Ventricular tachycardia

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ABSTRACT

Ventricular tachycardia is a malignant cardiac rhythm which frequently causes hemodynamic collapse if not treated early and aggressively. There are several ways to classify ventricular tachycardia, and it is important that physicians working in critical care units identify the cause and start appropriate treatment for effective care of these critically ill patients.

Keywords: ventricular tachycardia, ventricular fibrillation, treatment

INTRODUCTION

Ventricular tachycardia (VT) and ventricular fibrillation are malignant cardiac rhythms emanating from the ventricles frequently observed in the hospital and intensive care units. Ventricular tachycardia usually shows some regularity on the electrocardiogram but ventricular fibrillation represents rapid and chaotic electrical activity which emanates from the ventricles and appears as an entirely irregular electrical activity on the electrocardiogram. Cardiac arrest occurs within seconds, resulting in damage to the brain and heart which is not reversible unless the patient is treated with electrical shocks or anti-arrhythmic medications.[1]

CLASSIFICATION

Ventricular tachycardia can be classified using symptoms, mechanism, duration, and morphology. The major symptoms of VT are palpitations, syncope, and in extreme cases sudden cardiac death. Some patients also complain of chest pain and shortness of breath. In general, VT is a hemodynamically unstable rhythm, although sometimes it can be tolerated for a few minutes to hours, especially in presence of a left ventricular assist device.

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DOI: 10.12746/swrccc2017.0517.233

The mechanisms causing VT include: 1) increased automaticity, 2) triggered activity, and 3) reentry. Increased automaticity is a state in which single or multiple foci in the ventricles start to fire rapidly, mostly in response to a catecholaminergic state, ischemia, or electrolyte disturbance (e.g., hypokalemia). Triggered activity is dependent on after-depolarizations, which involves phases 3 and 4 of the action potential and is often due to calcium overload in the cells. Reentry, which is the most common mechanism akin to "chasing its tail," is dependent on the presence of two pathways and an excitatory gap in the myocardium so that the wave of depolarization circles continuously. The duration of the episode classifies tachycardia into: 1) nonsustained (fewer than 30 seconds), and 2) sustained (more than 30 seconds).

The three different morphologies of VT are: 1) monomorphic, 2) pleomorphic, and 3) polymorphic. Monomorphic VT has a single morphology throughout, with the QRS complex showing either a right bundle branch block pattern or left bundle branch block pattern. Typically the complex is broad (QRS more than 0.12 seconds), and the rhythm is regular. Pleomorphic VT has more than one morphologically distinct QRS complex occurring during the same episode of VT, but the QRS morphology is not continuously changing. Polymorphic VT has a continuously

changing QRS morphology and rhythm from beat to beat. In the presence of a baseline prolonged QT (QTc more than 440 ms), it is referred to as "torsades de pointes" where the QRS complexes appear to twist around the baseline. Ventricular fibrillation is classified as either primary, if it occurs in a structurally normal heart, or secondary, if it occurs in an ischemic or non-ischemic cardiomyopathy.

