Case Report:
Right Ventricular Outflow Tract tachycardia – Recognition and management

Dr Alastair Gray
Cardiology SPR
Craigavon Cardiac Centre, Northern Ireland

Presentation

A 53 year old male presented to the emergency room with palpitations. He was haemodynamically stable and his presenting electrocardiogram is shown in figure 1.

![Figure 1](image)

The emergency physician diagnosed ventricular tachycardia (VT) and commenced 300mg intravenous amiodarone and referred the patient to the cardiology team. He subsequently reverted to sinus rhythm and had an unremarkable electrocardiogram as shown in figure 2.
On review he reported having experienced intermittent palpitations for several years and although never investigated was recently commenced on bisoprolol by his general practitioner. He was found to have a structurally normal heart on echocardiogram assessment. Due to the ECG morphology and absence of adverse features on investigation, a diagnosis of right ventricular outflow tract tachycardia (RVOT VT) was made. Despite appropriate dose titration by the cardiology team he remained symptomatic and experienced multiple episodes of VT as demonstrated in figure 3. A subsequent trial of verapamil was similarly unsuccessful at alleviating his symptoms and as a result he underwent successful radiofrequency ablation.
Discussion

Ventricular tachycardia occurs most commonly in patients with structural heart disease. Metabolic and electrolyte abnormalities, channelopathies and other inherited cardiac conditions may also predispose an individual to ventricular tachycardia. However 10% of patients are found to have no discernable cause and are therefore termed idiopathic ventricular tachycardia. The distinction between these groups is important as idiopathic VT is generally well tolerated with a benign course and low risk of sudden cardiac death. As such the treatment strategies and prognosis differ from VT associated with structural heart disease.
The presenting surface electrocardiogram of our patient demonstrates a ventricular tachycardia with a left bundle branch (LBBB) morphology and inferior axis. Based on this morphology the most likely site of origin is the right ventricular outflow tract (RVOT). Indeed 80-90% of outflow tract tachycardia’s arise from the right outflow tract. This type of VT typically occurs in the third to fifth decade and is twice as common in females. (1,2) Whilst some patients are asymptomatic, the most frequently occurring symptoms are palpitations, dizziness and pre-syncope. More rarely, patients present with chest pain, tachycardiomyopathy, syncope, unstable VT or sudden cardiac death. The surface electrocardiogram may be unremarkable or may reveal isolated or frequent premature ventricular complexes (PVC) of characteristic morphology. Similarly episodes of non-sustained (NSVT) or sustained VT may be detected on cardiac monitoring. Episodes occur more frequently during the day and runs of NSVT may follow a period of exercise. PVC’s and NSVT may be supressed by exercise testing, however sustained VT may occur during or post exercise stress testing (EST) which is frequently used in the evaluation of RVOT VT.(1) It should be noted however that EST has a low diagnostic yield, initiating OTVT in approximately 25-50% of patients.(3)

Mechanism
The mechanism of this VT is considered to be cathecholaminergic mediated delayed after-depolarisations causing triggered activity. The rise in intracellular calcium precipitated by the catecholaminergic induced surge in cyclic adenosine monophosphate causes delayed after-depolarisation and initiation of tachycardia. This catecholaminergic drive explains the increase in symptoms during times of emotional stress, anxiety, chemical stimulation (e.g. caffeine), hormonal surges and during or post exercise.(2)

Localisation of origin

To be able to correctly interpret the ECG and localize the site of RVOT VT it is essential that the anatomy of the RVOT is appreciated. The borders of the RVOT are defined superiorly by the pulmonary valve, inferiorly by the superior aspect
of the tricuspid valve, laterally by the RV free wall and medially by the interventricular septum. Electrophysiologists generally consider the heart's position within the thorax as it lies *in situ*, describing this as the attitudinal orientation. This orientation concept is important when considering the morphology of the ECG and using it to localise the site of origin as the ECG is obtained from electrodes sampling in this attitudinal position. An in-depth description of the attitudinal relations of the RVOT and surrounding structures can be found in the clinical review by Hutchinson and Garcia (reference 4). Conventionally it is accepted to separate the septal and free wall of the RVOT into anterior, mid and posterior regions with the majority of RVOT VT arising from the anteroseptal region.(1,2)

Considering the anatomical basis of QRS morphology interpretation explains how the accuracy of ECG localisation is limited by many factors including, lead position, anatomical variation, chamber hypertrophy, cardiac axis and structural chest wall abnormalities. Indeed many attempts have been made to produce diagnostic algorithms to enable more accurate localisation of the site of arrhythmia. The accuracy of these is limited by the factors outlined above and has led to novel approaches such as the V2S/V3R index (5) being produced in a bid to facilitate more accurate localisation prior to planned catheter ablation. Despite these difficulties the following ECG characteristics are accepted to be associated with RVOT VT in a structurally normal heart.

