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CME Article

Electrocardiographical case. Asymptomatic patient with ST-segment elevation

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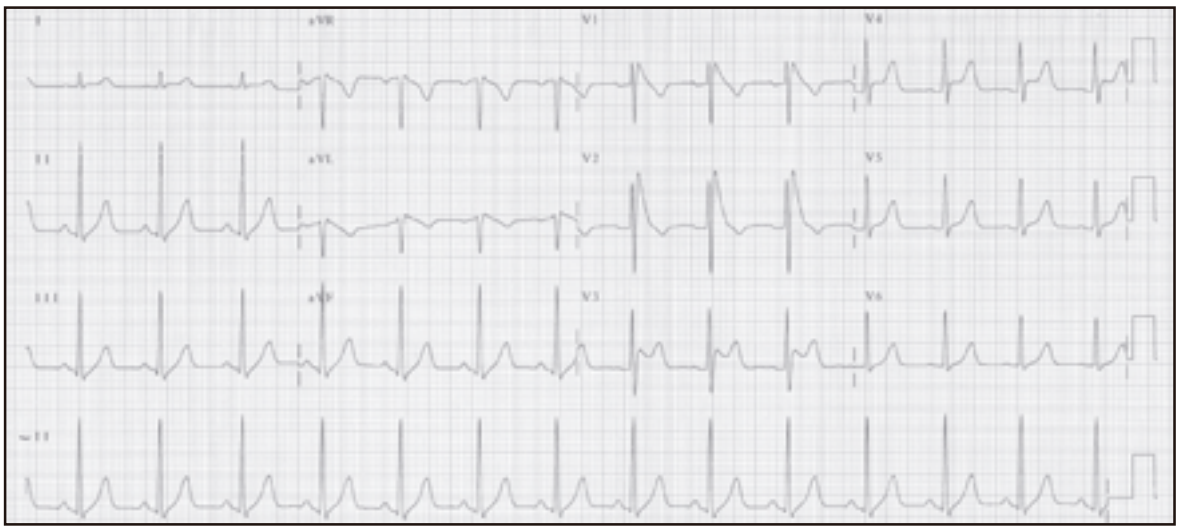

Fig. 1 12-lead ECG.

CLINICAL PRESENTATION

A 46-year-old man complained of recurrent episodes of giddiness which was not associated with chest pain or breathlessness. There was no family history of sudden death. Clinical examination was unremarkable. The 12-lead electrocardiogram (ECG) is shown in Fig. 1. What is the diagnosis?

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Table I. Diffential diagnoses for ST-segment elevation in the right precordial leads[9].

Right or left bundle branch block, left ventricular hypertrophy
Acute myocardial infarction
Left ventricular aneurysm
Acute myocarditis
Right ventricular infarction
Dissecting aortic aneurysm
Acute pulmonary embolism
Various central and autonomic nervous system abnormalities
Heterocyclic antidepressant overdose
Duchenne muscular dystrophy
Friedreich ataxia
Thiamine deficiency
Hypercalcaemia
Hyperkalaemia
Compression of right ventricular outflow tract by metastatic tumour
Cocaine intoxication

ECG INTERPRETATION

The ECG shows ST segment elevation in the right precordial leads. There is coved ST segment elevation ≥ 2 mm at its J point followed by a negative T wave with no isoelectric separation, specifically in V_2. These ECG features are characteristic of the Brugada syndrome[1]. The differential diagnoses for ST segment elevation in the right precordial leads are listed in Table I.

DIAGNOSIS

Brugada syndrome.

CLINICAL COURSE

Serial cardiac enzymes were normal and a coronary angiogram revealed normal coronary arteries. A transthoracic echocardiogram showed normal left ventricular systolic function and size. The right ventricular function was within normal limits.

This patient's ECG was diagnostic of the Brugada syndrome. He underwent an IV flecanide challenge which proved to be confirmatory with further elevation of ST segment at its J point and spontaneous ventricular ectopy (Fig. 2). Electrophysiological studies (EPS) was done and induced ventricular fibrillation (VF) with 3 extra stimuli. His genetic markers were positive for known mutations in SCN5A gene. Subsequently, an implantable cardioverter-defibrillator (ICD) was implanted for prevention of sudden cardiac death.

