ECG Quiz 62

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

ANSWER

OVERVIEW OF THE ECG

This ECG shows a regular, narrow complex rhythm with a ventricular rate of 90bpm. The P waves are compatible with sinus P waves with a P wave axis of 45 degrees. There is ST segment elevation at the J point that is marked in leads V1 to V3 and AVR. There is upsloping ST segment depression in leads II, III, aVF and V4 to V6.

MORE DETAILED ANALYSIS OF THE ECG

Closer inspection of V1, reveals a rSR' complex with a QRS width around 100ms. The marked ST segment elevation of 6mm begins at the top of the R' (or J point) and is downsloping followed by an inverted T wave. J point elevation is also seen in V2 and V3 with 2mm ST segment elevation which is mainly concave followed by upright T waves (Figure 1A).

In right bundle branch block (RBBB) with acute anterior/septal ST segment elevation infarction (STEMI), there may be an initial q wave in V1 followed by a delayed R' which results in a wide QRS (qR') (Figure 1B). There is a usually a distinct transition from the downstroke of the R' wave and the beginning of the ST segment (J point). There is usually convex ST segment elevation, although this may be variable.

The finding of ST segment elevation in V1 - V3 can be a normal finding in men and women (Figure 1C). Men tend to have greater ST segment elevation (1 - 3mm) with the elevation more marked in V2. The prevalence is very high (>90%) in young adults (17 - 24 years of age) which declines with increasing age in one study.[1] The ST segments are usually concave. While V2 and V3 could fit with a normal pattern, V1 does not fit with a normal variant.

Diffuse subendocardial ischaemia (usually with a troponin rise) presenting as a non-ST segment elevation myocardial infarction (NSTEMI) can cause an ECG pattern of diffuse ST segment depression (8 or more leads) with ST segment elevation in V1 and aVR (Figure 1D). ST depression is usually horizontal or downsloping and most prominent in V4 - V6 and ST elevation in AVR is usually larger than V1.

An epsilon wave is a small, low amplitude signal or wave between the end of the QRS complex and the onset of the T wave and does not produce ST segment elevation (Figure 1E). The T wave is often inverted after an epsilon wave. The epsilon wave is caused by low amplitude potentials due to delayed depolarization of "islands" of surviving cardiomyocytes between areas of fibrosis and fat usually in the right ventricle which explains why the wave is best seen in the right precordial leads V1 - V3 and is a feature of diseases like arrhythmogenic right ventricular cardiomyopathy and sarcoidosis (see SA Heart ECG Quiz 56[2]).

The classic Type 1 Brugada pattern is characterised by gradual downsloping or "coved" ST segment elevation with >=2mm at the J-point in >=1 precordial lead (V1 - V3) followed by a negative T wave (Figure 1A). Repolarisation changes (ST segment depression) in the inferior leads and ST segment elevation in aVR are well recognised features and may represent a more severe phenotype. The Type 2 Brugada pattern is characterised by "saddle-back" ST segment elevation with >= 0.5mm ST segment elevation at the J point (Figure 1F). The Type 2 pattern is only suggestive and not diagnostic of the Brugada syndrome.

Although there can be overlap of ECG features with each of the above conditions, the clinical presentation and ECG features best represent the Type 1 Brugada pattern. Sometimes the features can be equivocal and the Brugada pattern can be only made after the exclusion of other causes of ST segment elevation in V1 - V3 and interpreting the ECG in the context of the clinical presentation.

The answer is e. Brugada pattern.

DISCUSSION

In 1992, Brugada, et al. reported a series of 8 individuals who presented with sudden cardiac death with an ECG pattern of right precordial ST segment elevation in a structurally normal heart which has since become known as the Brugada syndrome.[3]

After the initial description, the diagnosis of Brugada syndrome required a typical Type 1 Brugada pattern AND a ventricular arrhythmia OR symptoms of a ventricular arrhythmia. Since 2013, the diagnosis of Brugada syndrome can be made with a

