A Case of Left Bundle Branch Block-shaped Wide QRS Complex Tachycardia With Diagnostic Ambiguity on a Surface Electrocardiogram

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ABSTRACT

A 78-year-old woman presented with palpitations and wide QRS complex tachycardia with left bundle branch block morphology on an electrocardiogram (ECG). The Brugada algorithm suggested that the tachycardia was supraventricular in origin. However, electrophysiological study showed that the tachycardia was ventricular in origin with 1:1 ventriculoatrial conduction. Here, we report a case of broad complex tachycardia with diagnostic ambiguity on a surface ECG.

Key words: arrhythmia catheter ablation premature ventricular contraction

Introduction

In cases of tachycardia with a broad QRS complex, it is important to differentiate between supraventricular tachycardia (SVT) and ventricular tachycardia (VT). Electrocardiogram (ECG)-based differential diagnoses include VT vs. SVT with aberrant conduction, pre-existing bundle branch block (BBB), intraventricular conduction disturbances, and pre-excitation. Several criteria have been described for differentiation between VT and SVT in the presence of a wide QRS complex. We report a case of wide QRS complex tachycardia with left BBB (LBBB) morphology and a retrograde P wave on the surface ECG.

Case report

A 78-year-old woman presented to our hospital with palpitations and chest discomfort. She had a 6-year history of non-ST segment elevation myocardial infarction (MI); however, she had not received treatment. On physical examination, blood pressure, pulse rate, and respiratory rate were 94/63 mmHg, 171 bpm, and 18/min, respectively. Echocardiography revealed an enlarged left ventricle (5.7 cm) and left atrium (5.0 cm) with preserved left ventricular systolic function (ejection fraction, 53%). There was moderate hypokinesia on the inferior wall from the base to the apex and from the mid-posterolateral wall to the apex of the posterolateral wall.
A 12-lead ECG showed wide QRS tachycardia with LBBB morphology (Figure 1A). The duration of the QRS complex was 148 ms, and the axis was normal. RS complexes were observed in leads V2–3, and R to S intervals in those leads were 72 and 84 ms, respectively. A retrograde P wave was observed on the terminal portion of the QRS complex. There was no S wave in lead V1, and the duration of the S wave in lead V2 was 40 ms. In lead V6, there was only an R wave.

**Figure 1.** Initial surface 12-lead ECG (A) and the ECG after administration of diltiazem (B).
Figure 2. Surface electrocardiogram with intracardiac electrograms was displayed from top to bottom. Wide tachycardia that was identical with clinical tachycardia was induced with ventricular pacing. Immediately after cessation of ventricular pacing, another morphology of PVC was observed and tachycardia was sustained. That was fusion beat. The arrow indicates a fusion beat.

Figure 3. Atrial pacing during tachycardia revealed atrioventricular dissociation. The response immediately after cessation of atrial pacing was a ventricular-ventricular-atrial response.
with no Q wave. The tachycardia was terminated by a 10 mg intravenous dose of diltiazem (Figure 1B). Based on the Brugada algorithm, the ECG findings of this patient strongly suggested a tachycardia of supraventricular origin.1

After informed consent was obtained, the patient underwent a cardiac electrophysiological study. Multipolar electrode catheters were advanced into the femoral vein and positioned in the right atrium, His-recording region, right ventricular apex, and coronary sinus. Retrograde conduction was existed via the atroventricular (AV) node. A fusion beat was observed immediately after cessation of ventricular pacing, and a sustained tachycardia, which was morphologically identical to the patient’s clinical tachycardia, was induced. During tachycardia, the ventricular electrogram preceded each His potential, and 1:1 ventriculoatrial conduction was observed (Figure 2). This tachycardia was entrained with ventricular pacing at the right ventricular apex, and the difference between the post-pacing interval and the tachycardia cycle length was 103 ms. Atrial pacing during tachycardia revealed AV dissociation and a ventricular–ventricular–atrial response, which was observed immediately after the last atrial paced complex (Figure 3). The morphology of the tachycardia was changed, and the tachycardia was terminated (Figure 4). Because the tachycardia had been considered supraventricular in origin before electrophysiologic study, a 3D mapping system was not prepared, and the procedure was finished.

