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CASE STUDY

Case study of Brugada syndrome presenting initially as acute myocardial infarction

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1. Introduction: chief complaint, history, physical examination findings and care

Mr J is a 54 year old smoker, who had dialled emergency medical services (EMS) with chest pain and shortness of breath. The paramedic unit identified ST segment elevation on his ECG (Fig. 1) and therefore raised the ST elevation Myocardial Infarction alert at our centre for a primary angioplasty. Mr J was admitted and underwent an emergency angiogram for a presumed myocardial infarction. However, the angiogram showed normal coronary arteries and no evidence of coronary artery disease or myocardial infarction. Mr J's physical examination and subsequent echocardiogram were normal.

Mr J is a poor historian. He lives on the border between the catchment area of two tertiary centres which both provide similar cardiac care, including primary angioplasty for myocardial infarction. There is a local district general hospital Mr J would also attend. The local district general hospital does not have an angioplasty service. Consequentially, at our centre there was no documentation of previous ECGs, other diagnostics or interventions. Subsequent investigation revealed Mr J had a similar episode three years previously; however, he was transferred to the other tertiary centre. Mr J had been diagnosed with Brugada syndrome. He had undergone a ventricular tachycardia stimulation study (VT stim) under the care of an electrophysiologist. The VT stim study was positive; Mr J was identified as being in a high risk category for sudden cardiac death and implantation of an ICD was recommended. At the time Mr J refused an ICD implant.

2. History of Brugada syndrome and pathophysiology

Brugada syndrome was first described in 1992, and has been researched extensively by three famous electrophysiology siblings, the Brugada Brothers. Brugada syndrome is included in the channelopathies – primary electrical disorders not associated with structural cardiac abnormalities. The disease is produced by alterations in the ion channels of the cardiac cell action potential, leading to susceptibility to arrhythmias. Hence the reason for Mr J's normal physical examination and echocardiogram (Benito et al., 2009). There is a debate that Brugada syndrome could have structural abnormalities which have not been unmasked yet as current diagnostic modalities make it difficult to distinguish Brugada abnormalities from normal cardiac structure (Hoogendijk et al., 2010).

The Brugada ECG pattern can become more prominent at night, at rest and after large meals, which incidentally also correlates with the majority of arrhythmias and sudden death episodes. Fever can also induce the ECG abnormalities (Mattu, 2014).

In Brugada syndrome, there is a genetic mutation, with an autosomal dominant pattern of inheritance. The SCN5A (terminology used to identify the genetic mutation for Brugada) both encode the cardiac sodium channel and are known to cause Brugada. The genetic mutation causes an imbalance between inward and outward ion currents in the myocytes leading to susceptibility to developing dangerous ventricular arrhythmias (Benito et al., 2009).

3. Brugada syndrome diagnosis

The diagnosis of Brugada syndrome is based on a specific ECG pattern. There have been 3 different ECG patterns described, all of which have been seen in Brugada patients; at different times, a patient can present with all 3 patterns. However, only one is diagnostic of Brugada – a Type 1 ECG pattern which presents with a coved-type ST segment elevation greater than 2 mm in more than one right precordial lead (V1–V3), followed by negative T waves (Benito et al., 2009). Mr J's ECG (Fig. 1) presents with a coved shape, in particular in V2, and partly in V1; this is followed by negative T-waves in both these leads. V3 interestingly shows a partial saddleback configuration, which although not diagnostic of Brugada, is in keeping with ECG changes associated with Brugada.

In addition to having a documented Type I ECG pattern, there must be additional clinical criteria: documented ventricular

