

Diagnostic Approach

A Wide Complex Tachycardia in a 31-year-old Male

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Abstract

We present the case of a young male presenting with a wide complex tachycardia. The diagnostic approach based on the electrocardiogram findings is discussed. The final diagnosis could be made following an adenosine challenge at baseline, underlying the use of this drug for diagnostic purposes even in the absence of ongoing tachycardia.

Keywords: Adenosine; antidromic AVRT; preexcitation; wide complex tachycardia; Wolff-Parkinson-White syndrome

Case Presentation

A 31-year-old male without any significant medical or surgical history presented to the emergency department of a community hospital complaining of acute onset palpitations. He was not on any medication, did not report any illicit drug abuse and was hemodynamically stable. The 12-lead ECG is shown in figure 1. Following electrical cardioversion, another 12-lead ECG was obtained (fig. 2). At this point, our arrhythmia unit was consulted for advice concerning further work-up and management.

Discussion

The patient presented with a regular wide complex tachycardia (QRS 220 ms) at about 250 bpm. There were no discernible P waves. Differential diagnosis included (1) supraventricular tachycardia (SVT) with aberrancy, (2) SVT with preexistent bundle branch block, (3) antidromic atrioventricular reentrant tachycardia (AVRT), (4) SVT with bystander accessory pathway (AP) and (5) ventricular tachycardia (VT). There was an atypical right bundle branch block-like QRS morphology with tran-

sition in V5 and superior axis (fully positive QRS in aVR), which made option (1) unlikely. The initial part of the QRS deflection was slurred, suggesting that the ventricle was not initially activated from His-Purkinje conduction, but rather through slower myocyte to myocyte conduction (fig. 3). Therefore, a left ventricle basal infero-lateral exit could be sus-

pected (fig. 3). It is impossible to differentiate between option (2) and (3) solely based on QRS morphology. As mentioned earlier, no atrial activity that would allow assessment of the atrioventricular (AV) relationship was seen (AVRT could have been excluded in the presence of AV dissociation). No capture or fusion beats that would suggest a VT were seen. Taking the patient's history into consideration, it would be unusual for a young and healthy individual to present with left ventricular – likely scar-related – VT. Of note, the QRS morphology was neither consistent with an outflow tract VT nor with a fascicular VT, which can both be seen in individuals with structurally normal hearts.

At this point, the diagnosis remained uncertain and therefore the tachycardia should be considered a VT until proven otherwise. However, it would have been reasonable to proceed with vagal maneuvers/carotid sinus massage as the patient was stable [1]. In the ab-

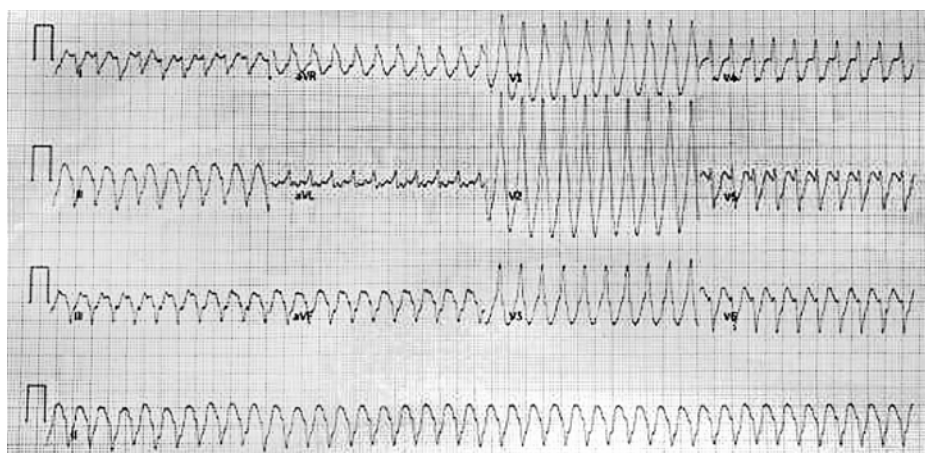


Figure 1: 12-lead ECG showing a regular wide complex tachycardia.

sence of documented pre-excitation on baseline ECG, adenosine may be used for acute treatment of regular wide complex tachycardia of unknown etiology. Termination would suggest an atrioventricular nodal reentrant tachycardia with aberrancy. However, termination may also be achieved in some forms of atrial tachycardia and outflow tract VTs. Although adenosine administration during antidromic AVRT may terminate it, atrial fibrillation could be triggered and lead to ventricular fibrillation secondary to rapid AP conduction [2, 3]. It is therefore not recommended in the current guidelines.

