Polymorphic ventricular tachycardia due to change in pacemaker programming

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Introduction

Bradycardia is a known risk factor for QT prolongation and polymorphic ventricular tachycardia (polymorphic VT).[1] With the widespread use of pacemakers, it is important to pay attention to different pacing modes while programming pacemakers to avoid bradycardia. We present a case of bradycardia-induced polymorphic VT that occurred after a VDD pacemaker was replaced with a VVIR pacemaker. The lower pacing rate of the new VVIR pacemaker was set at 50 beats per minute (bpm), similar to the old VDD pacemaker. With the absence of atrial tracking in the VVI mode, the patient was paced at the lower pacing rate. The resultant bradycardia caused significant QT prolongation with subsequent polymorphic VT. Increasing the lower pacing rate corrected the QT prolongation and resolved the polymorphic VT.

Case report

A 79-year-old woman was admitted after 2 episodes of syncope. These occurred without warning and the patient felt normal in between. She presented to hospital after the second episode.

Her background is significant for left total hip replacement, which was complicated by multiple infections. Other comorbidities include hypertension, dyslipidemia, type 2 diabetes mellitus, angina pectoris, obstructive sleep apnea, gastroesophageal reflux disease, and chronic obstructive airway disease from remote smoking history. She had a VDD permanent pacemaker for complete atrioventricular (AV) block.

She presented to our hospital a month earlier with sepsis and had positive blood cultures with methicillin-sensitive *Staphylococcus aureus*. Septic arthritis was excluded as the cause of her sepsis and a transthoracic echocardiogram revealed an independently mobile mass on the right ventricular pacemaker lead. With her bacteremia and pacing lead vegetation, the decision was made to extract the old pacemaker and she received a right-sided VVI pacemaker. She was monitored for a few days after the procedure and then discharged home the day prior to the current presentation.

Her medications at the time of her current presentation were pantoprazole 40 mg daily, acetaminophen CR 1300 mg thrice daily, aspirin 81 mg daily, atorvastatin 40 mg daily, vitamin B12 1000 mcg daily, furosemide 20 mg daily, lactobacillus 2 capsules daily, magnesium oxide 420 mg thrice daily, vitamin D 1000 units daily, salbutamol 100 mcg metered-dose inhaler 2 puffs as required, probenecid 1 g twice daily, and cefazolin 2 g intravenously every 12 hours.

On examination, she appeared well; she was afebrile with unremarkable cardiovascular and neurologic examination and she had no orthostatic hypotension. Her blood work revealed sodium 141 mmol/L, potassium 3.5 mmol/L, magnesium 0.69 mmol/L, creatinine 74 umol/L, urea 3.0 mmol/L, hemoglobin 81 g/L (was 77 g/L on discharge), white blood cell count 5.8 $\times$ 10^9/L, and platelet count of 292 $\times$ 10^9/L.

Her electrocardiogram (ECG) (Figure 1) revealed sinus rhythm with complete AV dissociation and a paced ventricular rhythm at 50 bpm. QT interval was markedly prolonged at 604 msec with a QTc of 550 msec. JTc was also prolonged at 383 msec. She had biphasic T wave in the anterior chest leads, which has been reported as a predictor of torsades de pointes in patients with AV block.[2]

Pacemaker interrogation revealed a pacing threshold of 0.75 V at 0.4 msec, an R wave of 8.1 mV, and a bipolar lead impedance of 550 ohm. There were 8 high-ventricular-rate episodes. The longest episode lasted 24 seconds, which correlated with the current presentation (Figure 2A). The other episodes ranged from 2 to 8 seconds and one of them correlated with her syncope the previous day. These episodes were consistent with polymorphic VT.

