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A Unique Case of Wide Complex Tachycardia Mimicking Ventricular Tachycardia

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INTRODUCTION

Wide complex tachycardia in patients with structural heart disease, such as heart failure with reduced ejection fraction or prior myocardial infarction, is often believed to be Ventricular Tachycardia (VT) [1]. Sustained monomorphic ventricular tachycardia is defined as a cardiac arrhythmia of 3 or more consecutive complexes originating in the ventricles at a rate greater than 100 bpm (cycle length: <600 msec) lasting more than 30 seconds with a uniform QRS morphology from beat to beat [2]. During VT, the ventricular rate is faster than the atrial rate along with Atrioventricular (AV) dissociation, except in situations where there may be retrograde 1:1 VA conduction. When this is identified in the ECG, it is considered the hallmark for VT with nearly 100% specificity [1]. In this case, we demonstrated a rare and unique presentation of a wide QRS rhythm that met the aforementioned criteria but was not VT.

Abbreviations

ACLS: Advanced Cardiac Life Support; ATP: Antitachycardia Pacing; BNP: Brain Natriuretic Peptide; CL: Cycle Length; ECG: Electrocardiogram; EF: Ejection Fraction; ICD: Implantable Cardioverter Defibrillator; LAD: Left Anterior Descending; LVOT: Left Ventricular Outflow Tract; MI: Myocardial Infarction; RCA: Right Coronary Artery; NYHA: New York Heart Association; VA: Ventriculoatrial; VT: Ventricular Tachycardia; VF: Ventricular Fibrillation

CASE PRESENTATION

The patient is a 59-year-old Hispanic male. Past Medical history includes: essential hypertension; Type 2 Diabetes Mellitus; prior cerebrovascular accident involving the right middle cerebral artery with residual left-sided hemiplegia, left carotid artery stenosis (50-79) %; and tobacco use. He was admitted to an outside hospital with

Non-ST-Elevation Myocardial Infarction (NSTEMI) and was newly diagnosed with acute severe LV systolic dysfunction with a Left Ventricular Ejection Fraction (LVEF) of 30%-35%. During Myocardial Infarction (MI), he developed complete heart block, necessitating temporary transvenous pacing, as well as cardiogenic shock requiring Intra-Aortic Balloon Pump (IABP) support. The coronary angiogram showed multivessel coronary artery disease involving 90% stenosis of the proximal Left Anterior Descending (LAD) artery with diffuse disease in distal LAD, chronic total occlusion of large caliber Right Coronary Artery (RCA) with collaterals from the left. After heart team approach, the patient underwent surgical revascularization in October 2023 with coronary artery bypass grafting with a left internal mammary artery graft to the left anterior descending artery, a saphenous vein graft to the first diagonal branch, and a saphenous vein graft to the posterior descending artery. On postoperative day 5, the patient had a witnessed in-hospital cardiac arrest caused by VT and progressed to Ventricular Fibrillation (VF). Resuscitation was successful with Cardiopulmonary Resuscitation (CPR) and emergent defibrillation in accordance with Advanced Cardiac Life Support (ACLS) protocols. A repeat coronary angiogram showed patent grafts. Prior to hospital discharge, a left-sided Medtronic dual chamber implantable Cardioverter-Defibrillator (ICD) was implanted for secondary prevention. The patient was discharged on oral Amiodarone loading dose followed by a maintenance dose of 200 mg daily, along with Mexiletine 200 mg every 8 hours for the management of VT and VF. In December 2023, the patient was readmitted to the hospital for pancytopenia, with laboratory findings showing a White Blood Cell (WBC) count of 1.8 k/μL, Red Blood Cell (RBC) count of 2.56 m/μL, platelet count of 73 k/μL, and an absolute neutrophil count of 0.1 k/μL. A bone marrow biopsy revealed a normocellular marrow (50%) with markedly decreased granulopoiesis and polyclonal (reactive) plasmacytosis. There was no evidence of overt dysplasia or malignancy. The findings also included normochromic, normocytic anemia and marked absolute neutropenia. Given that the patient had a normal neutrophil count three months prior during hospitalization in October 2023, the current neutropenia was suspected to be a result of medication, infection, or another toxic insult to the bone marrow. Plasma cells stained in a polytypic pattern for kappa and lambda by in Situ Hybridization (ISH) and flow cytometry confirmed a polyclonal pattern, indicating reactive plasmacytosis. The patient was evaluated by specialists in Hematology-Oncology and Infectious Disease. He was eventually diagnosed with drug-induced neutropenia, and mexiletine identified as the possible offending drug. Mexiletine was discontinued, leading to improvement in neutropenia. The patient continued amiodarone for the suppression of VT and was discharged from the hospital in stable condition. In May 2024, the patient transferred care to our group and was seen in the office of the Electrophysiology service. ICD interrogation showed several sustained episodes of slow VT, with a ventricular rate of 110 beats per minute, which was below the VT detection zone of the ICD. The patient was asymptomatic and tolerated the slow VT hemodynamically. ECG was obtained during sinus rhythm (Figure 1) and during VT (Figure 2). ECG during wide QRS tachycardia was diagnosed as VT based on captured/fusion beat, AV dissociation and QRS morphology criteria. The QRS morphology (right bundle superior axis, precordial transition at V2-V3, predominantly positive in limb leads I and aVL) was consistent with a left ventricular basal to mid-inferior septal exit. The patient continued to have multiple episodes of VT, some episodes lasting longer than an hour. These episodes were asymptomatic, and no ATP therapy or ICD shocks were delivered, as the episodes remained below

