SCHEINMAN/PETER ECG CORNER

A rapid wide complex tachycardia

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Case presentation

A 25-year-old woman presented with frequent bursts of palpitations and shortness of breath experienced while running in preparation for a marathon in 2 days. She experienced rapid palpitations without dizziness or syncope. Upon arrival in the emergency department, her baseline electrocardiogram (ECG) showed sinus bradycardia with right axis deviation (Figure 1A) and later a symptomatic wide QRS complex tachycardia (Figure 1B). Spontaneous premature ventricular complexes (PVCs) matching the ventricular tachycardia (VT) morphology occurred with a coupling interval of 380–400 ms.

She denied prior palpitations or significant medical problems. Her family history was unremarkable.

Commentary

The presenting ECG shows sinus bradycardia at 43 beats/min with right axis deviation. The PR interval is normal at 172 ms with normal QRS and QT intervals.

The subsequent wide complex tachycardia has a rate of 248 beats/min with an atypical right bundle branch block (RBBB) and right superior axis and QRS duration 160 ms. The tachycardia has a transition from a monophasic R wave in lead V_3 to a QS complex in lead V_4.

The tachycardia recurred despite oral metoprolol, diltiazem infusion, sotalol, intravenous procainamide, and cardioversion. Cardiac magnetic resonance imaging revealed normal biventricular systolic function without late gadolinium enhancement.

The patient underwent an electrophysiology study. There was no inducible supraventricular tachycardia (SVT). The wide complex tachycardia spontaneously occurred and exhibited atrial-ventricular dissociation consistent with VT. Activation mapping identified a focal origin of the VT with the earliest site of activation at the base of the posteromedial papillary muscle with a bipolar electrogram that preceded the QRS complex by 30 ms and QS unipolar electrogram (Online Supplemental Figure 1). Pace mapping produced a 12/12 pace-map match at the site of earliest activation. Open-irrigated radiofrequency catheter ablation was performed in the power-controlled mode at 40 W at this site. After successful ablation, the VT could not be induced with right ventricular and left ventricular programmed stimulation with triple extrastimuli with or without isoproterenol infusion.

Discussion

In this young patent with no evidence of structural heart disease, the differential diagnosis for the wide complex tachycardia includes SVT with RBBB aberrancy, mitral annular left VT, left posterior fascicle VT, or left ventricular posteromedial papillary muscle VT.

For this tachycardia, there is clear evidence of atrial-ventricular association; this is inconsistent with atrioventricular reentrant tachycardia or atrial tachycardia. In addition, for RBBB aberrancy, lead V_1 typically has an RsR' or rSr' configuration, which is inconsistent with the V_1 RR' morphology in this case. The monophasic R wave in lead avR and QS pattern in lead V_6 exclude SVT with aberrancy.

Once diagnosing the arrhythmia as VT, thoughts turn to identifying the site of origin for this VT. A'Aref et al[1] used an algorithm analyzing the axis of the tachycardia and subsequently the QRS morphology in leads V_5 and V_1 for differentiation of fascicular, mitral annular, and papillary muscle VT/PVCs. This VT has (1) an RBBB superior axis morphology with a QRS duration of 160 ms and is missing both (2) an $R > S$ in lead V_5 and (3) an $r < R'$ in lead V_1—all consistent with a posteromedial papillary muscle VT. The "RR" morphology in lead V_1 for this VT is 1 of 3 morphologies that has been shown to have a 93% sensitivity and 98% specificity for posteromedial papillary muscle VT.[2] Papillary muscle VTs (QRS duration 150 ± 15 ms) are typically wider than fascicular VTs (121 ± 11 ms).[1] Fascicular VTs also have a small q wave in leads I and avL reflecting left to right septal depolarization via the fascicular system in contrast to the monophasic R wave or an Rs complex in lead I for papillary muscle VT.[3] Papillary muscle VTs in the absence of structural heart disease are typically sensitive to catecholamines, noninducible with programmed stimulation, and unable to be entrained, consistent with a mechanism of triggered

