Case Report

Wide-QRS-Complex Tachycardia with a Negative Concordance Pattern in the Precordial Leads: is a supraventricular origin possible?

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Abstract

The initial electrocardiographic evaluation of every tachyarrhythmia should begin by addressing the question of whether the QRS complex is wide or narrow. The most important cause of wide complex tachycardia (WCT) is ventricular tachycardia (VT). However, supraventricular tachycardia (SVT) can also manifest with a wide QRS complex. The ability to differentiate between SVT with a wide QRS due to aberrancy or preexcitation and VT often presents a diagnostic challenge. The identification of whether WCT has a ventricular or supraventricular origin is critical because the treatment for each is different, and improper therapy may have potentially lethal consequences. When all QRS complexes in the precordial leads are either upright or negative (positive or negative concordance, respectively), VT is strongly suggested. Negative concordance is virtually diagnostic of VT generated from the anteroapical left ventricle. We report an extremely rare case of SVT presenting with a WCT and negative concordance. (J Cardiovasc Thorac Res 2009; Vol.1 (1): 29-32)

Keywords: Wide complex tachycardia • Ventricular tachycardia • Supraventricular tachycardia • Aberrant conduction

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Introduction

The initial electrocardiographic evaluation of every tachyarrhythmia should begin by addressing the question of whether the QRS complex is wide or narrow (of normal duration). The most important (“until proven otherwise”) cause of WCT is VT. Usually, a narrow complex indicates SVT, a term which subtends any mechanism in which the initial site of cardiac activation is at or above the atrioventricular (AV) junction (i.e., above the bifurcation of the bundle of His). However, SVT can also manifest with a WCT. The ability to differentiate between SVT with a wide QRS complex due to aberrancy or preexcitation, and VT, often presents a diagnostic challenge. The distinction is critical because the treatment for each is different, and improper therapy may have potentially lethal consequences. When all QRS complexes in the precordial leads are either upright or negative (positive or negative concordance, respectively), VT is strongly suggested. Negative concordance is virtually diagnostic of VT generated from the anteroapical left ventricle. Positive concordance is strongly suggestive of VT generated from the posterobasal left ventricle but may occur with a posterior bypass tract. WCTs are classically caused by 1 of the following 4 mechanisms: VT, SVT with aberrancy due to conduction slowing or BBB, SVT with anterograde conduction over an accessory AV pathway, and a wide QRS complex generated by ventricular pacing. Only three case reports of SVT proven by electrophysiologic study (EPS) and exhibiting a wide precordial negative QRS concordance have been reported in the literature.

Case report

A 57-year-old female presented with frequent episodes of well-tolerated, WCT. The 12-lead electrocardiogram (ECG) of the tachycardia showed a rate of 178 beats/min with left bundle branch block (LBBB) and left axis deviation morphology (Fig.1). A complete negative concordance pattern of the QRS complex in leads V1 to V6 was evident. The patient was a hypertensive, obese, and hypercholesterolemic. The base-line ECG and Echocardiography were both normal. We performed an EPS in order to elucidate the underlying mechanism of the tachycardia. The study was done following standard techniques and protocols. During atrial pacing at a cycle length of 400 ms followed by a single atrial extrastimulus with a coupling interval of 280 ms, the AV conduction time increased from 210 to 336 ms and initiation of AV nodal reentry tachycardia (AVNRT) of common type (slow-fast) was observed. However, spontaneously, the induced narrow-QRS AVNRT (Fig.2-a) was followed by a wide-QRS tachycardia, identical with the patient’s clinical tachycardia (Fig.2-b). Endocardial recordings during both narrow and wide QRS tachycardias showed a 1:1 relationship between ventricular and atrial activity. Atrial activation started in the His-bundle recording indicating normal VA conduction over the conduction system. Ventricular tachycardia and antidromic reentrant tachycardia were excluded easily by the H-V-A sequence occurring while the surface QRS was recorded after the His electrogram. Successful radiofrequency ablation of the slow pathway was subsequently performed. Repeated programmed stimulation, before and after isoproterenol infusion, did not succeed in displaying residual dual AV node physiology and in inducing any type of tachycardia.

Discussion

Aberrant conduction during SVTs is not uncommon. Although many electrocardiographic algorithms have been developed, aiming to efficiently differentiate SVTs from VTs, unusual morphology of QRS due to aberrant conduction in SVTs can be misleading, challenging the diagnostic accuracy of these algorithms. In our case, the occurrence of aberrancy after the onset of tachycardia without any detectable alteration in the cycle length of the tachycardia, could be attributed not to the classical Ashman phenomenon, but to the unusual fatigue phenomenon in the His-Purkinje system, which has been shown to occur in specific atrial rates and could result in the functional LBBB that was observed. Differential diagnosis of tachycardias in patients with special anatomical characteristics can also be misleading. Volders et al. have recently presented a similar case, which was attributed to morphological abnormalities of the patient’s chest. Rhee and Nam reported another patient with laterally detected ventricles in MRI and speculated that it may be involved in the abnormal ventricular activation during tachycardia, producing negative precordial
concordance. Our patient was obese, and hence, anatomically based particular orientation of the heart vector cannot be excluded, even though we observed no specific chest deformity, nor abnormal position of the heart in our patient’s chest x-ray. Interestingly, and contrary to our patient, the resting ECGs in all three of the previous reported cases, exhibited deep S waves in leads V6 and/or V5. It has been postulated that the deep S waves in lead V5 or V6 in patients with laterally directed cardiac apices may originate from the electrical impulse propagating away from the cardiac apex toward the base. If functional LBBB occurs in these hearts, the apical activation evoked by the intact right bundle branch occurs quickly, and the major portion of the wide QRS complex is formed by the impulse that propagates away from the apex without using the fast conducting system, hence, producing negative concordance during a VT with functional LBBB. Although this mechanistic proposal is very attractive, unfortunately, it could not be interpolated to our patient, since the baseline ECG was completely normal and did not show any deep S wave in lateral precordial leads.

In conclusion, although the commonly accepted electrocardiographic algorithms strongly suggest that the presence of wide-QRS tachycardias with negative concordance in the precordial leads is indicative of a VT originating from the apical area of the left ventricle, the diagnosis of VT with aberrancy cannot be definitely excluded on the basis of these electrocardiographic findings. The contribution of the EPS in the management of unusual cases of wide-QRS tachycardias with negative concordance pattern remains most valuable.

![Figure 1](image1.png)  
**Figure 1.** Twelve-lead surface ECG recording during clinical tachycardia with wide QRS complexes. A negative concordance pattern is evident in all precordial leads.

![Figure 2-a](image2-a.png)  
(Fig. 2-a)  

![Figure 2-b](image2-b.png)  
(Fig. 2-b)  

**Figure 2.** Surface ECG recordings and intracardiac electrograms recorded from the right atrium (RA), His bundle (HISp) proximal and distal (HISd), proximal (CS 9-10) to distal (CS 1-2) coronary sinus, and right ventricle (RV). The cycle length of the two tachycardias is identical (338 ms), a His bundle electrogram precedes every QRS complex with normal HV interval in both narrow (a) and wide (b) QRS tachycardias, while the pattern of retrograde conduction is identical in both tachycardias and occurs through the normal atrioventricular conduction system, and the retrograde P-wave coincides with the QRS complex. In both tachycardias, the electrograms are suggestive of the common type of AVNRT.
References