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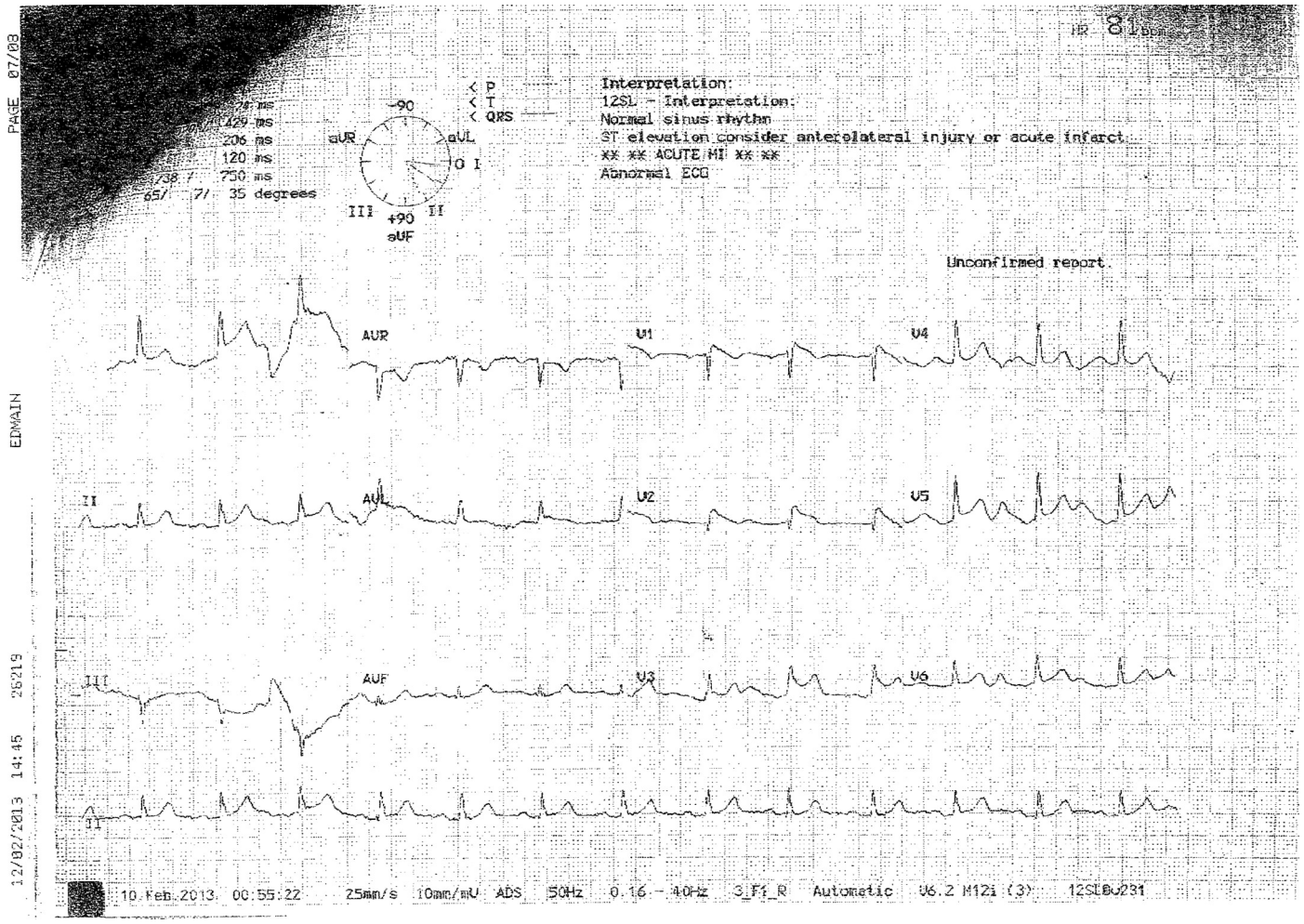

Fig. 1. Mr J's resting ECG in sinus rhythm with obvious evidence of cardiac channelopathy, Brugada Type 1 ECG pattern. Note the ST segment partially in V1 and more definitive in V2. There is a saddle back ST segment in V3 which is associated with Brugada syndrome.

fibrillation, polymorphic ventricular tachycardia or inducible ventricular arrhythmias during electrophysiology study (American College of Cardiology, the American Heart Association, Inc., and the European Society of Cardiology (ACC/AHA/ESC), 2006). In Mr J's case, he had previously had a positive VT stim study at another cardiac centre, therefore meeting all criteria for diagnosis of Brugada syndrome.

There have been no randomised control trials in Brugada syndrome; the level of evidence for management of Brugada is through consensus of opinion of experts (American College of Cardiology, the American Heart Association, Inc., and the European Society of Cardiology (ACC/AHA/ESC), 2006). Postema et al. (2013) reviewed the literature on drug therapy and Brugada. Given Brugada is a fault with the sodium channel (primarily, although not solely), sodium channel blocking medications should be avoided (Table 1). Although not specific in the case for Mr J, patients who present to the Emergency Department (ED) with 'epileptic seizure', should have rare arrhythmic syndromes, such as Brugada in the differential diagnosis (Table 2). Ventricular tachyarrhythmias cause cerebral hypoperfusion creating a picture easily confused with being postictal (Postema et al., 2013, Viskin and Rosso, 2010). Many antiepileptic drugs act by cerebral ion channel blockade, but will also affect the

Table 1
Drug implications in Brugada syndrome advised by the Brugada drugs organisation advisory board (Postema et al., 2013).

Drugs to be avoided: (having the potential to be pro-arrhythmic)
These are strongly advised to be avoided in Brugada syndrome patients or used only after extensive consideration and/or in controlled conditions.
Anti- arrhythmics: flecainide, ajmaline, pilsicainide, procainamide and propafenone.
Psychotropics: amitriptyline, clomipramine, desipramine, lithium, loxapoine, nortriptyline, trifluoperazine.
Anaesthetics: bupivacaine, propofol.
Others: Acetylcholine, alcohol, cocaine, ergonovine.
Drugs preferable to avoid (Drugs which have been associated with Brugada syndrome ECG; however there is yet no substantial evidence these drugs cause malignant arrhythmias)
Anti-arrhythmics: amiodarone, cibenzoline, disopyramide, lidocaine, propranolol, verapamil, vernakalant.
Psychotropics: Carbamazepine, clothiapine, cyamemazine, dosulepin, doxepin, fluoxetine, fluvoxamine, imipramine, lamotrigine, maprotiline, paroxetine, perphenazine, phenytoin, thioridazine
Anti-anginals: diltiazem, nicorandil, nifedipine, nitroglycerine, sorbidnitrate
Others: dimenhydrinate, edrophonium, indapamide
Potential antiarrhythmics that can be used
Isoproterenol, isoprenaline, orciprenaline, quinidine.
Diagnostic drugs in Brugada syndrome
Ajmaline, flecainide, pilsicainide, procainamide.

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