- Inferior axis
- Left bundle branch block
- First visible R-wave in V3 or later precordial leads (First visible R wave in V1/V2 which is larger than sinus R wave is suggestive of LVOT origin)
- rS – Rs transition usually V4 or later
- Negative complexes in aVL and aVR
- Lead 1 – positive with posteroseptal origin
- Lead 1 – negative/ flattened with anteroseptal origin
- Reduced negativity in aVL as origin moves more inferiorly and increased positivity in Lead 1
Evaluation

Interpretation of the ECG and further clinical evaluation of the patient should be performed by an experienced cardiology team. The emphasis is on identifying those patients in whom VT may be arising from a pathology that has a less benign course than RVOT VT. A normal surface electrocardiogram and transthoracic echocardiogram (TTE) with typical morphology of PVC’s and VT would support a diagnosis of RVOT VT. Further non-invasive or invasive tests would not typically be required. However further investigation would be warranted if the echocardiogram or electrocardiogram are abnormal, the morphology of PVC’s/ VT is unusual or variable, or the patient has adverse features such as syncope. This may raise the suspicion of less benign pathology such as arrhythmogenic right ventricular cardiomyopathy (ARVC). Whilst arrhythmias from ARVC may share a similar QRS morphology of inferior axis/LBBB pattern the surface ECG/ signal averaged ECG are usually abnormal. In particular the presence of an epsilon wave, inverted T waves in right precordial leads, PVC with QRS duration >120ms in Lead 1, QRS notching in multiple leads and a transition point after V5 are suggestive of ARVC. Additionally abnormalities in the right ventricle structure and function may be identified on echocardiogram and further assessment of these patients with cardiac MRI would be warranted.

Prognosis

Whilst the course of RVOT VT is generally benign it is recommended that these patients remain under cardiology follow up with periodic TTE to exclude latent disease progression. It has been noted that patients with reduced LV function experience a higher percentage of PVC burden however there is no clear cut-off of PVC frequency that is clearly associated with development of cardiomyopathy. Some studies have shown that as little as 10% PVC burden can result in cardiomyopathic changes yet other patients experience significantly higher PVC burdens and do not develop LV dysfunction. It is possible that assessment of PVC frequency using Holter monitoring does not provide an accurate assessment
of a patient's true PVC burden and this may explain the differences found in these studies and therefore risk stratification for LV dysfunction based on PVC frequency cannot be recommended. (6, 7)

**Management**

As most RVOT VT follows a relatively benign course the decision to treat rests on the severity and frequency of symptoms experienced by the patient. Options include pharmacological medical management and electrophysiological intervention with ablation. Acute termination of RVOT VT can be achieved with vagal manoeuvres, adenosine, IV verapamil or cardioversion if there is evidence of haemodynamic instability. (1, 2)

Due to the catecholaminergic drive of RVOT VT, first line chronic therapy is generally with an oral β-blocker. In those patients whom β-blockade is either contraindicated, poorly tolerated or ineffective the non-dihydropyridine calcium channel antagonists (verapamil and diltiazem) would be a suitable alternative. Therapeutic success rates for these oral agents are not clearly documented however some literature quotes up to 50% efficacy. (1) An alternative strategy would be the introduction of a class IA, IC or III anti-arrhythmic agent however due to the potential pro-arrhythmic effect of these agents they are generally reserved for those patients who remain symptomatic and are not willing to pursue catheter ablation.

Catheter directed radiofrequency ablation is associated with curative success rates of up to 98%. (1, 2, 5) It is a safe and feasible option for the management of RVOT VT in patients who remain symptomatic despite medical management. Additionally it is a useful first line strategy for those patients who have been found to have adverse features including syncope, very fast ventricular rates or PVC’s with short coupling intervals. (1, 2) Localisation of the suspected site of origin from the surface ECG is useful to aid planning of ablation strategies. Consideration should be given to the use of anaesthetic agents as these can suppress PVC’s and VT leading to difficulty in accurate mapping. Infusions of beta-agonists may be useful for mapping in patients who experience infrequent
PVC's/VT, conversely in those patients in whom arrhythmia is driven by bradycardia a beta-blocker infusion may be a useful strategy. Mapping strategies include activation mapping and pace-mapping and these may be supported by the use of 3D-mapping systems. Activation mapping aims to identify the earliest pre-systolic activation on the bipolar electrogram which, for RVOT VT, typically precedes the surface electrocardiogram by >20ms. This mapping strategy is useful in patients who experience frequent PVC's. Pace-mapping is particularly useful in patients with a low PVC burden. Pacing at or close to the site of origin at a frequency similar to that of the RVOT VT rate can produce a surface electrocardiogram with similar morphology to the RVOT PVC/VT. The complex geometry of the right ventricle and outflow tract combined with anatomical variation can present a challenge to radiofrequency ablation, however with an experienced operator and appropriate mapping systems high success rates are achievable.

**Conclusion**

This case demonstrates the typical ECG morphology of RVOT VT. The clinician must be able to accurately recognise this morphology to initiate appropriate management and to differentiate from VT associated with less benign conditions. This relatively benign form of VT typically responds well to pharmacological management with ablation reserved for those patients who remain refractory to treatment.
References

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