ECG for Students and Associated Professionals

Wide QRS Complex Tachycardia in a Young Healthy Man

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1. Case presentation

A 22-year-old man presented to the emergency room with palpitation. He had been suffering palpitations several times per year since the age of 17. Recently, the frequency of palpitations had increased to twice per week. The palpitation attacks usually lasted for one hour and terminated spontaneously. However, the present attack had been persisting for more than five hours. His pulse was 170/min and BP was 110/70 mmHg, and his general condition was good. A physical examination showed no abnormality except for rapid pulse. A chest X ray was normal without cardiomegaly. Echocardiography performed three months ago showed normal left ventricular contraction without any structural abnormality. Twelve-lead ECG showed wide QRS complex tachycardia (duration of 0.14 sec) with a right bundle branch block and left axis configuration (Figure 1).

What is your diagnosis of the tachycardia?
2. Commentary

His ECG showed a regular wide QRS complex tachycardia. The QRS duration was 0.14 sec and QRS configuration was a right bundle branch block with left axis deviation (Figure 1). Closer inspection revealed P waves of presumably sinus origin indicated by arrows in lead V1. Also, some QRS beats showed a slight configuration change with shorter duration indicated by stars in lead V2, suggesting fusion beats. These findings suggest that atrioventricular (AV) dissociation is present, which leads to the diagnosis of ventricular tachycardia (VT). AV dissociation became more apparent when the tachycardia rate was slowed by verapamil (Figure 2, P waves indicated by arrows).

When patients present with a wide QRS complex tachycardia, differentiation of VT from supraventricular tachycardia with aberration is an important challenge. Classically, VT has been believed to be nearly always associated with a hemodynamically unstable condition such as loss of consciousness or hypotension. However, it has been reported that about 50% of VT patients come to the emergency room complaining only of palpitation. This is especially common in patients with idiopathic VT.

Several criteria for differentiating VT from supraventricular tachycardia with aberration have been proposed. They are based on features of the QRS complex including its width, morphology in precordial leads and axis in the frontal plane. Brugada emphasized that the absence of an RS complex in all precordial leads or RS interval of more than 100 msec if an RS complex is present is suggestive of VT. Recently, Vereckei proposed that careful observation of lead aVR is useful in diagnosis of wide QRS complex tachycardia. The presence of an initial dominant R wave or an initial r or q wave > 40 msec is suggestive of VT. Unfortunately, the specificity of these criteria for VT is around 70%. More sophisticated criteria of QRS morphology for differentiating VT from supraventricular tachycardia with aberration have been proposed to improve the specificity and sensitivity.

In a wide complex QRS tachycardia, an atrial rate slower than the ventricular rate (AV dissociation) strongly suggests VT. While the presence of AV dissociation establishes VT as the diagnosis, its absence is not as helpful, since P waves are often difficult to identify during a wide complex QRS tachycardia. It has been reported that the presence of AV dissociation in a wide QRS tachycardia is 100% specific for VT, but its sensitivity is only about 10%. Thus, it is very important to look for a slight T wave morphology change during the tachycardia, which is suggestive of hidden P waves within T waves (Figure 1, arrows). Fusion occurs when one impulse originating from the ventricle and a second supraventricular impulse simultaneously activate the ventricular myocardium. The resulting QRS complex has morphology intermediate between that of a sinus beat and a purely ventricular complex. Intermittent fusion beats during a wide QRS complex tachycardia are diagnostic of AV dissociation and therefore of VT.

VT is usually associated with organic heart disease, ischemic heart disease and cardiomyopathy being the most common causes. However, VT may occur in patients who have no significant structural heart disease. Several distinct entities of idiopathic VT have been recognized, and they have been classified with respect to 1) VT origin (left or right), 2) response to pharmacological agents (verapamil-sensitive VT, adenosine-sensitive VT), and 3) behavior of VT (repetitive or sustained). The two most common types of idiopathic VT are a repetitive VT originating from the right ventricular outflow tract and a verapamil-sensitive sustained VT originating from the left ventricle.

Idiopathic verapamil-sensitive sustained VT has the following clinical characteristics: 1) the QRS configuration of VT is a right bundle branch block with either a left axis deviation (Figure 1) or a right axis deviation (uncommon); 2) the QRS duration is relatively narrow (0.12–0.16 sec), and 3) verapamil is highly effective in terminating the VT (Figure 2). Electrophysiological studies have shown that the VT is most commonly originated from the inferoseptal region of the left ventricle (left axis deviation pattern). The mechanism of the VT is macro-reentry, but the precise reentrant circuit has not been clarified yet. It has been reported that a false tendon of the left ventricle is a part of the reentrant limb, and diastolic potentials (pre-Purkinje potentials), which are almost invariably seen during
the VT, are believed to be originated from the false tendon.

Idiopathic verapamil-sensitive sustained VT is usually well tolerated and it is more of an annoyance than a life-threatening event. Thus, patients with this VT are usually treated on a symptomatic basis, and oral verapamil alone or in combination with other drugs is usually effective for alleviating the symptoms. However, some patients are resistant to oral medication and eventually require catheter ablation.

References