The patient initially received 1 unit of packed red blood cells and potassium and magnesium replacement; however, she continued to have runs of nonsustained polymorphic VT on the monitor (Figure 2B). She then had cardiac catheterization, which revealed mild stenosis (40%) in the proximal left anterior descending artery with minor plaques elsewhere; her left ventriculogram revealed an ejection

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KEY TEACHING POINTS

- Bradycardia is an important cause of QT prolongation and polymorphic ventricular tachycardia.
- A VDD pacemaker has a single ventricular lead with an atrial sensor; it senses the atrium and paces the ventricle to maintain atrioventricular synchrony. A VVI pacemaker has a single ventricular lead with no atrial sensor; it only paces the ventricle.
- It is important to program pacemakers to a lower pacing rate of 70–90 beats per minute, particularly in the first 3 months after a change in pacing mode that could create sudden bradycardia.

fraction of 57%. Echocardiogram revealed normal left ventricular size and systolic function with no significant valvular disease.

Her original pacemaker was inserted 7 years ago for complete heart block. That pacemaker had a VDD lead and was programmed to VDD mode with a base rate of 50 bpm and an upper tracking rate of 120 bpm. She was paced in the ventricle 99% of the time. After device extraction, a single-chamber pacemaker was inserted on the opposite side and programmed to VVIR mode with a base rate of 50 bpm and an upper rate of 110 bpm. The new pacemaker was programmed to similar rate cutoffs as her old device. It is not clear why the decision was made to implant a VVI system; however, the current guidelines recommend the use of a dual-chamber system if AV synchrony is desired.[3]

As bradycardia is a known risk factor of long QT, her lower pacing rate was increased from 50 to 80 bpm, which reduced her QTc from 550 to 485 msec and her JTc from 383 to 358 msec (Figure 3C) and she had no further polymorphic VT. She was observed for another 72 hours with no recurrence of arrhythmia and she was started on metoprolol 25 mg twice daily prior to discharge. She had no recurrence of her polymorphic VT after 2 months of follow-up.

Discussion

Long QT syndrome is a disorder of myocardial repolarization that manifests on surface ECG by an abnormal prolongation of the QT interval. It can be congenital owing to mutations in the genes encoding ion channels (Na^+ or K^+) or acquired owing to drugs or metabolic disorders.[1] Polymorphic VT is a form of VT that is characterized by continuous variation of QRS morphology, axis, or both. When polymorphic VT occurs in the setting of long QT it has a characteristic appearance with alterations in QRS axis of more than 180 degrees, giving the appearance of twisting around a point, or torsades de pointes.[4]

We excluded causes of acquired long QT in our patient in a stepwise approach. Although pantoprazole could cause QT prolongation,[5] she had been on it for a long time, and she was not on any other QT-prolonging drugs. Potassium and magnesium supplementation did not stop the polymorphic VT. Coronary angiogram excluded flow-limiting coronary artery disease, making ischemia an unlikely cause of presentation.

Bradycardia causes QT prolongation and torsades de pointes owing to prolonged ventricular repolarization. The QT interval is inversely proportional to the pacing

