

Radiofrequency Catheter Ablation; A Life-Saving Tool for Tachycardiomyopathy due to Incessant Idiopathic Ventricular Tachycardia

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Abstract

A 21 year old male patient presented at his primary care provider with symptoms suggestive of a congestive heart failure (CHF). After medical history, physical examination, preliminary laboratory and diagnostic tests were performed, the consensus was indeed that of a Dilated Cardiomyopathy (DCM) with a florid CHF syndrome.

Among all the clinico-diagnostic investigations performed, it was the surface electrocardiogram (ECG) which stood out as the key element in diagnosing his underlying condition. This peculiar ECG tracing featured a "wide-complex" pattern which was identified as a relatively uncommon idiopathic ventricular tachycardia.

It was the incessant nature of this arrhythmia which gave rise to what is known as a tachycardiomyopathy which accounted for his CHF symptoms. Medical treatment to combat symptoms included betablockers, ACE-inhibitors and diuretics, but these only constituted a "window" prior to definitive treatment comprising radiofrequency catheter ablation.

Radiofrequency catheter ablation is a treatment modality currently considered as the most effective "first-line" therapy for symptomatic ventricular tachycardia with no evidence of structural heart disease. Successful ablation of this tachycardia in our patient led to an immediate reversal of his abnormal surface ECG morphology. Most importantly however, was his significant clinical improvement in quality of life 6 months post-ablation, with a purported increase in life expectancy in the years to come.

Keywords: *Incessant Idiopathic Ventricular Tachycardia; Tachycardiomyopathy; Congestive Heart Failure; Radiofrequency Catheter Ablation*

Introduction and Case Report

The possible aetiology of a dilated cardiomyopathy (DCM) is diverse. In fact there are more than 70 established causes that have been cited to date [1]. Like most cardiac conditions their natural histories usually culminate in a congestive heart failure syndrome.

In many instances this diagnosis portends a rather ominous prognosis replete with multiple hospitalizations, elevated health costs and a marked reduction in life expectancy. Finding a potentially reversible cause constitutes a boon, which sometimes involves navigating

through seemingly irrelevant details during patient interrogation, and oftentimes demands a second-look at clinical and other data. This is a common challenge when confronted with a busy, noisy and regularly crowded hospital environment. We are therefore much in agreement with Walker, *et al.* who admits that since arrhythmias are frequently the result of cardiomyopathies, they tend to be overlooked as the potential cause of the problem [2].

Tachycardiomyopathy as a disease entity has been around since 1913, and is well known to produce symptoms of CHF whether induced or mediated by tachycardia [3,4]. In our 21 year old patient at the time of diagnosis, no other readily identifiable cause of his symptoms were forthcoming. Fortunately, the “wide-complex” pattern on his surface ECG tracing was recognized as an idiopathic ventricular tachycardia (Figure 1). Differing ostensibly from a regular rapid monomorphic ventricular tachycardia rhythm as seen in the ECG tracing in figure 5, a casual glance by an uninitiated viewer could have yielded nothing more than a complete right bundle branch block (RBBB) as observed in the ECG tracing in figure 3. This small but key element in the absence of prior structural heart disease (previous myocardial infarction, hypertension, valve disease etc.) established the diagnosis.

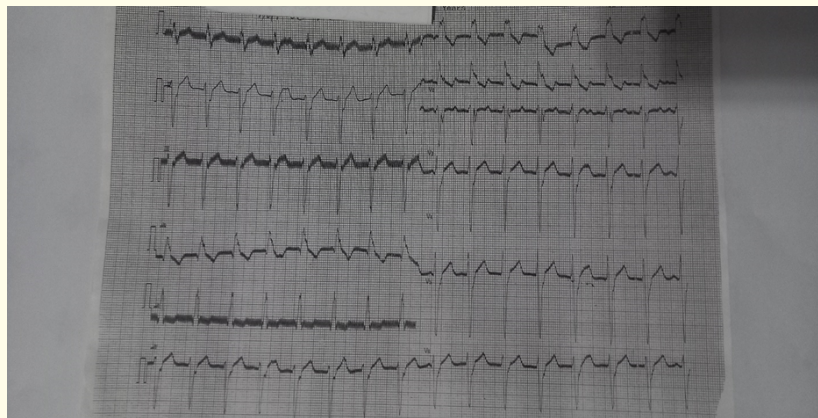


Figure 1: Shows RBBB morphology in 21 year old patient. Notice right superior axis in inferior leads DII, DIII and aVf. Monophasic “R” waves in V1 and R/S ratio < 1 in V6. Transition in V3. Rate a little less than 100 bpm.