the VT detection zone of the ICD. Due to increasing burden of slow VT, the patient experienced a gradual decline in hemodynamics, with worsening heart failure symptoms, including increased exertional dyspnea, bilateral pitting edema, and elevated BNP levels. The patient was readmitted to the hospital for heart failure in July 2024.

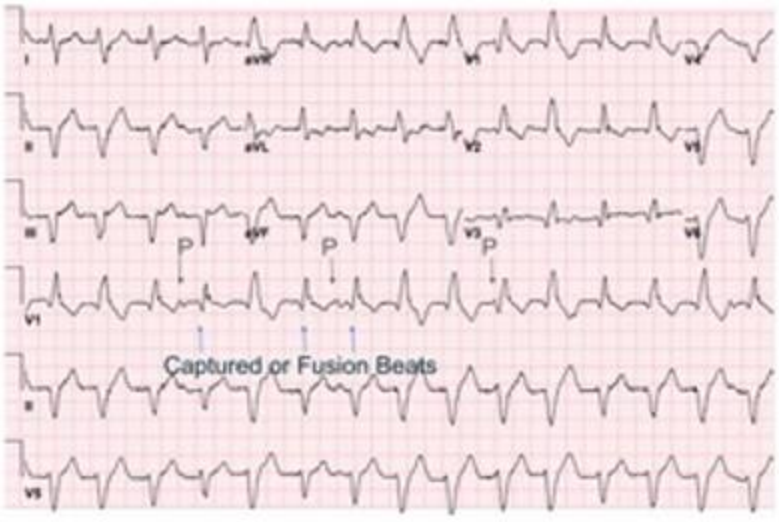

Figure 1: VT ECG.

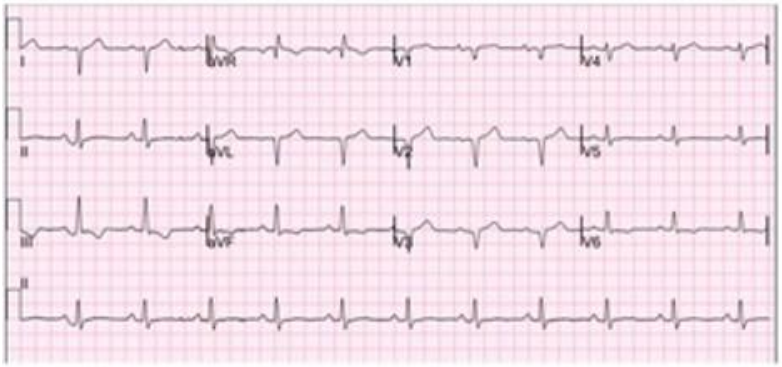

Figure 2: Sinus rhythm ECG.