The 12-lead ECG obtained after cardioversion shows subtle pre-excitation. To enhance the latter, we suggested administration of adenosine 6 mg IV (fig. 4). Upon adenosine challenge, a widening of the QRS complexes to closely match the QRS during tachycardia is evident. Some degree of pre-excitation may be seen at baseline when there is competing conduction through the AV node and an AP. However, if AV nodal conduction is predominant, pre-excitation may be subtle or completely absent. This may be the case in slowly conducting APs, decremental APs, or in left lateral APs. In the last example, pre-excitation is referred to as “latent”. Sinus node activity preferentially passes through the AV node as a left lateral AP is electrically located further away. By the time the sinus node impulse reaches the AP, the ventricle may be partially or fully refractory. Adenosine challenge can “unmask” the presence of an AP by blocking or slowing down the AV node conduction, as seen in our case. Full pre-excitation is indeed not achieved with adenosine administration shown by the slight morphologic QRS differences (i.e. negative QRS in lead I during tachycardia, but positive during adenosine challenge). Most likely, this represents a fusion of conduction through the AV node and the AP since a full effect of adenosine was not achieved. If the response is unsatisfactory, administration of adenosine at a higher dose of 12 mg, followed by an efficient saline flush, may be attempted. Of note, in rare circumstances, adenosine may also impair AP conduction and thus can be inaccurate to rule out its presence in case of a negative challenge.

Solution

Although very rare, option (4) cannot be ruled out per se at this point. The patient was brought to our electrophysiology lab where a bidirectional conducting AP at a four o'clock position on the mitral annulus was diagnosed and successfully ablated using radiofrequency. No SVT could be induced at the end of the study and there was no evidence of dual AV nodal

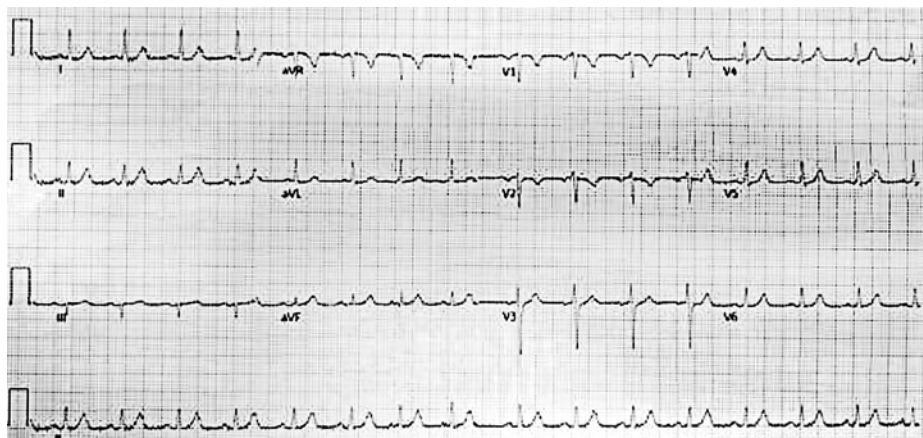


Figure 2: 12-lead ECG obtained after electrical cardioversion.