Discussion

Wide QRS complex tachycardia still presents a diagnostic challenge with a 12-lead ECG. ECG-based differential diagnoses include VT vs. SVT with aberrant conduction, pre-existing BBB, involv-
traventricular conduction disturbances, and pre-excitation, VT is the most important differential diagnosis because of its unfavorable prognosis. An accurate diagnosis with immediate treatment is usually required. A delayed diagnosis of VT or a misdiagnosis followed by inappropriate intravenous administration of drugs used for the treatment of SVT, such as verapamil and adenosine, can cause severe hemodynamic deterioration and may provoke ventricular fibrillation and cardiac arrest.

Surface ECG may provide important clues for the classification of a tachycardia as either SVT or VT. In 1978, Wellens et al. noted that among LBBB tachycardias, QR or QS complexes in lead V6 favored a diagnosis of VT, although all other QRS morphologies in this lead were nonspecific, and significant Q waves in V6 occurred infrequently in patients with VT. However, Kindwall et al. determined that SVT with LBBB exhibited features of intact right bundle branch conduction, reflected in a frequent occurrence of small, narrow (<30 ms) R waves in the anterior precordial leads (V1 and V2) followed by rapid and abrupt negative S waves with corresponding R to nadir of S intervals of <60 ms. In contrast, an R wave duration >30 ms, notched and slurred downstrokes to the S waves, and/or R to nadir of S intervals of >60 ms in leads V1 or V2 favored the diagnosis of VT, as did Q waves in lead V6. Based on a modification of these observations, Brugada et al. developed an algorithm for differentiation of wide complex tachycardia. The absence of an RS complex or an R to nadir of S interval of >100 ms in any precordial lead strongly favors VT. If neither of these criteria are satisfied, the presence of AV dissociation or, in the case of LBBB morphologies, a notched S wave in V1 or Q wave in V6 also suggest VT.

Taken in sequence, the algorithm provides a sensitivity and specificity of 98.7% and 96.5%, respectively, for the diagnosis of VT. However, in addition to the ambiguity of diagnosis, it is difficult to measure precise figures at a paper speed of 25 mm/s, as the difference between 30 and 40 ms is 0.25 mm. These factors, in combination with the difficulty in determining the onset of the initial deflection of the QRS complex, led to a degree of inter-observer variation in this study, thereby reducing the objective diagnostic potential of the technique. In 1991, Griffith et al. performed a multivariate analysis in 102 patients to identify which of 15 clinical or 11 ECG variables are independent predictors of VT. They found that the following factors assisted diagnosis of VT: (i) Previous MI is an independent predictor of VT. (ii) A predominant negative deflection in lead aVF is suggestive of VT, especially when a Q wave is present in right BBB (RBBB) pattern tachycardia. In LBBB pattern tachycardia, a QS or qR waveform in lead aVF is highly suggestive of VT, whereas an Rs complex is specific for SVT. (iii) In RBBB pattern tachycardia, a monophasic or biphasic waveform in lead V1 suggests VT and a triphasic RSR, rSR configuration suggests SVT. (iv) A ≥40° change in axis between sinus rhythm and tachycardia is an independent predictor of VT. If none of the above variables are observed, the diagnosis is almost certainly SVT. If one criterion is noted, the diagnosis is probably SVT. If 2 criteria were noted, the diagnosis is probably VT. If 3 or 4 criteria are observed, the diagnosis is almost certainly VT. The predictive accuracy of this method was 93%, which increased to 95% with the inclusion of 2 other criteria: independent P wave activity and ventricular ectopic beats during sinus rhythm with the same QRS morphology as that in tachycardia. According to Griffith’s criteria, the history of MI and presence of a QS wave in lead aVF in our case favors a di-
agnosis of VT. In 2008, Vereckei et al. presented a simplified algorithm using only lead aVR; this algorithm showed high accuracy in the analysis of 313 patients. The criteria for VT in lead aVR were as follows: (i) the presence of an initial R wave, (ii) >40 ms width of an initial R or Q wave, (iii) notching on the initial downstroke of a predominantly negative QRS complex, and (iv) \( \frac{Vi}{Vt} \leq 1 \). In the present case, \( Vi \) and \( Vt \) were 239 and 400 µV, respectively, resulting in \( \frac{Vi}{Vt} \leq 1 \) and therefore favoring a diagnosis of VT by Vereckei’s criteria.

In conclusion, wide complex tachycardia often exhibits an indistinct morphology, especially at higher frequencies, making diagnosis difficult. Despite all available morphological criteria, wide complex tachycardias are still misdiagnosed or can remain undiagnosed. To achieve a high positive predictive value of >95% in the identification of VT, a systemic approach that employs a combination of various ECG and clinical criteria is needed.

References