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Published: 2026.XX.XXTwo Cases of Refractory Torsades de Pointes:
Short-Coupled Variant and Acquired Long QT
Syndrome After Myocardial InfarctionAuthors' Contribution:
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Statistical Analysis C
Data Interpretation D
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Case series

Patients:

Female, 53-year-old • Female, 79-year-old

Final Diagnosis:

Torsades de pointes

Symptoms:

Loss of consciousness

Clinical Procedure:

Overdrive ventricular pacing

Specialty:

Cardiology

Objective:

Rare disease

Background:

Torsades de pointes (TdP) is a life-threatening form of ventricular tachycardia characterized by QRS complexes twisting around the isoelectric line on electrocardiography and can be associated with a prolonged QT interval. Management of these combined conditions can be challenging. This report presents 2 cases of refractory torsades de pointes arising from distinct mechanisms—short-coupled variant TdP and acquired long QT syndrome—following myocardial infarction.

Case Reports:

Case 1 was a 53-year-old woman with a prior unexplained cardiac arrest who presented with a TdP electrical storm in the context of short-coupled premature ventricular contractions (coupling interval 200 ms) and a corrected QT interval (QTc) within normal limits (459 ms), consistent with short-coupled variant TdP. Sequential pharmacological therapy and overdrive ventricular pacing escalated to 150 beats per minute both failed; arrhythmia cessation was temporally associated with subsequent intravenous lidocaine, an agent historically among the least effective for this entity. A dual-chamber implantable cardioverter-defibrillator was implanted. Case 2 was a 79-year-old woman who developed extreme QTc prolongation (708 ms) 4 days after primary percutaneous coronary intervention for anterior ST-elevation myocardial infarction, sustaining more than 50 daily TdP episodes refractory to pharmacological therapy; overdrive pacing at 100 beats per minute abolished the arrhythmia, with the QTc normalizing to 461 ms at 3-month follow-up.

Conclusions:

These cases illustrate how distinguishing the likely electrophysiological mechanism of refractory TdP may help guide rescue therapy. Overdrive pacing was effective in the acquired long QT case and ineffective in the short-coupled case, in which the arrhythmia was controlled only after lidocaine was added—an unusual observation in this setting.

Keywords:

Death, Sudden, Cardiac • Long QT Syndrome • Torsades de Pointes • Ventricular Fibrillation • Ventricular Premature Complexes

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Introduction

Torsades de pointes (TdP), first described by Dessertenne in 1966, is a potentially fatal polymorphic ventricular tachycardia characterized by progressive twisting of the QRS complex axis around the isoelectric baseline [1]. Although TdP is most commonly associated with congenital or acquired QT prolongation, a mechanistically distinct entity—the short-coupled variant of TdP (scTdP)—occurs in structurally normal hearts with a normal or near-normal corrected QT interval (QTc), initiated by premature ventricular contractions (PVCs) with coupling intervals of less than 300 ms in the absence of a preceding bradycardic pause [2].

Since its characterization by Leenhardt et al in 1994, scTdP has been reported almost exclusively as isolated cases, and its prevalence remains undefined [2-4]. In contrast, pause-dependent TdP in the post-myocardial infarction (MI) setting occurs in approximately 1.8% of MI admissions, arising from heterogeneous repolarization recovery in the ischemically remodeled myocardium [5]. First-line management of refractory TdP includes intravenous magnesium, electrolyte correction, and heart rate acceleration via overdrive pacing [6]. However, mechanistically distinct forms of refractory TdP may respond differently to heart rate acceleration and pharmacological rescue, and delayed recognition of this distinction can complicate acute rhythm control.

The aim of this report is to describe 2 cases of refractory torsades de pointes with contrasting electrophysiological mechanisms—short-coupled variant TdP and acquired long QT syndrome after myocardial infarction—and to detail their acute management, including an atypical successful pharmacological response with lidocaine in the short-coupled case.

Case Reports

Case 1

A 53-year-old woman with hypertension, bronchial asthma, prior acute kidney injury, and a history of an unexplained out-of-hospital cardiac arrest 2 years earlier presented by ambulance following a further out-of-hospital cardiac arrest. She was a lifelong non-smoker with no history of alcohol or substance use, and there was no family history of sudden cardiac death. Emergency personnel documented ventricular fibrillation (VF) terminated by a single shock from an automated external defibrillator (**Figure 1A**), achieving return of spontaneous circulation.

On arrival, vital signs were stable. Electrocardiography (ECG) demonstrated atrial fibrillation with frequent PVCs, reverting

spontaneously to sinus rhythm; the patient was admitted to the intensive care unit (**Figure 1B, 1C**). On day 2, repeat ECG showed T-wave inversions with a QTc within normal limits (459 ms). Later that day, frequent short-coupled PVCs with an R-on-T morphology triggered polymorphic ventricular tachycardia (VT) degenerating into VF (**Figure 2A**). The initiating PVCs had a measured coupling interval of 200 ms, well below the 300 ms threshold, and arose without a preceding compensatory pause or short-long-short sequence; together with the normal QTc, these features formed the electrocardiographic basis for the short-coupled classification. Multiple direct-current cardioversions were delivered, but TdP and VF recurred, establishing a TdP electrical storm. All corrected QT interval (QTc) values were measured manually using the Bazett formula after restoration of sinus rhythm—rather than during the presenting atrial fibrillation—with the end of the T wave assessed in the lead showing the most prominent and clearly defined T wave.