Figure 2: Notice the right superior axis in inferior leads DII, DIII and aVf. In V1, notice “qR” waves and more QRS complexes than “p” waves. R/S ratio still < 1 and transition in V3.

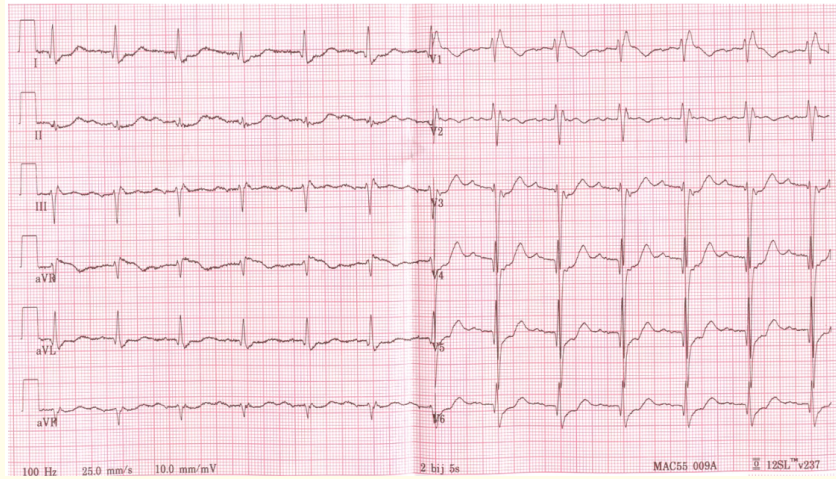


Figure 3: Typical right bundle branch block morphology. Notice rSR in V1 and absence of A-V dissociation where for every “p” deflection there is an “R” wave. PR interval prolonged.

It is our view that as commonplace as it may have been seen, this ECG tracing proved to be the deciding factor in the entire management and prognosis of our patient. Looking at the tracings in figure 1 and 2 which belong to the same patient, they portray heart rates which do not represent any cause for alarm. This is not a feature one expects to see in a “classic” ventricular tachycardia. On the contrary, rapid rates of at least 150 bpm, broad QRS complexes and possibly haemodynamic compromise is the name of the game in most VT’s. Moreover, it is not uncommon to find a patient with a prior QRS aberrancy, whether a LBBB or a RBBB in mild sinus tachycardia. And even though figure 1 and 2 do demonstrate a RBBB morphology in V1 somewhat different than that seen in figure 3, to the untrained eye the diagnosis of a VT may not be immediately obvious. Nevertheless, one of the key elements in the assessment of all 3 tracings constituted the QRS axis. In figure 3 the QRS axis is normal but in ECG tracings 1 and 2 belonging to the same patient, the QRS axis is right superior or north west, falling within the so-called indeterminate sector with values between +/- 180 degrees and -90 degrees. This finding indicates an unusual axis relatively common in ventricular tachycardias [5].

Unlike the ECG tracing in figure 3 where a “p” wave deflection can be identified before each QRS complex, this is not the case in tracing 1 (Figure 1) where there seems to be no readily identifiable “p” wave deflection. One can perhaps speculate that it may even be retrograde and forming the “notch” observed in the “S” waves in multiple leads. In ECG tracing 2 (Figure 2) a “p” wave deflection may be observed before some QRS complexes, but not before all of them. That is to say, there are more QRS complexes than “p” waves. This indicates a dissociation between the atrial and ventricular activity and is a highly specific criterion in diagnosing a ventricular tachycardia [5].

According to the Wellens criteria for a VT (See figure 4), there would be evidence of a ventricular tachycardia in figure 1 which presents a monophasic “R” wave in lead V1 as well as an R/S ratio < 1 in V6. Similarly in figure 2 a “qR” morphology is also seen in V1 which would again point towards a VT [6]. Sometimes “capture” beats, which are normal sino-atrial beats that manage to successfully depolarize the ventricles are observed, or “fusion” beats which are like hybrids between a normal sino-atrial descending current via the His-Purkinje fibres and a ventricular ascending current may be observed. In none of these tracings could these be found, but if present they would have also suggested a ventricular origin of the tachyarrhythmia.