Causes

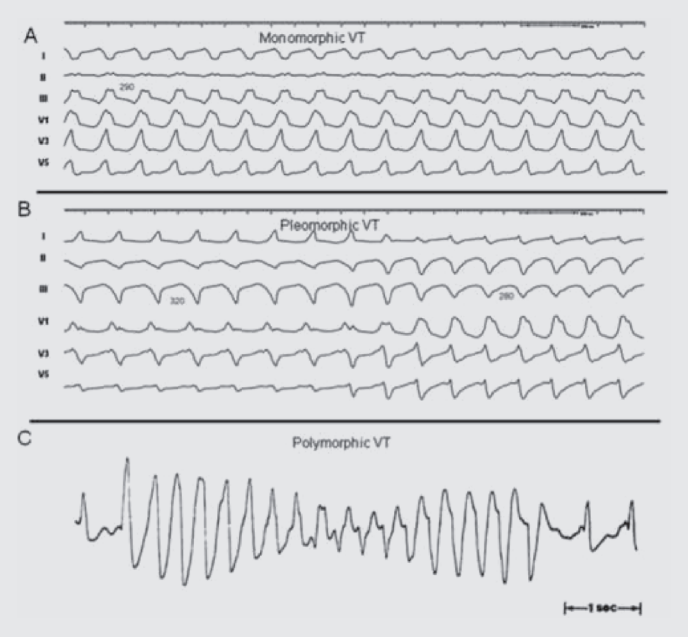
The most common causes of VT are

1. Coronary artery disease
2. Electrolyte disturbance (e.g., hypokalemia/ hypomagnesemia/hypercalcemia)
3. Heart failure exacerbation
4. Dilated cardiomyopathy
5. Drug toxicity (e.g., digoxin)
6. Long QT syndrome (congenital and/or drug-induced)
7. Hypertrophic cardiomyopathy
8. Arrhythmogenic right ventricular cardiomyopathy
9. Brugada syndrome
10. Catecholamine induced polymorphic VT
11. Idiopathic VT

Diagnosis

Patients with VT are usually pulseless. The diagnosis typically requires monitoring of the cardiac rhythm on the telemetry or 12-lead ECG. Any wide complex tachycardia on electrocardiogram should be considered as VT unless diagnosed otherwise. Typical confounders include existence of pre-excitation, aberrancy, or an electrically paced rhythm. Clues to a diagnosis of VT include:

1. Concordance of QRS complexes in the precordial leads (either all positive or all negative).
2. Atrioventricular dissociation with P waves appearing independently of the QRS.
3. Fusion or capture complexes.
4. QRS axis shift from baseline ECG axis.



Treatment

Prompt and judicious treatment is required to prevent hemodynamic collapse and death. The options include:

1. Cardioversion (usually electrical and occasionally chemical)
2. Cardiopulmonary resuscitation
3. Antiarrhythmic drug therapy
4. Pacing/intracardiac defibrillator
5. Ablation

Management of patients with hemodynamically unstable rhythm requires standard advance cardiac life support (ACLS) resuscitation algorithms with immediate high energy electrical shock and cardiopulmonary resuscitation (CPR). Patients should initially

be treated with a synchronized 200 joule shock from a biphasic defibrillator or a 360 joule shock from a monophasic defibrillator. Patients who are hemodynamically stable should be considered for intravenous sedation followed by chemical cardioversion with antiarrhythmic medications, such as lidocaine or amiodarone, preferably amiodarone. In case of failure of cardioversion with lidocaine or amiodarone, procainamide may be considered but only after emergent cardiology consultation. Treatment of the underlying disorder, especially electrolyte disturbance, myocardial ischemia, and heart failure exacerbation, should be started immediately. Amiodarone and beta-blockers remain the cornerstone of pharmacological therapy.[2]

For patients with sustained or non-sustained VT, an echocardiogram should be performed to evaluate left ventricular ejection fraction. For patients with ejection fractions less than 30%, an intracardiac defibrillator is advised. For patients with ejection fraction between 30-40%, an electrophysiological study for induction of VT should be considered and, if positive, an intracardiac defibrillator should be implanted (MUSTT trial).[3] These patients should also be placed on beta-blockers if there is no contraindication.[4]

Patients with idiopathic VT , also known as outflow tract VT or adenosine sensitive VT in which the ECG frequently reveals premature ventricular contractions of outflow tract morphology (QRS axis positive in inferior leads), typically require an ablation (not an ICD), which offers cures in up to 90% of cases and is the first line of therapy.[5]

