Wide Complex Tachycardia

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KEYWORDS

• Wide complex tachycardia • Ventricular tachycardia • Supraventricular tachycardia with aberrancy • Catheter ablation

KEY POINTS

- A patient with a wide complex tachycardia needs immediate attention because it is often a life-threatening arrhythmia, especially in the presence of structural heart disease.
- Common electrocardiographic (ECG) criteria for ventricular tachycardia (VT) include wide QRS, right superior axis, positive QRS concordance, atrioventricular dissociation, capture beat, fusion beat, and absence of typical bundle branch block pattern.
- Supraventricular tachycardia with aberrancy (SVT-A) has a typical BBB pattern, and a baseline ECG may be helpful. When doubt exists, patients should be treated for VT, especially those with structural heart disease.
- Hemodynamically unstable rhythm is treated with DC shock, and stable rhythm due to SVT-A can be terminated with intravenous adenosine and atrioventricular nodal blocking agents.
- Preexcited atrial fibrillation or flutter should be treated with intravenous procainamide or amiodarone.
- SVT-A and VT are treated effectively with catheter ablation.

A patient with a wide complex tachycardia (WCT) needs immediate attention because it is often a life-threatening arrhythmia, especially in the presence of structural heart disease. Therefore, physicians must have a good understanding of the differential diagnosis of WCT so that a malignant arrhythmia, such as ventricular tachycardia (VT), can be differentiated from a benign arrhythmia, such as supraventricular tachycardia with aberrancy (SVT-A) (**Box 1**). A hemodynamically stable VT in a patient with structural heart disease mistaken for a benign arrhythmia such as SVT-A is one of the most common errors in the interpretation of WCT. This error in the judgment may be life threatening when these patients are treated with an intravenous calcium blocker, which may lead to hemodynamic instability. The quick and correct diagnosis not only helps in prompt treatment, but also helps in the optimal long-term management of these patients. This review discusses the differential diagnosis of WCT using various electrocardiographic (ECG) criteria, correlation with intracardiac findings during electrophysiology (EP) study, and its management in brief.

MECHANISM OF WCT

In adults, normal QRS is usually less than 110 ms and mostly is between 60 and 80 ms, because the ventricular activation is rapid via the His-Purkinje system (HPS; His bundle, bundle branches, and fascicles).[1] During a normal

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Box 1
Terminology

Wide-complex tachycardia (WCT): A wide QRS complex on tachycardia on electrocardiogram is defined as rhythm with QRS duration ≥120 ms and rate ≥100 beats per minute.

Ventricular tachycardia: tachycardia with a focus or reentrant circuit in the ventricle below the bundle of His

Supraventricular tachycardia: tachycardia requiring participation of structures above bundle of His

Left bundle branch block (LBBB) configuration[1]:

1. QRS duration ≥120 ms with predominantly negative terminal portion of lead V1.
2. Broad-notched or slurred R wave in leads I, aVL, V5, and V6 and an occasional RS pattern in V5 and V6 attributed to displaced transition of QRS complex.
3. Absent q waves in leads I, V5, and V6, but in the lead aVL, a narrow q wave may be present in the absence of myocardial pathology.
4. R peak time greater than 60 ms in leads V5 and V6 but normal in leads V1, V2, and V3, when small initial r waves can be discerned in the above leads.
5. ST and T waves usually opposite in direction to QRS.

Right bundle branch block (RBBB) configuration (positive T wave in leads with upright QRS may be normal [positive concordance])[1]:

1. QRS duration ≥120 ms with predominantly positive terminal portion of lead V1
2. Rsr′, rsR′, or rSR′ in leads V_1 or V_2. The R′ or r′ deflection is usually wider than the initial R wave. In a minority of patients, a wide and often notched R-wave pattern may be seen in lead V1 and/or V2.
3. S wave of greater duration than R wave or greater than 40 ms in leads I and V6.
4. Normal R peak time in leads V5 and V6 but greater than 50 ms in lead V1.

myocardial conduction, QRS can widen when one of the bundle branches fails to conduct due to anatomic block at baseline or functional block during a rapid rate (aberrancy) during SVT. This is because the ventricular activation is transmyocardial from the nonblocked portion of the HPS (conducting bundle branch or fascicle) to the contralateral ventricle with a nonconducting bundle branch. The QRS also may widen when the major part of ventricular activation is transmyocardial, such as during VT, antidromic atrioventricular tachycardia (ART) via a manifest pathway, paced rhythm, electrolyte imbalance, or with the use of antiarrhythmic therapy.

VT

VT is the most common cause of WCT, accounting for 70% to 80% of cases, which occurs mostly in the presence of significant structural heart disease, such as coronary artery disease (CAD) or cardiomyopathy and less commonly in the presence of normal heart. The QRS complex has a left bundle branch block (LBBB) or a right bundle branch block (RBBB) pattern during VT depending on the origin of the VT (right vs left ventricle). As a general rule, the LBBB-pattern VT arises from the right ventricle (RV) or septal aspect of the left ventricle (LV), and the RBBB pattern VT arises from the LV.

SVT-A

SVT-A is the second most common cause of WCT. Patients with permanent BBB will have a similar QRS morphology during sinus tachycardia or an SVT, whereas rate-related BBB during SVT resembles VT, and needs a closer look by using diagnostic criteria for differentiation.

Antidromic AV Reentrant Tachycardia (ART)

In Wolff-Parkinson-White (WPW) syndrome, abnormal ventricular activation occurs via an accessory pathway (AP) that almost always inserts into the epicardial aspect of the ventricular muscle near the atrioventricular (AV) groove. This results in preexcitation (delta waves). During ARTs, ventricular activation initially occurs transmyocardially when the impulse reaches the ventricle from the atria via the AP, and the transmyocardial impulse conduction continues till it reaches the HPS to return to the atrium again via the AV node. The initial portion of the QRS deflection (delta wave) during ART is similar to the sinus rhythm QRS. Therefore,