Ventricular Tachycardia in Normal Heart: Approach and Management

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Summary

Ventricular tachycardia (VT) in normal heart (also known as idiopathic VT) arising from ventricular outflow tract of right ventricle (RV) or left ventricle (LV) are referred as ventricular outflow tract ventricular tachycardia (OT VT). Another group of idiopathic VT, arising from LV Purkinje network is called as fascicular tachycardia (Belhassen's tachycardia). This article presents the symptoms, EKG characteristics and the management approach for these VTs. Among all the treatment options, RFA remains a treatment of choice due to very high degree of success with minimal complication rates. (Indian J Cardiol 2012,15:20-26)

Keywords: Fascicular tachycardia, Radio frequency ablation

Ventricular tachycardia (VT) in normal heart is also called as idiopathic ventricular tachycardia, which means VT in absence of structural heart disease like hypertrophy, recent MI, cardiomyopathy or valvular heart disease, where cause is typically unknown. We know how the VT presents, but not very much is known about the 'why' part of it. It essentially rules out syndromes of sudden cardiac death including genetic channelopathies. It constitutes about 10% of VT cases presenting to the Emergency Room (ER) and usually is a benign syndrome with lot of symptoms. These VTs arise either from ventricular outflow tract (OT) of right ventricle (RV) or from left ventricle (LV). RVOT-VTs typically arise from pulmonary artery area to bundle of His region and LVOT–VTs arise from aortic cusps in LVOT–left and right, aorto-mitral continuity, mitral annulus or epicardial area of LV. (Table 1)

Outflow tract ventricular tachycardia (OT VT)

Most patients belong to age group 20–50 years with a disease gender bias in terms of origin of ventricles. In females 70% of VT is from RVOT and in males 70% of VT is from LVOT. Three types of EKG presentations are well known

- Frequent premature ventricular complexes (PVCs)
- Salvos of VT
- Sustained Monomorphic VT (MMVT)

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Common clinical symptoms include palpitation, lightheadedness, dyspnoea, chest pain or syncope. These symptoms are mostly catecholamine driven and are precipitated by exercise, stress, anxiety or stimulants. In females, hormonal triggers are known and are present during certain parts of menstrual cycle. PVCs and salvos can occur during rest or during recovery from exercise and may show circadian pattern.

### Table 1: VT in Structurally Normal Heart (Idiopathic VT)

<table>
<thead>
<tr>
<th>Outflow Tract VT</th>
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<tr>
<td>• RVOT: Pulmonary Artery to His bundle region</td>
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<tr>
<td>• LVOT: Aortic cusps, Aorto-mitral continuity, Epicardial, Mitral annulus</td>
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<th>LV Fascicular VT</th>
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<tr>
<td>• Posterior fascicular</td>
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<tr>
<td>• Anterior fascicular</td>
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<tr>
<td>• Upper septal</td>
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Lack of structural heart disease is rule. However, subtle MRI abnormalities may be present. Three types of relationship are found reported with respect to tachycardia, a third are related to exacerbation by baseline sinus tachycardia, third with Bradycardia and the other third have no relationship with heart rate at all. Diurnal variation to susceptibility is not known.

### Mechanism of outflow tract VT

Delayed after-depolarization is electrophysiological mechanism of initiation of outflow tract VTs. Sequence of events appear to be by some unknown mechanism. There is initial activation of beta-receptors and G-protein stimulation resulting in increased intracellular increase in cAMP which activate release of intracellular calcium (Ca) from sarcoplasmic reticulum and L-channels, which in turn increase transient sodium (Na) influx via Na/Ca pathway. Final result is development of delayed after-depolarization which reach threshold and trigger arrhythmias. Hence, these arrhythmias are responsive to calcium channel blockers and adenosine.

### Anatomical / Electrophysiological characteristics (Fig. 1-4)

Following are EKG and anatomical salient features:

#### RVOT–VT

Anatomically, area responsible consists of RVOT from the level of pulmonary valve to bundle of His in midline and muscle sleeves from RVOT to pulmonary artery trunk, 70–80% origin from septal wall, 20–30% origin from free wall below pulmonary valve. However, origin may be from above the pulmonary valve and from lower area up to His bundle.