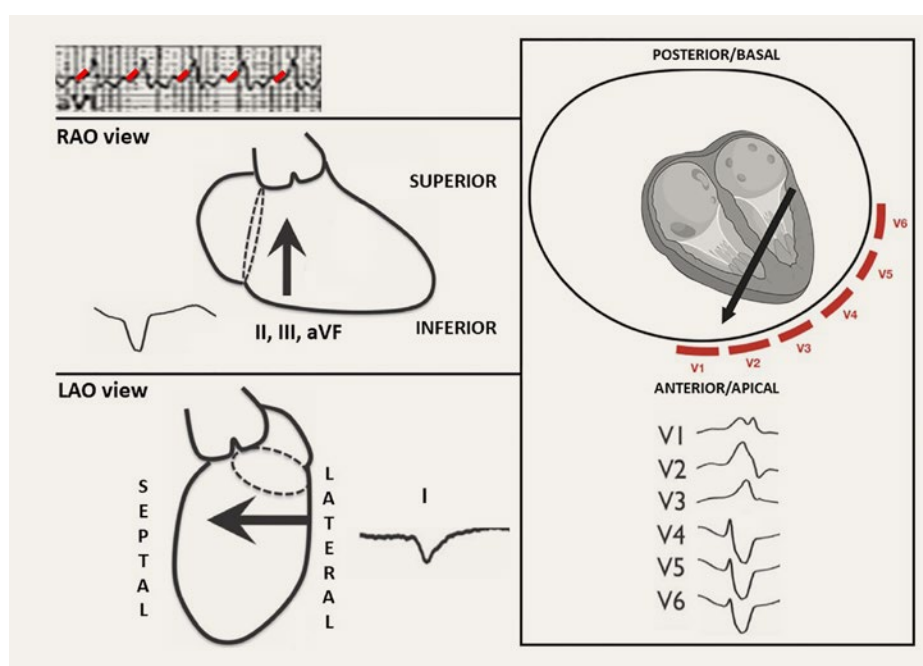


Figure 3: Schematic depiction of the heart showing ventricular depolarization wavefront moving away from the postulated site of origin with expected QRS morphology on ECG derivations. Left upper quadrant: Focus on ECG lead aVL during tachycardia showing initial slurring of the QRS (red oblique line).

LAO: Left anterior oblique. RAO: Right anterior oblique.

physiology. In conclusion, this patient presented with an antidromic AVRT with anterograde conduction through a left posterior-lateral/posterior-inferior AP as part of a Wolff-Parkinson-White syndrome [4].

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Ethics Statement

Written informed consent was obtained from the patient to publish this case report.

Conflict of Interest Statement

The authors have no potential conflicts of interest to declare.

Author Contributions

HV drafted the work.
GA critically reviewed the work for important intellectual content.
Both HV and GA made substantial contributions to the conception of the work, approved the version to be published and agreed to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work were appropriately investigated and resolved.

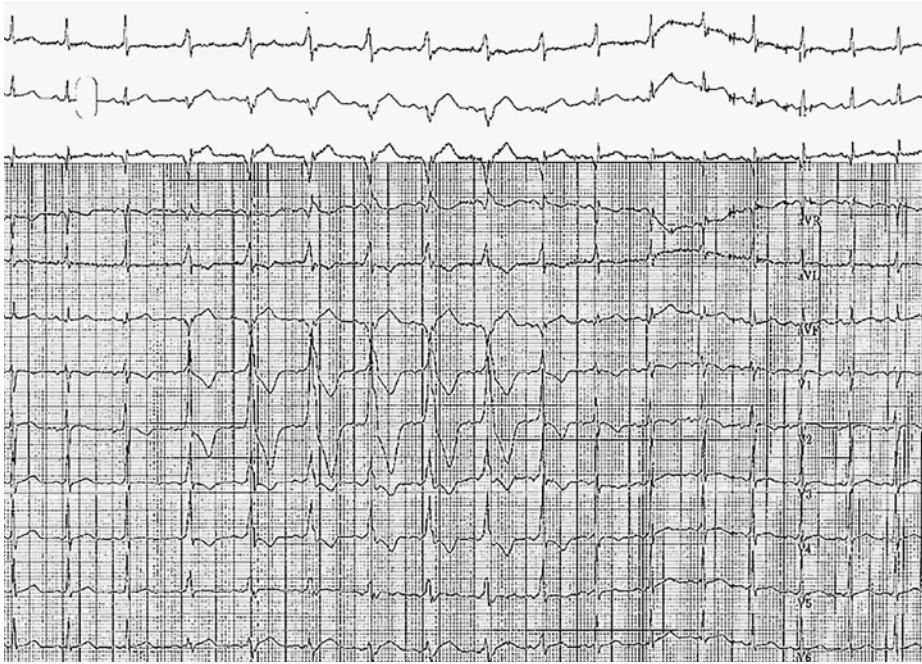


Figure 4: 12-lead ECG obtained during administration of adenosine 6 mg IV.

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