invariable and bradycardia, due to raised intracranial pressure is common. Other more bizarre patterns may occur, including some which closely mimic acute myocardial infarction (MI). The mechanism is thought to be a sudden release of catecholamines. A similar pattern and mechanism may be seen in phaeochromocytoma and acute pulmonary oedema.

Class III antiarrhythmic drugs

Sotalol and amiodarone, especially in high doses, cause QT prolongation. In some cases, this is accompanied by otherwise unexplained anterior T-wave inversion (see Figure 1 I, from a young woman on sotalol for intractable SVT, and 1 J from a patient with amiodarone toxicity).

Takotsubo cardiomyopathy

In this syndrome of acute stress-induced regional left ventricular dysfunction, the ECG may resemble acute ST elevation MI, but the pattern of anterior T-wave inversion is common.[7]

Arrhythmogenic right ventricular cardiomyopathy (ARVC)

Inverted T- waves in V1 to V3 in patients >14 years is a major criterion for ARVC. T-wave inversion in V1 and V2 or in V4, V5 and V6 is a minor criterion for ARVC. In the ARVC registry of South Africa, T-wave inversion was very common occurring in 76% of definite probands in the registry.[8]

SECONDARY T-WAVE INVERSION

All the above causes are examples of primary T-wave abnormalities which occur without changes in the activation

sequence of depolarisation. Figure 2 shows examples of anterior T-wave inversion which are secondary to QRS abnormalities. T-wave inversion may persist for days, or weeks, after normalisation of the QRS, such as after wide QRS tachycardias or after ventricular pacing. The phenomenon of "T-wave memory" is thought to be a physiological adaptation to changes in rate and activation sequence and is related to different responses of different layers of the myocardium to these changes.[9]

LESSONS AND CONCLUSIONS

- Anterior T-wave inversion, with or without QT prolongation, is a common pattern, seen in many different conditions from normal to life threatening.
- The clue as to the cause resides in the clinical context – the ECG cannot be adequately interpreted in isolation.

Conflict of interest: none declared.

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ECG and **QUESTION** on page 88