- **EKG morphology:** Typical morphology is (LBBB) with inferior axis (normal axis); typical transition is in V4 lead. (Fig. 1)

#### LVOT-VT

Four anatomical areas are known to be origin viz.,

- Aortic cusps: Right and left, non-coronary cusp is typically atrial septal related and hence, originate-cuspal atrial tachycardia and not VT
- Aorto-mitral continuity (AMC)
- Peri-mitral region
- Epicardial region in relation to cardiac veins

- **EKG morphology:** Signature pattern of LVOT-VT / PVCs are found. Aortic cuspal VTs and PVCs have LBBB with inferior axis morphology with transition in V2 or V3. In case of PVCs V3 ratio is important method of differentiating RVOT PVCs from LVOT PVCs.

VT/PVCs having origin from left coronary cusp showed LBBB with inferior axis, W or M in V1 and R wave transition in V1 or V2. (Fig.2)
PVC/VT having origin from right coronary cusp showed LBBB with inferior axis morphology with R wave transition in V₂ or V₃, qS or qR pattern in V₁. It presupposes normal situs/location of the heart in the chest.

- Aorto-mitral continuity area VT/PVCs: Right bundle branch block (RBBB) morphology with broad monophasic R across precordial leads.
- LV epicardial PVCs/VT: Presence of a very slurred upstroke (pseudo-delta wave >34 milliseconds), long intrinsicoid deflection time (>85 milliseconds), and shortest precordial RS complex >121 milliseconds. Maximum Deflection Index (MDI) which is ratio of initial 50% of QRS to distal one ratio more than 60% is important criteria.

Management of outflow tract PVCs and VT

Non-invasive investigations including chest X-ray, echocardiography or MRI imaging should be conducted to rule out the presence of structural heart disease. Exercise stress test is usually done to see effect of exercise on tachycardia. Coronary angiography is usually not required, but if coronary artery disease (CAD) is strong suspicion in relatively elderly patient, it should be done.

Indication of treatment of outflow tract PVCs or non-sustained VT depends on severity of symptoms and disease burden in terms of frequency and duration of PVCs or VT. Treatment options include drug treatment or radiofrequency ablation (RFA). Sustained VT, however, needs immediate treatment because of frequent likelihood of presence of haemodynamic impairment.

- **Acute treatment**: Unlike other VTs, these VT are sensitive to adenosine and calcium channel blockers. Intravenous use of adenosine or verapamil in weight-based doses is usually successful in terminating acute VT. Sustained VT with haemodynamic impairment requires immediate DC synchronized cardioversion.
- **Long-term management**: Long-term management is done either by drug treatment or RFA. Beta blockers, calcium channel Class Ic or III drugs, such as flecainide has been shown to be useful, but long term use is associated with lot of side effects and hence, RFA is considered to be better option because of its almost curative results. Amiodarone is an useful and effective drug but, because of its propensity of universal side effects on use beyond six weeks, it is not a preferred drug. Medications, including beta blockers, verapamil and diltiazem have 25%–50% rate of efficacy. RFA ablation now has cure rates of 90%, which makes it a preferable option, given the young age of most patients with outflow tract VT. It is the most curative treatment option available.

**Left ventricular fascicular tachycardia (LV VT)** (Fig. 5–12)

This VT in normal heart has signature EKG pattern and as the name suggests, this ventricular tachycardia involves participation of fascicles of left bundle in left ventricle. There is no structural heart disease and age group of patients is again relatively young, typically 15 to 40 years (unusual after 55 years). Males are more commonly affected (60%–80%). Fascicular VT is the most common form of idiopathic LV VT, and it accounts for 10%-15% of idiopathic VTs.

Three varieties with unique electrocardiograph or electrophysiology characteristics are described, viz.,
- Posterior fascicular
- Anterior fascicular
- Upper septal varieties.

