Case Report

Brugada ECG Pattern Unmasked by IV Flecainide in an Individual with Idiopathic Fascicular Ventricular Tachycardia

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Abstract

A 45-year old man presents with stable monomorphic ventricular tachycardia. He had previously been diagnosed with idiopathic fascicular ventricular tachycardia. Intravenous flecainide results in termination of his tachycardia but unmasks a latent type 1 Brugada ECG pattern not seen on his resting ECG. We discuss his subsequent management and the need to consider an alternative diagnosis in individuals with a Brugada type ECG pattern who present with stable monomorphic ventricular tachycardia.

Case Report

A 45-year old man presented with sudden onset palpitations whilst exercising. There was no associated hemodynamic compromise. Electrocardiogram (ECG) showed monomorphic ventricular tachycardia (VT) with a right bundle branch block pattern and a superior axis (Figure 1A).

He gave a history of 3 prior episodes over the preceding 20 years and had seen an electrophysiologist previously. There was no history of syncope and no family history of sudden cardiac death. Resting ECG was within normal limits (Figure 1B) and echocardiography demonstrated a structurally normal heart. At that time, a diagnosis of idiopathic fascicular VT was made and after discussion the patient elected not to undergo an electrophysiology study (EPS).

On this occasion he was treated in the Emergency Department with IV flecainide at the instruction of his treating electrophysiologist. This resulted in termination of tachycardia with restoration of sinus rhythm but unmasked a latent type 1 Brugada ECG pattern (Figure 1C), not seen on his resting ECG (Figure 1B). In view of his history of ventricular arrhythmia and a type I Brugada ECG pattern he was referred by the admitting team for consideration for an implantable defibrillator (ICD).

However, following discussion with the electrophysiology service, a diagnostic EPS with a view to ablation of his idiopathic fascicular VT was recommended. Following administration of intravenous isoproterenol, monomorphic VT (CL 260ms) was easily induced on multiple occasions with
programmed ventricular stimulation. This VT had an identical morphology to that seen on arrival at the Emergency Department. An externally irrigated ablation catheter (Cool Path Duo, St Jude Medical, St Paul, MN, USA) was advanced retrogradely to the left ventricle and a 3 dimensional (3D) left ventricular geometry and activation map was created using Ensite Velocity (St Jude Medical, St Paul, MN, USA) (Figure 2).

Earliest activation of the ventricle was seen at the insertion of the posterior fascicle with centrifugal activation of the rest of the chamber. The catheter was drawn back along the inferior septum to the region of the posterior fascicle until a discrete Purkinje potential could be identified preceding each ventricular electrogram (Figure 1D). Ablation was performed (maximum 40W) in the region of the posterior fascicle (Figure 2). Following ablation no polymorphic or monomorphic VT or ventricular fibrillation was inducible with programmed stimulation and up to 3 extra beats either in the baseline state or with isoproterenol.

Discussion

We present a case of monomorphic VT in a patient with a type 1 Brugada ECG pattern following injection of intravenous flecainide. This raises the possibility of either monomorphic VT as part of the Brugada syndrome or of a latent Brugada ECG pattern in an individual with idiopathic fascicular VT. The common form of idiopathic fascicular VT is characterized by a relatively narrow QRS width with right bundle branch block morphology and a superior axis [1]. It commonly presents in the second to fourth decade, is usually paroxysmal and is associated with a structurally normal heart. It is commoner in males and has a good prognosis. It can be induced by exercise, atrial and ventricular premature beats
and atrial and ventricular pacing, and can be terminated using verapamil. The tachycardia is thought to be due to a reentrant circuit close to the posterior fascicle of the left bundle branch. In this case the administration of intravenous flecainide presumably slowed a critical part of the circuit sufficiently to produce block thereby resulting in termination of the tachycardia and restoration of sinus rhythm. At EPS, high frequency Purkinje potentials are seen preceding the site of earliest ventricular activation during VT. Catheter ablation is the preferred treatment choice for fascicular VT with a success rate of greater than 90%.

Brugada syndrome was first described in 1992 and is characterized by episodes of syncope or unexpected sudden death in patients with structurally normal hearts [2]. It classically presents with polymorphic VT/VF. Whilst a Brugada ECG refers to the manifestation of a type 1 ST-segment elevation, Brugada syndrome is only definitively diagnosed when these changes are observed in conjunction with one or more of the following: documented VF, polymorphic VT, a family history of sudden cardiac death below 45 years of age, coved-type ECG changes in family members, inducibility of polymorphic VT with programmed electrical stimulation, syncope or nocturnal agonal respiration [3].

In this case although he undoubtedly has a type 1 Brugada ECG pattern he has no other features to indicate he has Brugada syndrome. The ease of inducibility of VT, the intra-cardiac EGMs, the activation map and the site of successful ablation all support a diagnosis of idiopathic fascicular VT as the likely cause for his tachycardia.
While monomorphic VT has previously been reported in individuals with Brugada Syndrome [4] it is extremely rare. An ICD in this gentleman would have been inappropriate putting him a risk of potential defibrillation, with its associated morbidity and mortality risks, for non-life threatening arrhythmias.

This case highlights the importance of excluding other, potentially treatable causes of idiopathic VT prior to ICD implantation. This is especially true in those with a Brugada type ECG pattern who are considered at low risk of sudden cardiac death, where appropriate ablative strategies can negate the need for a defibrillator.

References

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