

Risk Stratification in Frequent Ventricular Extrasystoles: The Importance of Cardiac Magnetic Resonance

Abstract

Ventricular tachycardia (VT) has a direct relationship with sudden cardiac death (SCD) and risk stratification is essential for further treatment. In a context of a structural normal heart, idiopathic VT is the principal suspicion. Cardiac magnetic resonance (CMR) is a powerful tool to evaluate the anatomic substrate of ventricular arrhythmias. Myocarditis is a frequent cause of a arrhythmias, and is properly diagnosis by CMR. The presence of late gadolinium enhancement is relate with the diagnosis and pronostic of myocarditis.

Keywords: Ventricular extrasystole; Ventricular tachycardia; Myocarditis

Case Report

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Abbreviations: BPM: Beats Per Minute; CMR: Cardiac Magnetic Resonance; LBBB: Left Bundle Branch Block; LGE: Late Gadolinium Enhancement; LV: Left Ventricle; LVOT: Left Ventricular Outflow Tract; RVOT: Right Ventricular Outflow Tract; VT: Ventricular Tachycardia; SCD: Sudden Cardiac Death

Introduction

Ventricular tachycardia has a direct relationship with SCD, so risk stratification is essential for further treatment, which fluctate from expectant management or pharmacological treatment, to catheter ablation procedures or the implantation of an automatic defibrillator device. The initial step is to know the coexistence of structural heart disease using imaging technics. Myocarditis has been associated with the presence of ventricular arrhythmias with a variable prognosis. An appropriate diagnosis and risk stratification by magnetic resonance imaging can turn the treatment. The objective of the present review is to report a case of ventricular arrhythmia in which the result of CMR changed the diagnosis and subsequent treatment.

Case Presentation

A 21 year-old man was evaluated by a cardiologist in an emergency unit with a three hours history of palpitations, lightheadedness and diaphoresis. Physical examination was unremarkable except for a heart rate of 150 bpm. No laboratory abnormalities were observed. Electrocardiogram (ECG) showed a wide QRS complex tachyarrhythmia (Figure 1) with monomorphic ventricular tachycardia (VT) criteria. After amiodarona administration, a second ECG showed sinus rhythm and monomorphic ventricular trigeminism (LBBB pattern with inferior axis) (Figure 2). Transthoracic echocardiogram was unremarkable. In follow-up, ECG and 24 hours ambulatory Holter showed the same of monomorphic ventricular extrasystoles in 30% of the beats. In this context, the diagnosis of idiopathic ventricular tachycardia was suspected. Three weeks later, cardiac magnetic resonance was requested and basal interventricular

septum myocarditis scar between both outflow tract, and systolic dysfunction (LVEF=48%) was reported (Figure 3). In that context, what is the next step?

Discussion

Ventricular tachycardia is a rhythm associated with sudden cardiac death (SCD) [1], therefore an adequate diagnosis is fundamental in the prognosis. ECG is the initial tool in differential diagnosis of a wide QRS complex tachycardia. The most commonly used algorithm is the so-called Brugada algorithm or Brugada criteria, in which the isolated presence of atrioventricular dissociation has specificity of 100% to support VT diagnosis [2] (Figure 1).

Premature ventricular complexes (PVCs) are ectopic beats originated from the Purkinje system or ventricular myocardium. PVCs can be classified according to the place of origin (right or left ventricle), frequency (isolated, bigeminy, trigeminy and repetitive) shape (monomorphic, polymorphic) and relationship with cardiac cycle (relationship with diastole) [3]. In cases that PVCs or VT has a LBBB morphology with a inferior axis, idiopathic VT (adenosine-sensitive outflow tract) should be suspected⁴, especially in young patients without structural heart disease (Figure 2). In these cases, its important to define the possible location of the ectopic focus, most of them between or inside the right and left ventricular outflow tracts, using ECG data (Table 1 & 2) [5-8]. In our case, posterolateral superior region in RVOT is the most possible source. Evolution, treatment and prognosis of this arrhythmia could be benign [9]. In other hand, this morphology is a minor criteria of arrhythmogenic right ventricular displasia, becoming the main differential diagnosis of idiopathic VT.

Mechanism of idiopathic VT in most cases are adenosine sensitive and are thought to be caused by catecholamine induced cyclic adenosine monophosphate (cAMP) mediated delayed after-depolarizations and triggered activity [10]. There are two phenotypic forms of adenosine sensitive VT, the first one is

repetitive monomorphic VT (most common characterized by frequent premature ventricular complexes), and the second one is paroxysmal exercise induced VT (characterized by sustained episodes of VT precipitated by exercise or emotional stress) [11]. Idiopathic VT usually arises from outflow tracts (80-90% from RVOT), pulmonary artery, aortic cusps, papillary muscles, mitral or tricuspid inflow tracts and epicardial foci [12]. This arrhythmia appears at a relatively early age (30 - 50 years), with equal distribution between sexes in LVOT, whereas RVOT is

more common in females. Common symptoms are palpitations, dizziness and syncope (rare) [13]. Acute management can be achieved by vagal maneuver or adenosine, verapamil or cardioversion; and chronic management include medical therapy (mild to moderate symptoms: beta blockers, verapamil, diltiazem, antiarrhythmic class IA, IC and III agents). Catheter ablation is reserve for symptomatic, drug refractor patients, with a success rate of 90% and only 5% of recurrence [14].