DISCUSSION

The Brugada syndrome was first described by the Brugada brothers as a distinct clinical entity associated with a high risk of sudden cardiac death (SCD) in 1992[2]. This familial syndrome displays an autosomal dominant mode of inheritance with incomplete penetrance. It has been reported to be most prevalent in Southeast Asia, especially in Thailand where it is a cause of the sudden unexplained death syndrome[3]. Brugada syndrome has a male predominance (8:1 ratio) and arrhythmic events tend to manifest before 40 years of age. This syndrome has been

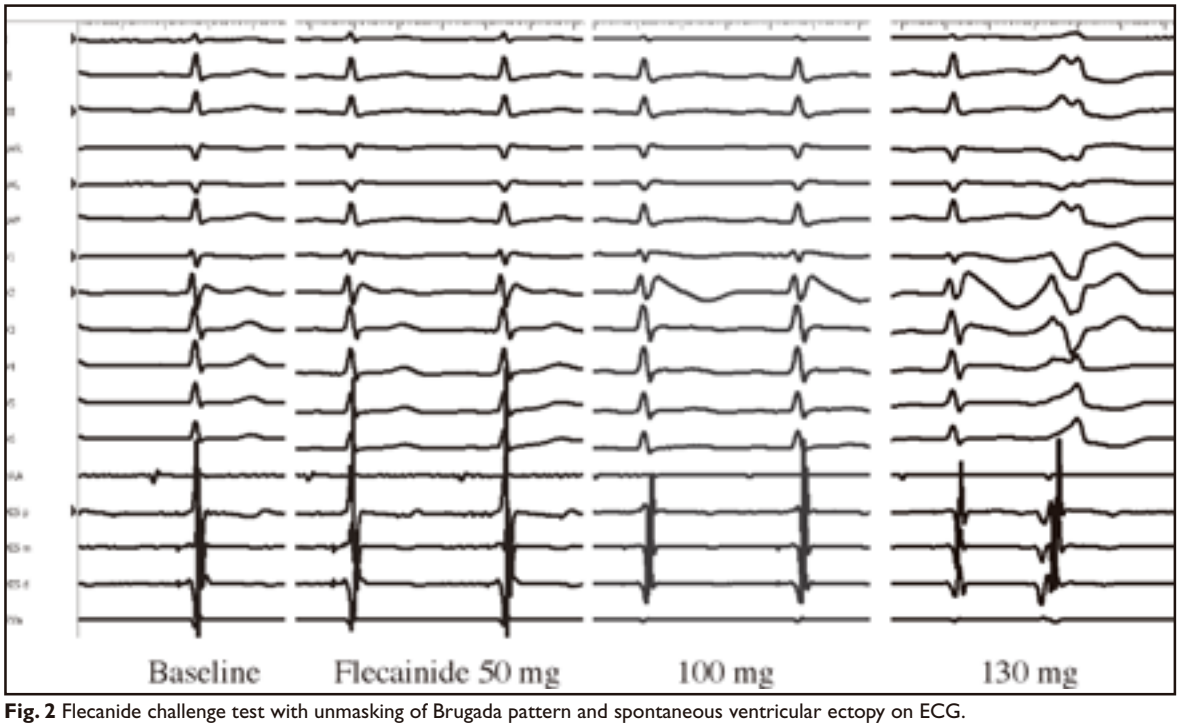

Fig. 2 Flecanide challenge test with unmasking of Brugada pattern and spontaneous ventricular ectopy on ECG.

linked to mutations in SCN5A, the gene coding for the sodium channel[4].

Clinical presentation can vary from syncope to SCD. In some cases, SCD may be the first manifestation of the disease. Self- terminating rapid polymorphic ventricular tachycardias (VT) are responsible for repeated episodes of syncope. Clinical reports appear to suggest that SCD occurs commonly during sleep and in the early morning hours[5]. Febrile states and certain drugs such as sodium channel blockers, vagotonic agents, alpha-adrenergic agonists, beta-adrenergic blockers, tricyclic antidepressants, first-generation anti-histamines (dimenhydrinate) and cocaine toxicity can potentially unmask Brugada syndrome or accentuate ST-segment elevation in these patients[6].