Incessant VT, also referred to as electrical storm, is defined as three or more VT/VF episodes requiring DC cardioversion over a 24 hour period. Usual triggers are worsening heart failure, electrolyte disturbance, or acute myocardial ischemia. Drug toxicity, especially with digoxin, should always be considered. In patients with pre-existing ICDs, the ICD device therapy should be turned off, and the patient should be sedated and intubated and placed on accelerated doses of antiarrhythmic medications. Emergent VT ablation may be needed in these patients. Intravenous amiodarone is the drug of choice; for ischemic cardiomyopathy lidocaine may also be considered. A recent trial (VANISH-Ventricular Tachycardia Ablation versus Escalation of Antiarrhythmic Drugs) reported in the *New England Journal of Medicine* suggested that catheter ablation should be offered to patients with ischemic cardiomyopathy, an ICD, and recurrent VT while taking amiodarone. For patients who have VT while on another antiarrhythmic drug or no antiarrhythmic drug, catheter ablation does not appear to be superior to treatment with high doses of amiodarone.[6] The findings are controversial and do not form part of current guidelines, and catheter ablation remains the procedure of choice for patients with VT storm.

Ventricular tachycardia ablation is a highly complex procedure with procedure times lasting from 6-12 hours performed by cardiac electrophysiologists and a 4-5% risk of complications, including death. In the vast majority of cases ablation can be life-saving.[7] In cases where VT cannot be controlled, patients should be considered for heart transplantation which unfortunately does not provide a cure but changes the current set of problems for another set of problems.

Article citation: Singh A. Ventricular tachycardia. The Southwest Respiratory and Critical Care Chronicles 2017;5(17):38-41.
From: Department of Internal Medicine, Texas Tech University Health Sciences Center, Lubbock, TX
Submitted: 12/9/2016
Accepted: 1/1/2017
Reviewer: Scott Shurmur MD, Leigh Ann Jenkins MD
Conflicts of interest: none

References

1. Singh A, Nanda NC. Chapter 24: Ventricular Arrhythmia in Women. In: *Heart Disease in Women,* 1st Edition, New Delhi, India: Jaypee Brothers Medical Publishers (P) Ltd., 2015.
2. European Heart Rhythm Association, Heart Rhythm Society, Zipes DP, et al. ACC/AHA/ESC 2006 guidelines for management of patients with ventricular arrhythmias and the prevention of sudden cardiac death: a report of the American College of Cardiology/American Heart Association Task Force and

the European Society of Cardiology Committee for Practice Guidelines (Writing Committee to Develop Guidelines for Management of Patients With Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death). J Am Coll Cardiol 2006; 48:e247-346.

3. Buxton AE, Fisher JD, Josephson ME, et al. Prevention of sudden death in patients with coronary artery disease: the Multicenter Unsustained Tachycardia Trial (MUSTT). Prog Cardiovasc Dis 1993; 36:215-226.
4. Wyse DG, Talajic M, Hafley GE, et al. Antiarrhythmic drug therapy in the Multicenter UnSustained Tachycardia Trial (MUSTT): drug testing and as-treated analysis. J Am Coll Cardiol 2001; 38:344-351.
5. Coggins DL, Lee RJ, Sweeney J, et al. Radiofrequency catheter ablation as a cure for idiopathic tachycardia of both left and right ventricular origin. J Am Coll Cardiol 1994; 23:1333-41.
6. Sapp JL, Wells GA, Parkash R, et al. Ventricular Tachycardia Ablation versus Escalation of Antiarrhythmic Drugs (VANISH). N Engl J Med 2016 Jul 14; 375(2):111-21.
7. Aliot EM, Stevenson WG, Almendral-Garrote JM, at al. European Heart Rhythm Association; European Society of Cardiology; Heart Rhythm Society. EHRA/HRS Expert Consensus on Catheter Ablation of Ventricular Arrhythmias: developed in a partnership with the European Heart Rhythm Association (EHRA), a Registered Branch of the European Society of Cardiology (ESC), and the Heart Rhythm Society (HRS); in collaboration with the American College of Cardiology (ACC) and the American Heart Association (AHA). Europace 2009 Jun; 11(6):771-817.