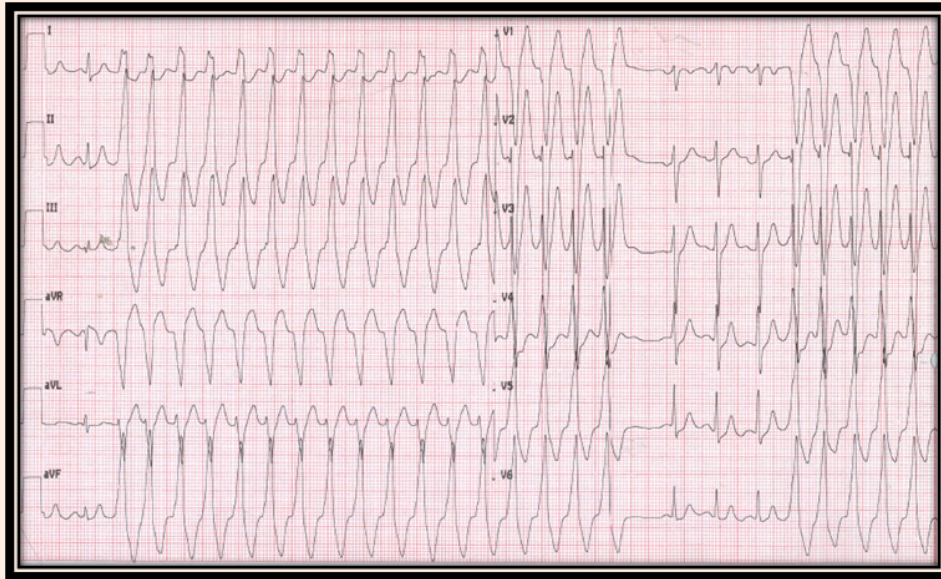


Figure 1: Wide QRS complex tachycardia. AV dissociation with AV relation < 1 , and onset QRS to peak in lead DII > 50 msec was observed, supporting the diagnosis of VT.

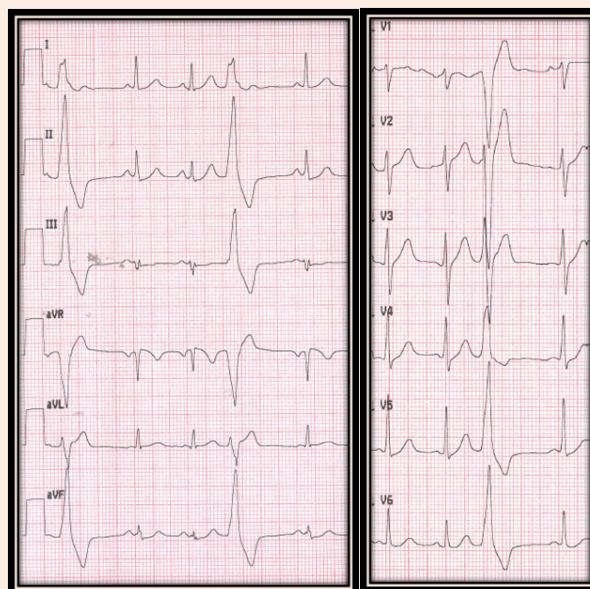


Figure 2: ECG in sinus rhythm and frequent extrasystoles with LBBB morphology, absence or R wave in V1 and QS in aVR derivation \geq aVL. This findings are consistent with a posterolateral portion of the right outflow tract as the genesis of the extrasystole and VT.