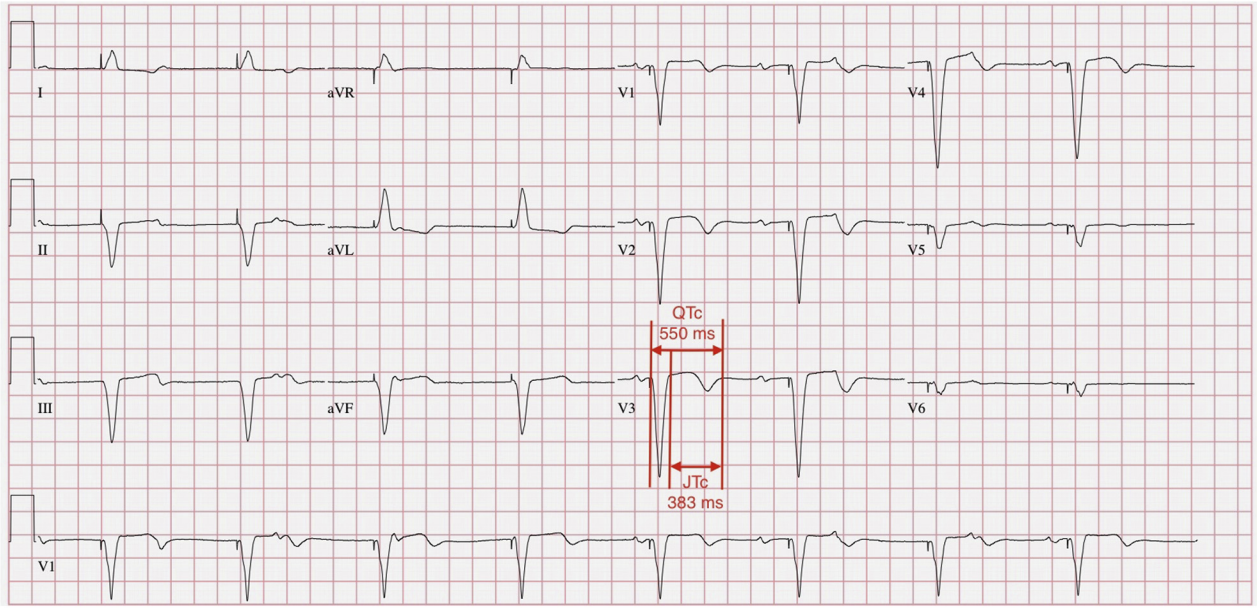

Figure 1 Electrocardiogram on presentation. Sinus rhythm with complete atrioventricular (AV) dissociation and ventricular paced complexes. Ventricular rate 50 beats per minute. QTc 550 msec. JTc 383 msec. Biphasic T wave is a high-risk feature in patients with acquired AV block.

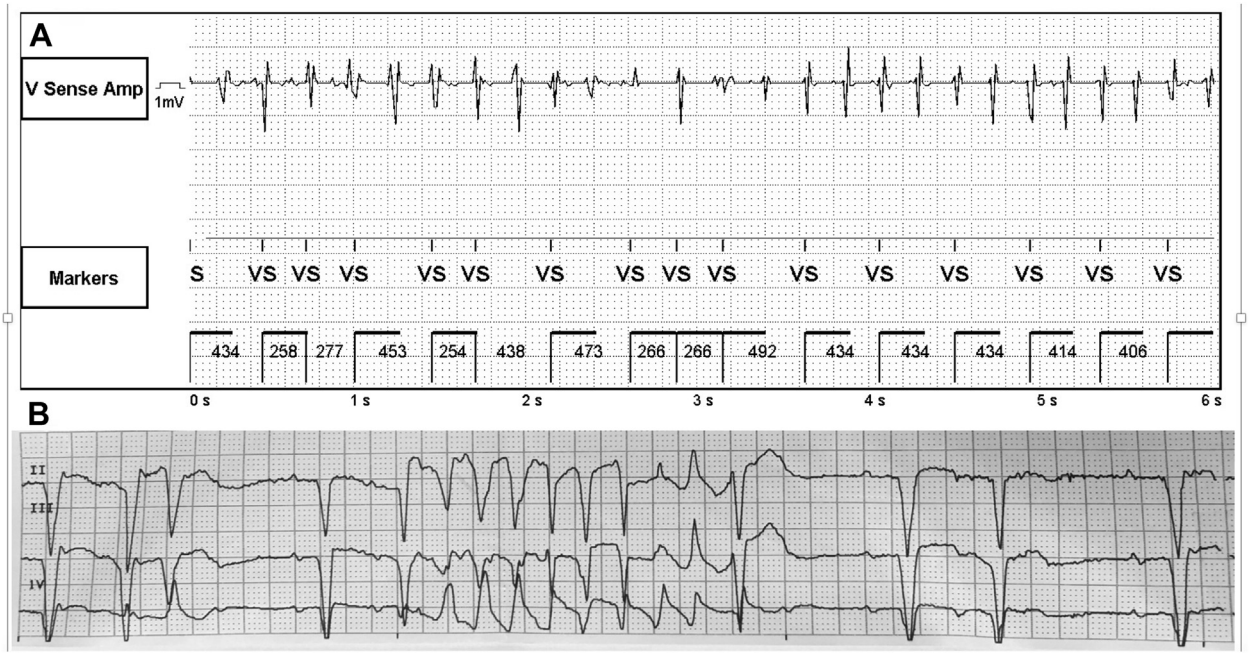

Figure 2 **A:** Polymorphic ventricular tachycardia on pacemaker interrogation (note occasional ventricular under-sensing). **B:** Polymorphic ventricular tachycardia on telemetry.