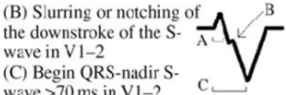

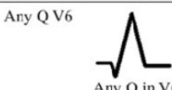
Classical, Wellens, criteria favouring VT	
AV dissociation, capture or fusion beats, negative or positive concordance, tachycardia QRS more narrow than sinus QRS	
RBBB configuration	LBBB configuration
QRS width >140ms, left axis	QRS width >160ms, right axis
QR, R, RSr' complex in V1 	(A) Initial R in V1 >30ms (B) Slurring or notching of the downstroke of the S-wave in V1-2 (C) Begin QRS-nadir S-wave >70ms in V1-2 
RS <1 in V6 	Any Q V6 

Figure 4: Wellens morphological criteria favouring a VT in RBBB and LBBB.

Tracing 4 (Figure 5) represents a rapid sustained monomorphic ventricular tachycardia which is probably what most physicians expect to encounter as the default “wide-complex” tachycardia for a VT. Perhaps arriving at the diagnosis of a ventricular tachyarrhythmia with a similar tracing may not be as challenging since this is the classic morphology we normally anticipate, and certainly not the kind encountered in figure 1 and 2. We believe that here lies the true value of this case report and the importance of analysing well each ECG tracing, particularly in the field of cardiac electrophysiology given the peculiar characteristics offered by many tracings. The latter constitutes an invaluable tool both in sinus rhythm as well as during episodes of tachycardia, thereby emphasizing the need to give an exhaustive first look, and oftentimes a second and third look. Repeat analyses are of particular importance for EP fellows, cardiologists and primary care attending physicians and ought to be “a rule of thumb” before conclusions are drawn within a busy or even a slow laid-back setting.

Naturally with the passage of time and given the incessant nature of this ventricular arrhythmia (VA), a dilated cardiomyopathy developed with characteristic cardiac dilatation, severely reduced ejection fraction and CHF symptoms.

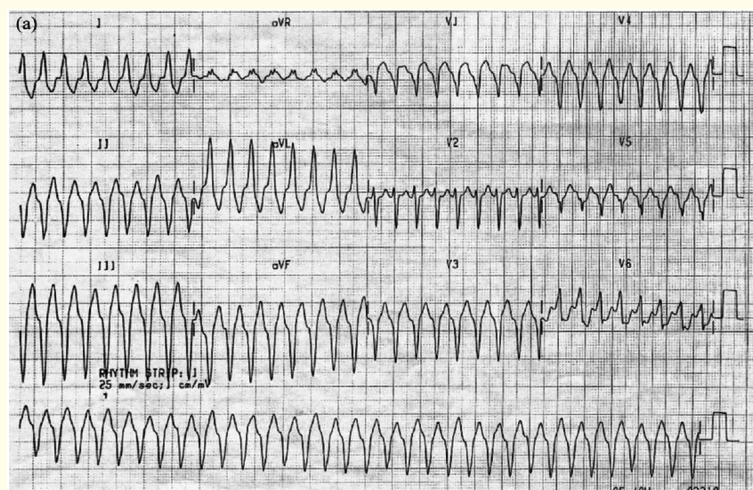


Figure 5: Demonstrates a typical rapid sustained monomorphic ventricular tachycardia. Notice the right superior axis, left bundle branch (LBBB) morphology, wide-complex of nearly 200 ms and rapid rate of around 190 bpm. In V1, “R” duration is > 30 ms and R to nadir of S > 100 ms.

Standard medical treatment for CHF included betablockers, ACE-inhibitors and diuretics among others but these merely served as a “window” to a more definitive and optimal treatment strategy. Radiofrequency catheter ablation for this particular sub-set of patients with idiopathic ventricular tachycardia, constitutes “first-line” therapy which is capable of offering a high potential for cure [7].