In August 2024, the patient was scheduled for VT ablation under general anesthesia. Transseptal and transaortic access was obtained. Intracardiac echocardiography and the RhythmiaTM HDx Cardiac Mapping System were utilized in the case. First, a three-dimensional electroanatomic voltage map was collected in sinus rhythm using the INTELLAMAP ORION™ Mapping Catheter. During the case, spontaneous VT was observed that resembled the clinical VT. A timing activation map was collected and the critical isthmus was localized to the basal to mid-infero-septal segment of the left ventricle, near the posteromedial papillary muscle. Using an INTELLANAV STABLEPOINT™ ablation catheter, the VT was successfully ablated using 40 watts of energy, 5-10 grams of

contact force. Each ablation resulted in a 15% to 20% impedance drop, indicating an effective ablation lesion. Intracardiac echocardiography during ablation showed bright echogenicity at the ablation sites, further supporting the effectiveness of the lesions.

Post-ablation, an attempt to induce VT using programmed extra stimulus pacing with triple extra stimuli resulted in a wide QRS rhythm (Figure 3), with a ventricular-to-ventricular cycle length of 580 msec to 600 msec, AV dissociation, and a faster ventricular rate than the atrial rate. This rhythm was initially diagnosed as VT, with an exit site differing from the clinical VT due to the presence of AV dissociation and an atrial rate slower than the ventricular rate. Despite this tachycardia was slow (CL 580-600 sec), VT was still considered since his initial presentation was slow VT. However, the QRS morphology was identical to that of sinus rhythm, with a 99% match. This contradicted the diagnosis of VT, as the QRS morphology in sinus rhythm results from supra ventricular conduction. The INTELLAMAP ORION™ Mapping Catheter was positioned in the left ventricular outflow tract (LVOT) during this tachycardia. The left bundle potentials preceded the QRS with consistent H-V intervals, supporting supra ventricular conduction.

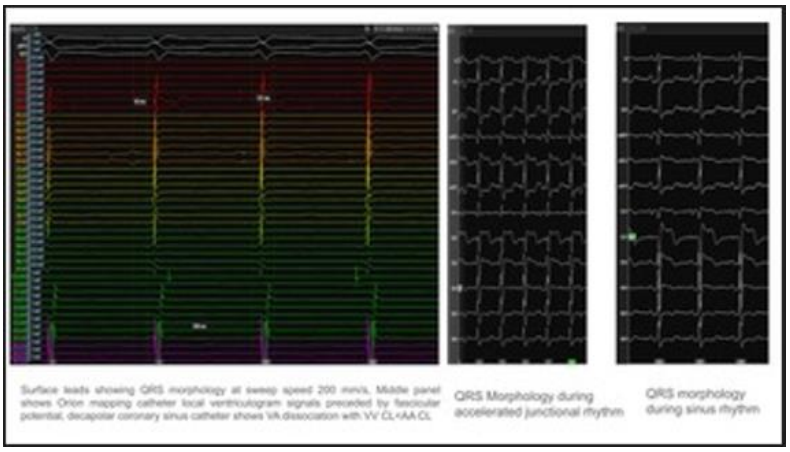

Figure 3: QRS rhythm.

The tachycardia was terminated with overdrive pacing from a quadripolar catheter in the right ventricle. When observing the electrograms on the ORION™ mapping catheter in the LVOT during sinus rhythm, the HIS or left bundle potentials were consistently preceding the QRS. The HV interval during sinus rhythm was shorter than during tachycardia. Conduction disease involving the Purkinje system was suspected due to HIS or left bundle electrograms being complex and fractionated with a distinct split. When tested, the AV Wenckebach was seen at relatively longer cycle length at 500 msec. The patient had no baseline VA conduction.