rate (ie, the slower the pacing rate, the longer the QT interval). In a study of 14 patients with complete heart block, Kurita et al documented that relationship, proving that patients who develop torsades de pointes have a bradycardia-sensitive repolarization abnormality; the critical heart rate that induced abnormally long QT intervals in patients with torsades de pointes was $\leq$60 bpm.[6] This heterogeneity of repolarization response supports the concept of impaired repolarization reserve; that is, a genetically determined subclinical repolarization abnormality becomes manifest when the patient is challenged by other stressors,[7] in our case bradycardia. The potential adrenergic dependence of torsades de pointes in our patient was the rationale for adding the beta blocker.

A lower rate limit of 50 bpm in VVI mode is not equivalent to a lower rate limit of 50 bpm in VDD mode. VDD mode tracks atrial activity and thus would have a near-normal heart rate distribution. This can be observed by comparing the ECGs (Figure 3) and the heart rate histograms (Supplemental Figure 1, available online) in the 2 pacing modes.

Palanca et al reported a similar finding in 3 cases with VDD pacemakers that had polymorphic VT owing to atrial under-sensing: the first patient had her pacemaker upgraded to a dual-chamber system, the second patient received a dual-chamber defibrillator, and the third patient had his base rate increased to 90 bpm.[8]

With the scarcity of evidence on the effect of pacing mode on development of polymorphic VT, we believe the literature on AV nodal ablation offers the best explanation for our case. The complete AV dissociation in VVI mode combined with the lower pacing rate of 50 bpm created a "functional AV nodal ablation." Torsades de pointes after AV nodal ablation has been well described.[9,10] An initial pacing rate of 90 bpm following AV nodal ablation, which is reduced over the following months, was found to be protective from polymorphic VT and ventricular fibrillation.[11,12] This approach is supported by the current guidelines.[13,14]

Although our patient did not have AV nodal ablation, the sudden drop in heart rate combined with the AV dissociation mimicked what occurs post AV nodal ablation. The presence of low-normal potassium and magnesium levels might have exaggerated the QT prolongation caused by bradycardia. Correction of electrolytes, however, did not correct the problem and it was only after increasing the pacing rate that the polymorphic VT stopped.

Our case highlights the importance of pacemaker programming and the adverse events that might arise from overlooking different pacing modes. It also highlights the importance of increasing the lower pacing rate in patients with complete AV block, particularly in the first few months after changing pacing mode. This observation can probably be extended to patients with complete heart block who develop atrial fibrillation. If the automatic mode switch base rate is low, they could potentially be at similar risk of ventricular arrhythmias, which could contribute to the poorer prognosis in this subset of patients.