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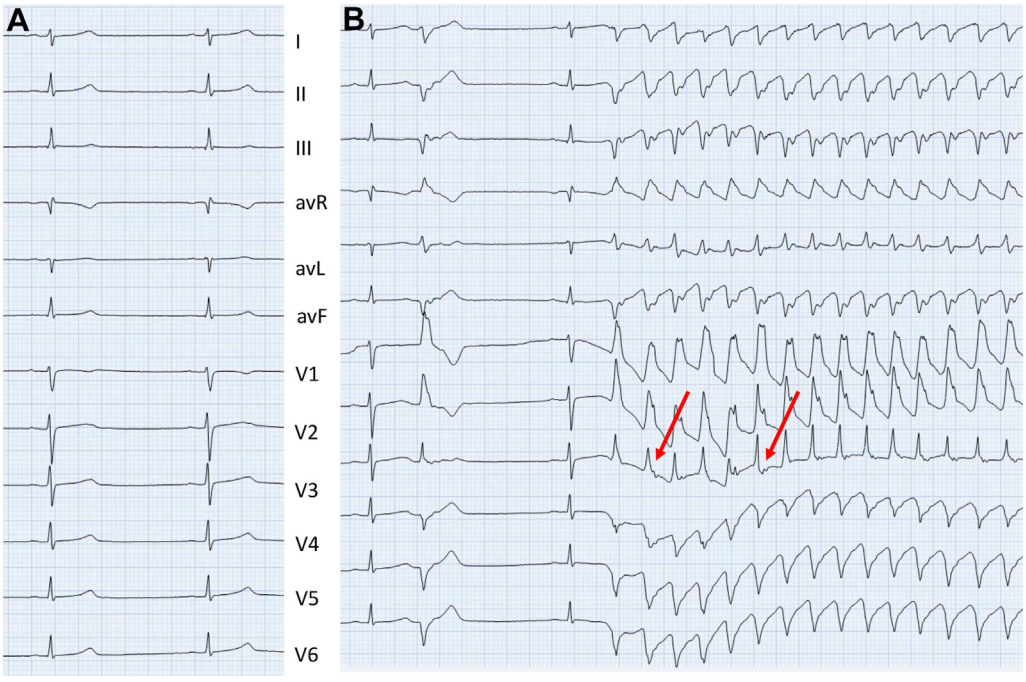

Figure 1 Electrocardiogram. **A:** Presenting rhythm with sinus bradycardia at 43 beats/min and right axis deviation. **B:** Sinus rhythm with right axis deviation, PVC, and wide QRS tachycardia at 248 beats/min. The fourth beat of tachycardia exhibits fusion of the conducted QRS morphology and the VT morphology. Lead V_3 shows a sinus rhythm P wave after the second and sixth beats that are dissociated from the tachycardia. *Red arrows* indicate P waves dissociated from the tachycardia.

activity or abnormal automaticity. In a canine model, the junctional cells located at the Purkinje–ventricular myocardium junction have been shown to exhibit triggered activity that can be suppressed with verapamil, consistent with the clinical manifestation of papillary muscle VT.[4]

Left ventricular papillary muscles are a well-known source of ventricular arrhythmias (VAs) in patients with and without structural heart disease. These VAs can manifest as PVCs, nonsustained VT, sustained VT, and PVC triggers for ventricular fibrillation. The site of origin for these arrhythmias can be mapped to the base, body, or apex of the papillary muscles. The dynamic motion of the papillary muscles can create challenges with achieving adequate catheter stability for successful ablation. Contact force sensing catheters and detailed anatomic maps of the papillary muscles created with intracardiac echocardiography have been shown to increase the rate of successful ablation of papillary muscle VA with acute and long-term success rates of 95% and 91%, respectively.[5]

Conclusion

The differential diagnosis for RBBB wide complex tachycardias in patients with no evidence of structural heart disease include SVT with RBBB aberrancy, antidromic atrioventricular reentrant tachycardia using a left-sided accessory pathway, mitral annular VT, papillary muscle VT, and fascicular VT. Application of diagnostic ECG criteria can allow an individual to successfully differentiate SVT from VT as well as to identify the site of the origin of the VT.

Appendix

Supplementary data

Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.hrthm.2022.04.010.

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