In addition to detailed history- taking, thorough physical examination, ECG and a 24- hour ambulatory Holter monitoring, a transthoracic echocardiogram should be done to rule out structural heart disease. In borderline cases, the diagnosis of Brugada syndrome can be established with the use of sodium channel blockers that modify the ECG pattern. Intravenous administrations of flecanide or procainamide in a controlled setting have been used to unmask the ECG pattern if it is initially absent. Electrophysiological studies (EPS) may be performed to assess for inducibility of VT or ventricular fibrillation (VF) as part of risk stratification for SCD.

Patients with Brugada syndrome who present with aborted sudden deaths are at highest risk for SCD. Those in whom ST-segment elevation appeared only after provocation with sodium channel blockers appeared to be at minimal or no risk for arrhythmic events. In a series of 190 asymptomatic patients with Brugada-type ECG, those at highest risk were males with inducible VT and a spontaneously elevated ST-segment[7].

Implantation of an implantable cardioverter-defibrillator (ICD) is the only established effective treatment for the disease for symptomatic patients[8]. The management of asymptomatic patients remains controversial. If there is a possible risk of SCD, they should undergo an EPS and if VT or VF is induced, ICD implantation should be recommended.

ABSTRACT

A 46-year-old man complained of recurrent episodes of giddiness which was not associated with chest pain or breathlessness. There was no family history of sudden death. Clinical examination was unremarkable. 12-lead electrocardiogram (ECG) showed ST segment elevation in the right precordial leads, with coved ST segment elevation at its J point followed by a negative T wave with no isoelectric separation, specifically in V_2. These ECG features are characteristic of the Brugada syndrome. He underwent a flecanide challenge which produced further elevation of ST segment at its J point and spontaneous ventricular ectopy. Electrophysiological studies induced ventricular fibrillation with 3 extra stimuli. An implantable cardioverter-defibrillator was implanted for prevention of sudden cardiac death. The Brugada syndrome is discussed.

Keywords: Brugada syndrome, cardiac disease, electrophysiological studies, flecanide challenge, ST-segment elevation

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SINGAPORE MEDICAL COUNCIL CATEGORY 3B CME PROGRAMME

Multiple Choice Questions (Code SMJ 200411A)

	True	False
Question 1. ST segment elevation in the right precordial leads can be found in the following conditions except:		
(a) Right ventricular infarction.	❑	❑
(b) Pulmonary embolism.	❑	❑
(c) Hypothyroidism.	❑	❑
(d) Early repolarisation syndrome.	❑	❑
Question 2. The following is true of Brugada syndrome:		
(a) It has a female predominance.	❑	❑
(b) It is associated with sudden cardiac death.	❑	❑
(c) It has an autosomal recessive mode of inheritance.	❑	❑
(d) It is found only in Asia.	❑	❑
Question 3. The following drugs can unmask or accentuate ST- segment elevation in patients with the Brugada syndrome:		
(a) Flecainide.	❑	❑
(b) Procainamide.	❑	❑
(c) Atenolol.	❑	❑
(d) Aspirin	❑	❑
Question 4. Investigations for diagnosis of symptomatic Brugada syndrome should include the following:		
(a) Chest radiographs.	❑	❑
(b) ECG.	❑	❑
(c) Transthoracic echocardiogram.	❑	❑
(d) Electrophysiological study (EPS).	❑	❑
Question 5. Implantation of an ICD in Brugada Syndrome is indicated for:		
(a) Asymptomatic patients with a negative EPS.	❑	❑
(b) Aborted sudden deaths.	❑	❑
(c) Patients with family history of SCD.	❑	❑
(d) Patients with recurrent syncope and positive EPS.	❑	❑

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Submission instructions:

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1. Answers will be published in the SMJ January 2005 issue.
2. The MCR numbers of successful candidates will be posted online at http://www.sma.org.sg/cme/smj by 20 January 2005.
3. Passing mark is 60%. No mark will be deducted for incorrect answers.
4. The SMJ editorial office will submit the list of successful candidates to the Singapore Medical Council.