All these three varieties have classical signature EKG patterns.

**Symptoms and clinical presentation**

Most patients present with mild-to-moderate symptoms of palpitations and lightheadedness. The clinical course is benign and the prognosis is excellent. Sudden cardiac death is rare. Spontaneous remission of the VT may occur with time.
Anatomical / electrophysiological characteristics

Three types of fascicular VTs have diagnostic EKG features
- Posterior fascicular VT includes RBBB with left axis deviation.
- Anterior fascicular VT: RBBB plus right axis deviation and
- Upper Septal Variety VT: Narrow RBBB and normal axis.

Patients have structurally normal heart, and these VTs show typical verapamil sensitivity. Evaluation to exclude structural heart disease is necessary and typically includes echocardiography examination, stress test, and/or cardiac catheterization, depending on patient’s age and risk factors.

Mechanism of Fascicular VT

Fascicular VT is caused by a reentrant circuit confined to the posterior Purkinje system, which shows evidence of excitable gap and a slow conduction area. The VT substrate can be a small macro-reentrant circuit consisting of the left posterior fascicle (LPF) serving as one limb and abnormal Purkinje tissue with slow, decremental conduction serving as the other limb. The entrance site to the slow conduction zone is thought to be located closer to the base of the LV septum. The exit site, site of earliest ventricular activation, is located in the infero-posterior aspect of the LV septum in the region of the LPF closer to the apex. The retrograde limb consists of Purkinje tissue from or contiguous to the LPF, which gives rise to the Purkinje potentials (PP). The anterograde limb of the circuit appears to be composed of abnormal Purkinje tissue, which exhibits slow, decremental conduction, gives rise to the late diastolic potentials (LDP) along the mid-septum. The zone of slow conduction appears to depend on the slow inward calcium current, because the degree of slowing of tachycardia CL in response to verapamil.

Management of VT

Acute management

Intravenous verapamil is typically successful in terminating the VT. Termination with adenosine is rare, except for patients in whom isoproterenol is used for induction of the tachycardia in the EP laboratory.

Long term management

Long-term therapy with verapamil is useful in mild cases; however, it has little effect in patients with severe symptoms.

Radio frequency ablation (RFA) is highly effective (85%–90%) and is recommended for patients with severe symptoms.

EP study and radiofrequency ablation in EP lab

Patient should be brought to EP lab in fasting state. Sedation should be avoided and procedure is performed under local anesthesia to the groin region for introduction of vascular sheaths for placement of EP electrode catheters.

Both varieties of VTs i.e., OT VT and fascicular VTs can usually be initiated in EP lab with atrial extra stimulation (AES), VES, atrial pacing, or ventricular pacing. Often, isoproterenol, alone or during concurrent programmed stimulation, facilitates induction. An inverse relationship is observed between the coupling interval of the initiating VES or ventricular pacing CL and the first VT beat.

EP lab procedure for OT VT (Fig 13-15)

In the EP lab mapping of PVCs/VT is done along with attempt to find out mechanism of arrhythmia, automatic focus versus reentrant. Isoproterenol is used for induction of arrhythmia, if spontaneous arrhythmia is not present. Programmed electrical stimulation from
atrium and ventricle is done using technique of burst pacing and introduction of extra stimuli in singles, doubles or triples at various derive cycle length usually succeeds in induction of arrhythmia. Three types of mapping procedures are usually done viz., activation map, pace map or 3D electro anatomical map.

Recently, non-contact mapping using array multielectrode catheter on balloon is great tool. These techniques are complimentary to each other. Fluoroscopy is main tool of imaging.

Outflow PVC/VT once mapped in the outflow tract can be ablated using RF energy, power 30–40 Watts to temperature about 50 degrees for 5–15 seconds. Only few lesions are usually required to ablate the focus. RVOT, being the thinnest part of RV extra care is required to prevent complication of perforation of cardiac wall and pericardial tamponade. Loss of cardiac silhouette pulsation in LAO 35 degree is the earliest sign on fluoroscopy of tamponade and can be confirmed on echocardiography. Immediate needle aspiration is the only treatment. Fascicular VT in EP lab is usually induced by burst atrial pacing. Re-entry as the mechanism can be confirmed by activation mapping and resetting mechanism on programmed electrical stimulation. 3D electroanatomical mapping is useful and almost mandatory tool for making diagnosis and for ablation using RF energy. Examples of intracardiac tracings, fluoroscopy and 3D electroanatomical map that we use in our lab are shown.