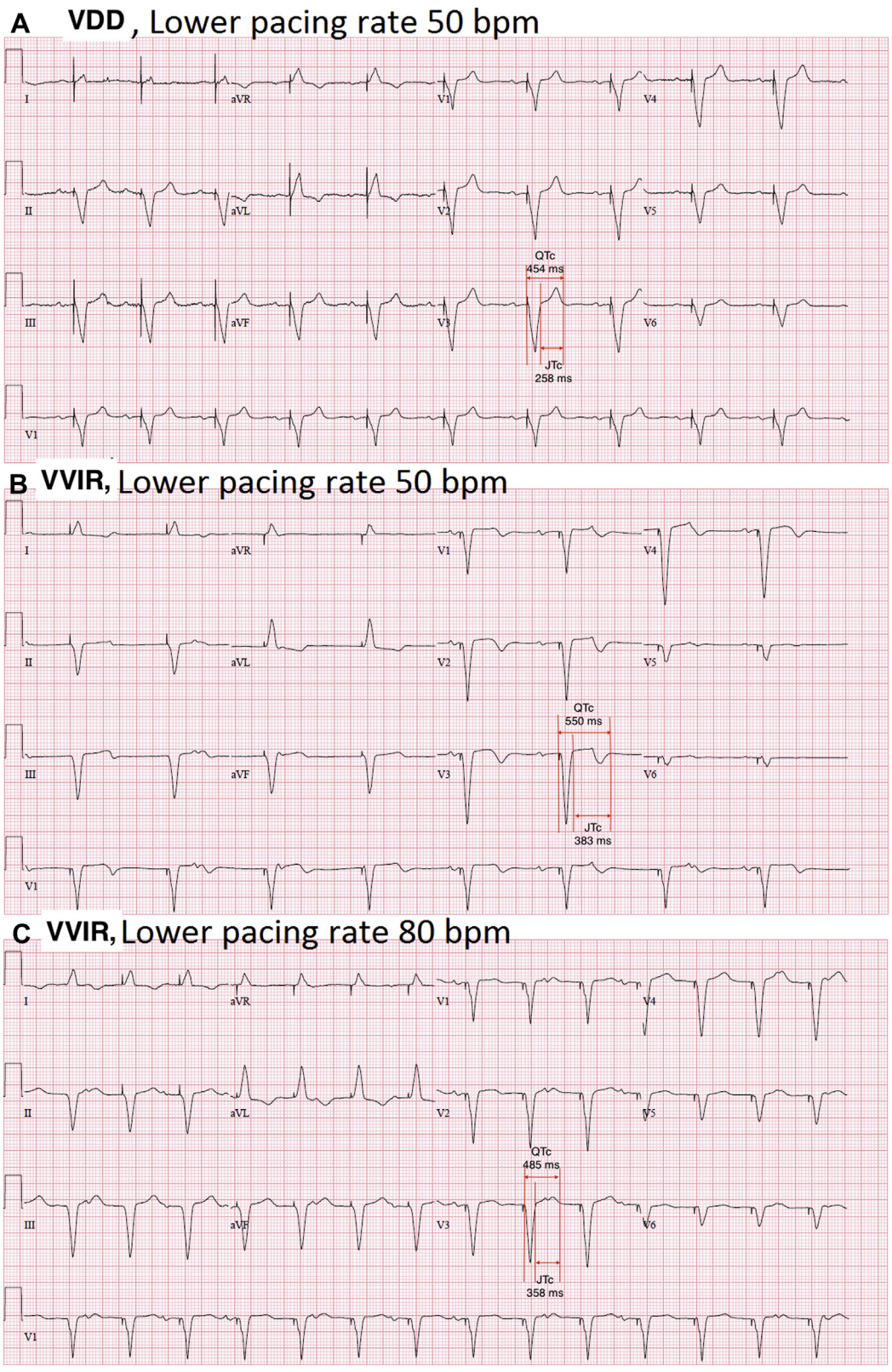

Figure 3 **A:** Electrocardiogram (ECG) prior to patient's VDD pacemaker extraction: Patient is paced in VDD mode with a lower pacing rate of 50 beats per minute (bpm). P waves are tracked by the pacemaker, resulting in a ventricular rate of 64 bpm. QTc 454 msec, JTc 258 msec. **B:** ECG on presentation: Patient is paced in VVIR mode with a lower pacing rate of 50 bpm. QTc 550 msec, JTc 383 msec. **C:** ECG prior to discharge: Patient is paced in VVIR mode with a lower pacing rate of 80 bpm. QTc 485 msec, JTc 358 msec.

Appendix

Supplementary data

Supplementary data associated with this article can be found in the online version at http://dx.doi.org/10.1016/j.hrcr.2017.02.003.

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