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Electrical organization of torsades de pointes into monomorphic ventricular tachycardia

Pishoy Gouda , Soori Sivakumaran , and Janek Senaratne *

Division of Cardiology, Faculty of Medicine and Dentistry, University of Alberta, 8440 112 Street NW, Edmonton, AB, T6G 2B7, Canada

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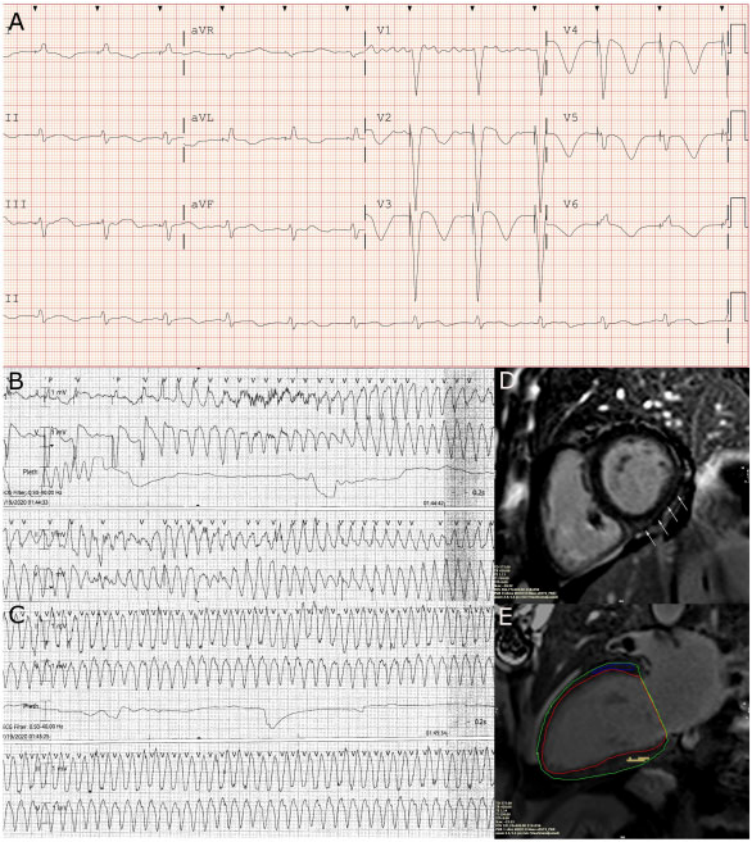
A 55-year-old male with a history of atrial fibrillation and atrioventricular node ablation with permanent pacemaker implantation, presented with atypical chest pain and a positive troponin I (1.15 μg/L; normal range: 0.00–0.04 μg/L). His admission electrocardiogram (ECG) demonstrated a ventricularly paced rhythm with underlying atrial fibrillation (Supplementary material online, *Image S1*). His home



* Corresponding author. Tel: 780-463-2184, Fax: 780-450-8359, Email: janeks@ualberta.ca
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medications included: bisoprolol 5 mg daily, furosemide 40 mg daily, amitriptyline 150 mg daily, zopiclone 15 mg daily, pantoprazole 40 mg daily, and long-acting oxycodone 60 mg three times a day. On presentation, he was treated as a non-ST-segment elevation myocardial infarction; however, coronary angiography demonstrated non-obstructive coronary disease (Supplementary material online, *Videos S1* and *S2*). His echocardiogram demonstrated reduced (35%) left ventricular ejection fraction (Supplementary material online, *Videos S3* and *S4*). Overnight, the patient became increasingly agitated and received a dose of 5 mg of haloperidol. He subsequently had an ECG that demonstrated a paced rhythm at 70 b.p.m. and a QTc of 648 ms (*Panel A*). Shortly afterwards, he experienced a torsades de pointes polymorphic ventricular tachycardia (VT) arrest (*Panel B*) that organized into a regular monomorphic VT (*Panel C*). He was electrically defibrillated and returned to a paced rhythm. A cardiac magnetic resonance imaging study demonstrated diffuse increase in T2 signal (*Panel D*) and non-ischaemic enhancement of the inferior wall and inferolateral wall compatible with myocarditis (*Panel E*). He underwent an implantable cardioverter-defibrillator implantation and was discharged uneventfully.

The patient's use of amitriptyline, pantoprazole, and in-hospital administration of haloperidol likely precipitated his long QT and subsequent torsades de pointes. While early afterdepolarization and triggered activity commonly induce torsades de pointes, this rhythm is generally non-sustained or degenerates into ventricular fibrillation. However, in our case, the rhythm organized into a regular monomorphic VT suggestive of underlying myocardial substrate (in this case myocarditis) that resulted in a re-entrant circuit. While extremely rare, in cases of organizing polymorphic VT, investigations to assess for a focal myocardial arrhythmia substrate may be warranted.

Supplementary material

Supplementary material is available at *European Heart Journal - Case Reports* online.

Consent: The authors confirm that written consent for submission and publication of this case report including image(s) and associated text has been obtained from the patient in line with COPE guidance.