Narrow QRS tachycardia to wide QRS tachycardia with LBBB morphology. What is the mechanism?

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1. Case

A 42-year-old woman was referred for radiofrequency catheter ablation of narrow QRS tachycardia that was terminated with intravenous adenosine. Baseline twelve-lead ECG was normal during sinus rhythm and transthoracic echocardiography demonstrated no evidence of structural heart disease. The electrophysiological study showed a normal AH interval of 114 ms and HV interval of 37 ms during sinus rhythm. Atrial burst pacing reproducibly induced two tachycardias 1) regular narrow QRS tachycardia and 2) wide QRS tachycardia with left bundle branch block (LBBB) morphology. Fig. 1 shows spontaneous transition of narrow QRS tachycardia to wide QRS tachycardia. What are the likely mechanisms of the tachycardia?

2. Discussion

The differential diagnosis for wide QRS tachycardia with 1:1 AV relationship includes supraventricular tachycardia with aberrancy, ventricular tachycardia with 1:1 retrograde conduction and pre-excited tachycardia (supraventricular tachycardia with bystander accessory pathway activation, antidromic AV re-entrant tachycardia (AVRT) and pathway to pathway reentrant tachycardia).

The wide QRS tachycardia has LBBB morphology with left axis, negative HV interval (Fig. 1) with right bundle (RB) electrogram preceding the His bundle (HB) electrogram and earliest retrograde atrial activation at His bundle electrode (HBED). The differentials for a wide QRS tachycardia with negative HV interval are preexcited tachycardia and VT with 1:1 retrograde conduction. To differentiate between the two, an atrial premature beat (APB) was delivered from the right atrium (RA) free wall [1]. The APB delivered here was relatively early coupled even though the classical recommendation is a late coupled APB. This APB resulted in advancement of ventricular electrogram without any change in the QRS morphology (Fig. 2) excluding ventricular tachycardia and making SVT with bystander pathway activation less likely. The ventricular advancement following the APB was not preceded by advancement of atrial electrogram in the His region (HBED) suggesting that antegrade conduction has happened through an extranodal circuit i.e. an accessory pathway. The advanced ventricular electrogram has subsequently reset the LBBB tachycardia by advancing the next atrial electrogram with same retrograde atrial activation sequence suggesting the active participation of the accessory pathway ruling out supraventricular tachycardia with bystander accessory pathway activation [2]. During the LBBB tachycardia, the right bundle activation (Fig. 1) was earlier than that His bundle activation. Moreover the AV interval after the pulled A following the V advancement in response to the APB is prolonged suggesting antegrade conduction by a decrementally conducting accessory pathway. The diagnosis of antidromic AVRT involving right atriofascicular (AF) accessory pathway was confirmed by the above mentioned pacing protocol.

The narrow QRS tachycardia had short VA interval with near simultaneous activation of atrium and ventricle (45 ms) and earliest retrograde activation at His bundle electrodes (HBED). The differentials entertained were slow fast AV nodal reentrant tachycardia (AVNRT) and atrial tachycardia. The entrainment response was suggestive of AVNRT.

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The spontaneous transition of the AVNRT to antidromic AVRT involving AF bypass tract (Fig. 1) is heralded by a block in the slow AV nodal pathway and consequently preventing anterograde conduction through AV node (as the fast AV nodal pathway is already refractory) and thereby allowing anterograde conduction over the AF bypass tract to take over with a faster and wider tachycardia, i.e. antidromic AVRT. The possible mechanism for the transformation may be sudden change in antegrade conduction time in slow AV nodal pathway in response to change in autonomic tone. Another possibility is RV catheter movement at the time, because the HB and RB electrogram in the His catheter are not very good during the narrow QRS tachycardia, which suddenly become much sharper and clearer during the broad complex tachycardia, resulting in a RV catheter induced change in the tachycardia.

Incremental atrial pacing near the lateral RA has resulted in the manifestation of preexcitation over the AF pathway. The AF bypass tract was mapped to 8 o’clock position of the tricuspid annulus and it was successfully ablated. Slow pathway ablation was also performed. Neither tachycardia could be induced after the procedure and no antegrade extra nodal conduction was demonstrated.

Disclosures

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