Radiofrequency ablation (RFA) was first introduced as early as 1931 by Martin Kirschner as a treatment option for trigeminal neuralgia. However, its use caught on in cardiology after Huang and colleagues first introduced radiofrequency catheter ablation for the management of cardiac arrhythmias in 1985 [8]. This technique employs radio waves to create heat energy of at least 50 degrees C at the catheter-tip, in an effort to destroy abnormal areas of conduction.

From another historical perspective it is interesting to note that, although intra-cardiac catheters were in use approximately 20 years before Huang and his team they were not being used for ablation. In fact by 1967 both Durrer, Coumel., *et al.* and later in 1971 Wellens were already inducing tachyarrhythmias via programmed electrical stimulation, but the mainstay of non-pharmacological therapy was still surgical in nature [9].

An essential consideration for RFA is to obtain ECG patterns both in sinus rhythm where available, as well as during a tachycardia episode. This enables the operator to design a pre-ablation strategy with the objective of limiting as much as possible the “area of interest” for ablation, thereby minimizing the use of time, resources, complications and augmenting the chance of procedural success.

Medical literature reports that “among patients referred for evaluation of ventricular tachycardia, 10% present with ventricular tachycardia in the absence of overt structural heart disease” [10]. The majority of idiopathic ventricular arrhythmias (VA’s) originate from the area surrounding the ventricular outflow tract, resulting in a QRS with inferior axis on the 12-lead ECG. The right ventricular outflow tract (RVOT) is by far the most common site to encounter the origin of an idiopathic VT. This location comprises more than 70% of all such VT’s in the electrophysiology (EP) lab [10,11].

Discussion and Conclusion

In this particular case report, the RBBB pattern on surface ECG suggested a left ventricular outflow tract (LVOT) origin. Moreover, as was previously mentioned it demonstrated a superior axis with predominantly negative vectors in leads DII, DIII and aVF, a variety of VA with a rather low prevalence. Suspected sites of origin for this tracing could have potentially involved the mitral annulus, tricuspid annulus, or the epicardium but in our patient the site identified was the posterior papillary muscle [12].

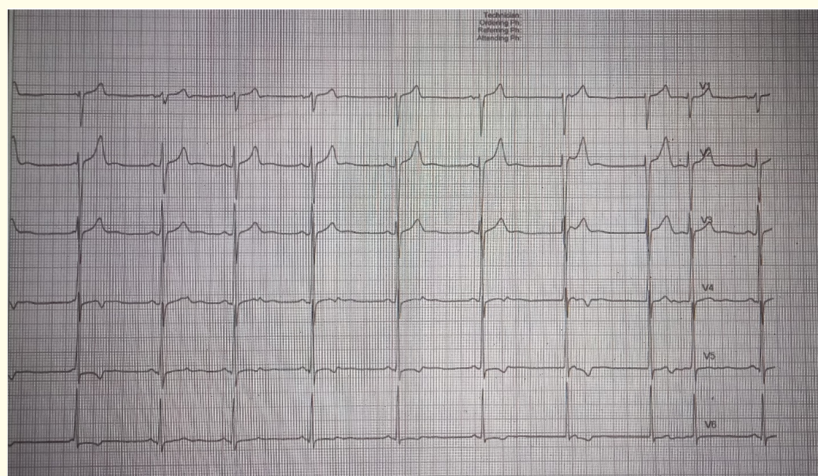


Figure 6: Rhythm strip of our 21 year old patient immediately post-ablation showing a mild sinus bradycardia of 52 bpm. Note the dramatic morphological changes in precordial leads V1-V6.

After successful ablation surface ECG depicted a normal QRS morphology (See figure 6) and follow-up 6 months later demonstrated a virtual absence of CHF symptoms, a significant reduction in cardiac dilatation and a subsequent improvement in myocardial contractility.

Had the ECG tracing been missed as an idiopathic ventricular tachycardia or if it had not been properly differentiated as a “wide-complex” tachycardia [13,14], the alternative natural history may have been at the very worst certain death. At the very best it could have involved later cardiac transplantation, or probably the implantation of a pacemaker (ICD/CRT) or even some LV assist device. All in all it may be somewhat reassuring to know that despite the velocity in which new technologies are developing in the field of EP, the common surface ECG still maintains its position of being a vital and indispensable tool for arrhythmia diagnosis and as an adjunct in intra-cardiac localization.

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