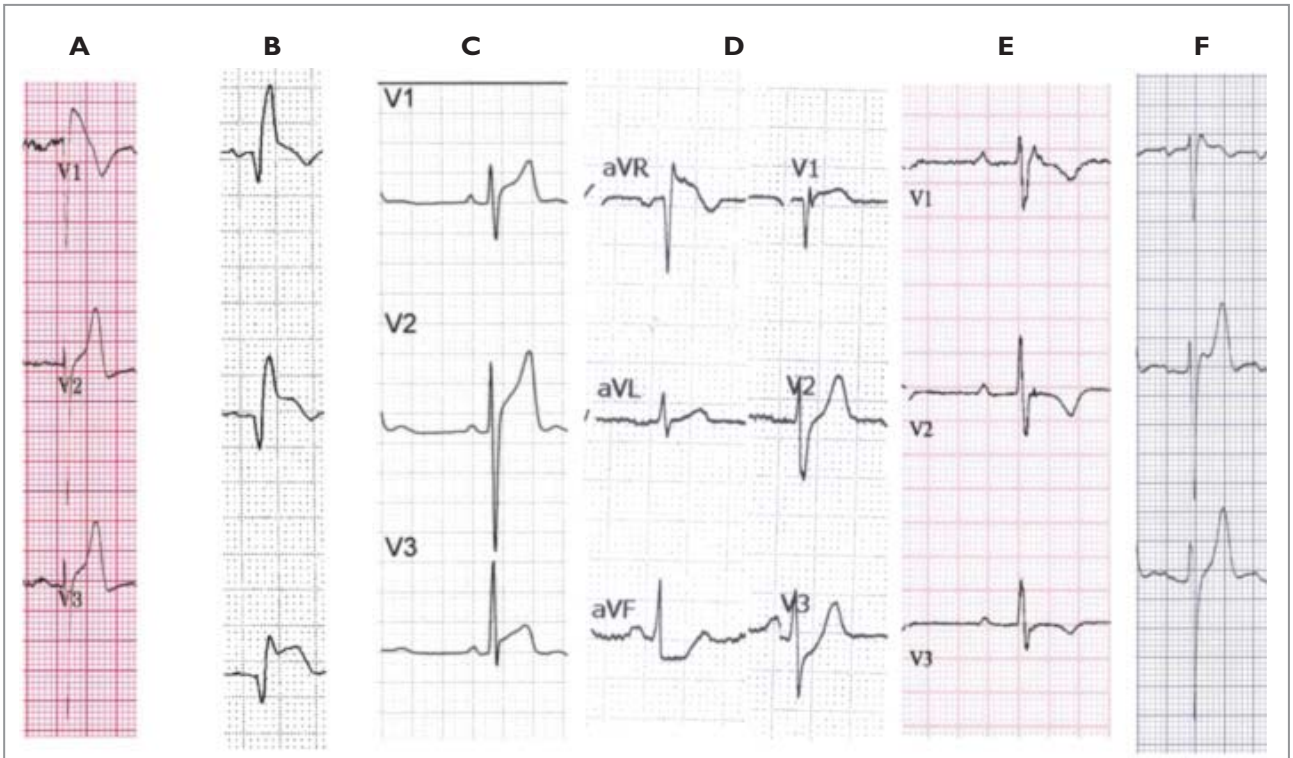

FIGURE 1: A: Brugada pattern Type 1. B: Right bundle branch block and acute STEMI. C: Normal variant ("male pattern"). D: Diffuse subendocardial ischaemia. E: Epsilon wave. F: Brugada pattern Type 2.

typical Type 1 Brugada pattern in the absence of symptoms.[4] This is because most patients with Brugada syndrome remain asymptomatic from ventricular arrhythmias.

The Brugada syndrome has been discovered to be a genetic ion channelopathy usually caused by loss of sodium channel function mutations in the SCN5A gene which codes the alpha subunit of the sodium channel Nav1.5. Around 500 mutations have been described mostly involving the SCN5A gene which accounts for up to 30% of genotyped individuals.[4]

Patients can present with syncope, seizures or agonal nocturnal breathing, polymorphic ventricular tachycardia, ventricular fibrillation or cardiac arrest. Arrhythmias typically occur at night during sleep or periods of fever.

The cellular basis responsible for the typical ECG features of the Brugada pattern is incompletely understood. According to the popular "repolarisation theory", there is a reduction in the inward sodium current in phase 0 of the action potential with an unopposed I_{to} outward current in phase 1 which causes a loss of the dome of the action potential. There also exists a transmural dispersion of repolarisation between the endocardium and epicardium (more I_{to} expressed in the epicardium) which creates the notch in the action potential and the resultant ST segment elevation. This dispersion is most pronounced in the right ventricular outflow tract (RVOT) - hence the localisation of the abnormalities to V1 - V3. Interestingly, ablation of tissue in the epicardium can result in disappearance of the Brugada pattern supporting the cellular theory.[5]

It is well known that the Brugada pattern can be "revealed" by placing the V1 and V2 electrodes in the second or third intercostal spaces. This is because of the anatomical variation that exists between individuals with the RVOT sometimes closest to the second or third intercostal space.

The Brugada pattern is well-known to be dynamic and variable. Vagal stimulation and pyrexia tend to augment the J point and ST segment elevation and catecholamines and exercise tend to have the opposite effect. Sodium channel blockers (like flecainide) can be used as a diagnostic test to unmask the Brugada Type 1 pattern and can convert the Type 2 pattern to a Type 1 pattern. This can be performed in patients with unexplained syncope or cardiac arrest when the diagnosis is suspected.

Documented ventricular arrhythmias with a Type 1 Brugada pattern usually require insertion of an implantable cardioverter defibrillator. Beta-blockers and calcium channel blockers are contraindicated in Brugada syndrome. Quinidine (I_{to} blocking effect is greater than sodium channel blocking effect) has been used to treat recurrent ICD shocks. Drugs that block the sodium channel may precipitate arrhythmias and must be avoided (a comprehensive list can be found on the website: BrugadaDrugs.org).

This patient had no symptoms suggestive of an underlying arrhythmia. He was counselled regarding the immediate treatment of any future pyrexial illnesses with paracetamol, to avoid drugs that worsen the Brugada pattern, and has been told to present promptly if he develops any syncopal episodes.

SUMMARY

The Type 1 Brugada pattern is characterised by gradual downsloping or "coved" ST segment elevation with >=2mm at the J-point in >=1 precordial lead (V1 - V3) followed by a negative T wave.

Many conditions that can cause ST segment elevation in V1 (e.g. RBBB with an acute anterior/septal STEMI, normal variant of ST segment elevation, diffuse subendocardial ischaemic pattern) require exclusion before the diagnosis Brugada syndrome can be confirmed. The clinical context is important when interpreting the ECG and can be useful to exclude these conditions.

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Conflict of interest: none declared.

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