Based on these findings, it was postulated that this was an accelerated junctional rhythm with antegrade conduction over the His-Purkinje system, but there was no retrograde VA conduction to explain the AV dissociation. There was significant sinus node dysfunction with a slow resting sinus rate, and a competing junctional pacemaker took over the conduction. To confirm this, we programmed the pacemaker at AAI 110/min to make it faster than the junctional

rate. Multiple attempts to induce the arrhythmia using programmed extra stimulus pacing, even up to triple extra stimuli from the RV catheter, failed to induce the arrhythmia. This confirmed the diagnosis of a competing junctional pacemaker rather than VT.

Post-ablation, Amiodarone was discontinued. The pacemaker was programmed with AAIR<=>DDDR 80-120 bpm to promote a faster sinus rate and minimize the chronic RV pacing burden, given the low ejection fraction. The patient has shown improvement and had no recurrence of VT.

DISCUSSION

This patient presented with a complex medical and cardiac history despite being a relatively young individual. He had been experiencing the progressive deterioration of cardiomyopathy and slow VT occurring below the detection zone of the ICD. The 12-lead ECG with a wide QRS rhythm confirmed the diagnosis of slow VT with AV dissociation, exhibiting a QRS morphology different from that during sinus rhythm. Caution is required when interpreting such ECGs, as the slow rate of arrhythmias like slow VT, which can have a significant hemodynamic impact and worse prognosis, may be overlooked or misdiagnosed. On the other hand, the wide complex rhythm that presented post-ablation led to confusion. It would spontaneously induce and could be reproducibly induced using RV pacing. The VT morphology was different from the clinical VT and presented with a Ventricular-to-Ventricular (VV) interval that was shorter than the Atrial-to-Atrial (AA) interval along with AV dissociation. The wide QRS was explained by infra-hisian conduction disease. The ventricular rate was faster than the atrial rate because of the sinus node dysfunction, where the competing junctional rhythm with no retrograde conduction to the atrium looked like VT. This is unusual because, in most cases, the sinus node typically takes over the competing junctional rhythm. However, in this case, the sinus node was particularly slow. When the atrium was paced faster than the junctional rhythm, the arrhythmia was no longer inducible.

Bundle Branch Reentrant Ventricular Tachycardia (BBRVT) was a possible diagnosis, as it is typically seen in patients with dilated cardiomyopathy, can be reproducibly induced with atrial pacing, and requires some infra-hisian delay to facilitate re-entry. During tachycardia, the HV interval is longer than during sinus rhythm, and the QRS morphology is identical to sinus rhythm. This patient exhibited all these features, but BBRVT is usually fast due to the efficient conduction of the His-Purkinje system and is hemodynamically poorly tolerated due to a reduced EF and dilated cardiomyopathy. In addition, VT could not be induced with pacing from the RV catheter when the pacemaker was set to overdrive the junctional rhythm with faster atrial pacing. These factors argued against BBRVT. Accelerated idioventricular rhythm was ruled out as His bundle electrograms preceded QRS. Supraventricular tachycardia with aberrant conduction was ruled out because orthodromic reciprocating tachycardia requires a 1:1 AV relationship. Unusual AV nodal reentrant tachycardia with an upper common pathway block would still require at least a 1:2 AV relationship, ruling out the possibility of this diagnosis. Focal atrial tachycardia was also excluded, as the atrial rate was slower than the ventricular rate.

CONCLUSION

A careful and methodical approach to slow wide QRS tachycardia is essential to avoid missing the diagnosis of slow VT, which can lead to progressive hemodynamic deterioration. In these cases, ablation is feasible. Post-ablation, although there was a wide QRS rhythm >100 bpm with AV dissociation and a ventricular rate faster than the atrial rate-features suspicious for VT-the ablation was successful. This case provides a valuable example of the importance of careful analysis of the QRS morphology when making clinical decisions. During tachycardia, the QRS morphology closely resembled that of sinus rhythm. After electrophysiological testing, the diagnosis was ruled to be a competing junctional pacemaker rhythm rather than VT. This condition was managed with overdrive atrial pacing from the pacemaker, programmed to a faster rate to suppress the competing junctional rhythm.

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