**EP lab procedure for Fascicular VT**

Fascicular VT is easily induced by burst atrial pacing. The site of earliest ventricular activation during VT is (i) in the region of the LPF in inferior posterior LV septum, explaining the RBBB–superior axis configuration of the QRS and (ii) in the region of the LAF anterosuperior LV septum (in 5% to 10% of LV VTs), explaining the RBBB–right axis configuration of the QRS. In most cases, ventricular electrogram are discrete during both normal sinus rhythm (NSR) and VT. The HB is not a component of the re-entrant circuit,
because a retrograde His potential is often recorded 20–40 milliseconds after the earliest ventricular activation.

The Purkinje potential (PP) is a discrete, high-frequency potential that precedes the site of earliest ventricular activation by 15–42 milliseconds and is recorded in the posterior third of the LV septum during VT and NSR. Because, this potential also precedes ventricular activation during NSR, it is believed to originate from activation of a segment of the LPF, and to represent the exit site of the re-entrant circuit.

The late diastolic potential (LDP) is a discrete potential that precedes the PP during VT, and is recorded at the basal, middle, or apical septum. The LDP is thought to originate from activation at the entrance to the abnormal Purkinje tissue that is thought to serve as the anterograde limb of the re-entrant circuit.

During NSR, conduction propagates anterograde (proximal to distal or basal to apical) and rapidly down the LPF, generating an anterograde PP and followed by ventricular activation. In parallel, the impulse slowly conducts anterograde over the abnormal Purkinje tissue, and such slow conduction and/or block in the proximal segment allows the wave front traveling down the LPF to conduct retrograde up the slow pathway, resulting in fusion of delayed (late) ascending and descending potentials that follow or are buried in local ventricular depolarization, which likely represent the LDPs recorded during VT.

During fascicular VT, activation propagates retrograde (distal to proximal) over the LPF, generating a retrograde PP, and subsequently propagates anterograde over the abnormal slow Purkinje tissue, producing an anterograde LDP. Thus, both limbs are activated in series in contrast to NSR and ventricular pacing. For a VES to initiate VT, retrograde block has to occur in the abnormal Purkinje tissue with retrograde conduction of the wave front up the LPF generating a retrograde PP with some delay, and then down the abnormal Purkinje tissue generating an anterograde LDP to initiate reentry. Thus, during VT, the LDP precedes PP, which in turn precedes ventricular activation.

Radiofrequency ablation

Ablation is targeted to a site over the middle or infero-apical portion of the LV septum where the earliest PP and LDP are recorded. Verification of these sites can be achieved with entrainment mapping demonstrating concealed fusion and progressive prolongation of the LDP-PP interval with increasing pacing rate. In addition, pressure applied to the catheter tip at the LDP region occasionally results in VT termination with conduction block between LDP and PP. Pace mapping can also be used as an adjunct to verify this site.

Summary and conclusion

VT in structurally normal heart also called as idiopathic VT is usually a benign syndrome with lot of symptoms especially palpitation. Outflow VT from RV or LV and LV fascicular VT are unique in the sense EKG signature patterns are now known and it is possible to plan EP study RF ablation protocol with high degree of specificity, which saves lot of time and radiation in EP lab. Structural heart disease should be ruled by preferably non-invasive methods. Clinical presentation is either PVC, NSVT or VT. Drug treatment especially with beta-blockers is not preferred by these young patients due to unacceptable side effects and RFA remains a treatment of choice due to very high degree of success with minimal complication rate. It is pertinent to mention that Intracardiac Defibrillator (ICD) implantation is not required following RFA.

References