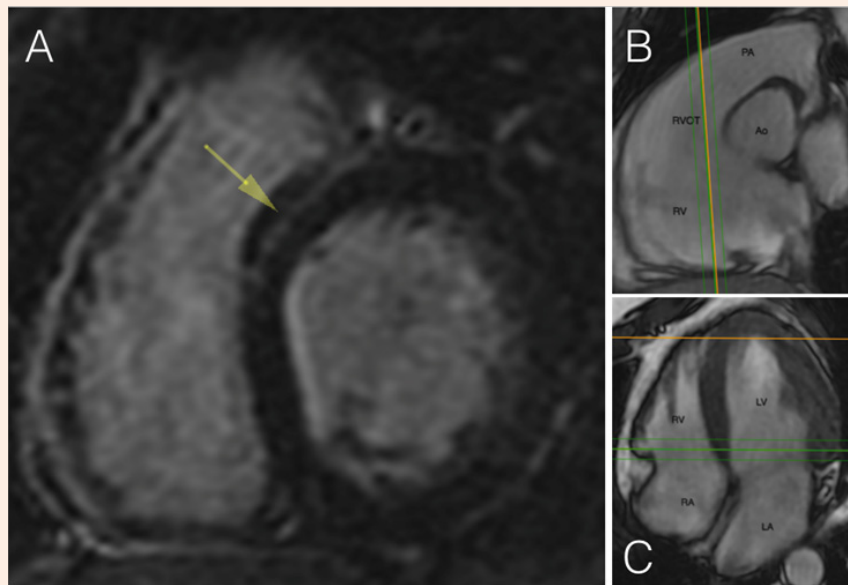


Figure 3: CMR. In A we observe a lineal intramyocardial LGE in the basal portion of interventricular septum, in correlation to the right ventricle outflow tract (B, C). This finding support the possible origin of the extrasystole and VT in relation to the scar.

Table 1: Differentiation between right ventricular outflow tract versus left ventricular outflow region VT origin by 12 lead ECG.

	QRS Morphology	R Relationship in V1 Lead	Precordial QRS Transition in RS	Precordial QRS Transition in VT (da= 100%)	V2 Transition Ratio* (da = 91%)	V3 R/S Ratio (da=83%)
RVOT	LBBB	Absence	V3-V4	earlier than RS	< 0.6	≥ 1
LVOT		Present	V1-V2		≥ 0.6	< 1

LBBB: Left Bundle Branch Block; LVOT: Left Ventricle Outflow Tract; NSR: Normal Sinusal Rhythm; RVOT: Right Ventricle Outflow Tract; VT: Ventricular Tachycardia.

Table 2: Estimation of exactly localization of right ventricular outflow tachycardia origins by 12 lead ECG.

Anterior (Free wall side) versus Posterior (Septal)		
	QRS duration (da = 80%)	Leads II and III R wave pattern (da=86%)
Free wall side	> 140 mseg	RR' or Rr'
Septal	≤ 140 mseg	R
Left (anteromedial attachment) versus Right (posterolateral attachemnt)		
	Leads aVR and aVL QS wave amplitude (da = 80%)	Lead I polarity (da = 83%)
Left side	aVR < aVL	Negative
Right side	aVR ≥ aVL	Positive
Superior versus Inferior relationship to the pulmonic valve		
	Leads V1 and V2 initial r Wave amplitude (da = 66%)	
Superior (proximal below pulmonic valve)	≥ 0.2 mV	
Inferior (distal below pulmonic valve)	< 0.2 mV	

mseg: Milliseconds.

In patients with malignant arrhythmias, CMR has been shown to be a powerful tool in the evaluation of the arrhythmogenic substrate. In a study of 82 patients with resuscitated SCD or VT, CMR diagnosed underlying myocardial disease in 74% of cases, and 50% were reassigned to a new diagnosis [15]. In the case of myocarditis, CMR has shown a high specificity (91%) making it the gold standard now-a-days [16]. Most of these patients present a combination of oedema, hyperemia and late gadolinium enhancement (LGE), supported by the presence of systolic dysfunction and pericardial effusion. However, oedema and hyperemia are closely related to the acute event, and disappear after 3 weeks. Therefore, in the subacute or chronic phase, LGE may be the only histological mark of a recent inflammatory process. The presence of cardiac arrhythmias (PVC or VT) occur frequently in viral myocarditis. In a study involving 222 patients with biopsy proven myocarditis, 19.2% all-cause mortality was observed in the follow-up, mostly SCD-related, and LGE was the main independent predictor [17]. On the other hand, myocarditis was identified as the cause of SCD in 40% of cases in a Air Force recruits study [18]. The mechanisms by which arrhythmias occur in these patients remain controversial, however, it is hypothesized that fibrosis induced by viral inflammation leads to reentrant circuits. Other proposed mechanisms are: myocyte necrosis, proarrhythmic effects of cytokines, altered function at myocardial gap junctions, altered calcium handling, infarction and protease release [18]. In other cases, the inflammatory process could unmask a subclinical cardiomyopathy or channelopathy and predispose the genesis of malignant arrhythmias.

In most cases of malignant arrhythmias secondary to myocarditis, no further treatment is required. In general, when the inflammation turn off, the arrhythmogenic substrate also disappears, and most have a good prognosis. In cases where a scar persists, we can assume a torpid evolution with ventricular remodeling, progression to cardiac failure and a high risk of SCD. However, in the context of primary prevention, there is limited evidence to support the implantation of ICDs in patients with severe sequelae of myocarditis, so it often depends on the patient's clinical context and their own evolution.

Conclusion

In patients with sustained monomorphic ventricular tachycardia and frequent extrasystole it is essential to evaluate a possible anatomical substrate with imaging techniques such as CMR. Adequate etiological diagnosis is related to prognosis and treatment.

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