A temporary transvenous pacemaker was inserted via the left femoral vein for overdrive pacing. At a ventricular pacing rate of 130 beats per minute, TdP persisted. Escalation to 140 beats per minute achieved only transient and incomplete suppression (**Figure 2B**). On day 3, recurrent TdP and VF were again documented. Intravenous magnesium sulfate (2 g bolus followed by infusion), esmolol, and intravenous verapamil were administered sequentially without efficacy. The pacing rate was further escalated to 150 beats per minute, which likewise failed to suppress the arrhythmia. A continuous intravenous lidocaine infusion was subsequently initiated, after which all further arrhythmia episodes were abolished.

Following stabilization, the patient was transferred to the general cardiac ward. Twenty-four-hour Holter monitoring on day 3 documented no PVCs or VT. Transthoracic echocardiography (TTE) on day 3 demonstrated mild left ventricular (LV) systolic dysfunction with a regional wall motion abnormality (RWMA) at the posterolateral LV base. Coronary angiography demonstrated no significant coronary artery stenosis. A subsequent 24-hour Holter recording on day 5 documented a 2% PVC burden without symptoms, and repeat TTE on day 10 confirmed complete normalization of LV systolic function with full resolution of the RWMA. A dual-chamber implantable cardioverter-defibrillator (ICD) was implanted on day 7, programmed with atrial demand pacing at 90 beats per minute (**Figure 2C**). The patient was discharged on oral carvedilol with potassium and magnesium supplementation. Genetic testing for known scTdP-associated channelopathy variants was not available at our institution at the time of treatment; outpatient referral for genetic evaluation was recommended. The working diagnosis was a TdP electrical storm secondary to short-coupled variant TdP. Cardiac magnetic resonance imaging and an electrophysiology study were not performed during the admission.

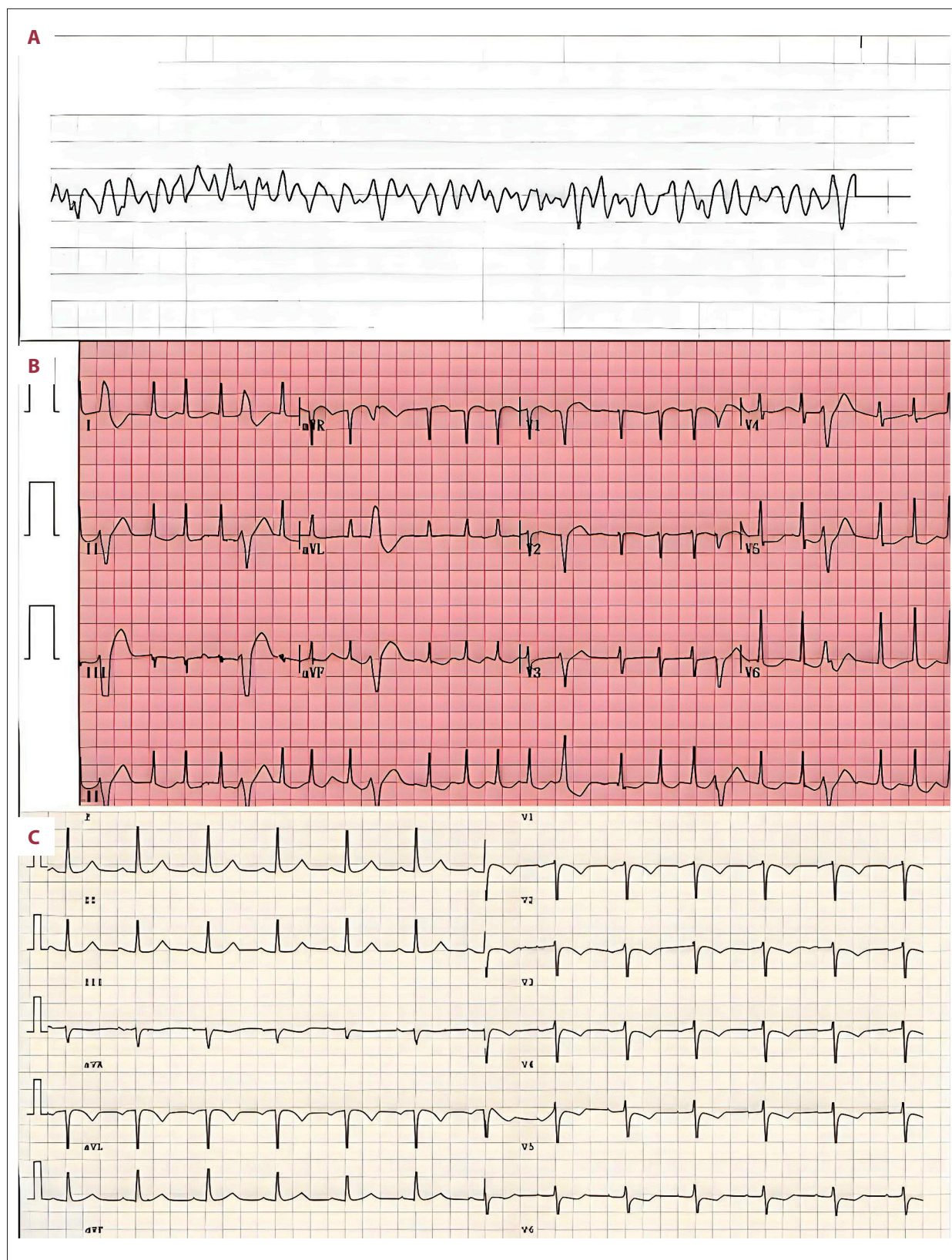


Figure 1. (A) Documented out-of-hospital ventricular fibrillation. (B) Atrial fibrillation with frequent premature ventricular contractions on arrival. (C) Reverted sinus rhythm with T-wave inversions and a corrected QT interval within normal limits (459 ms).

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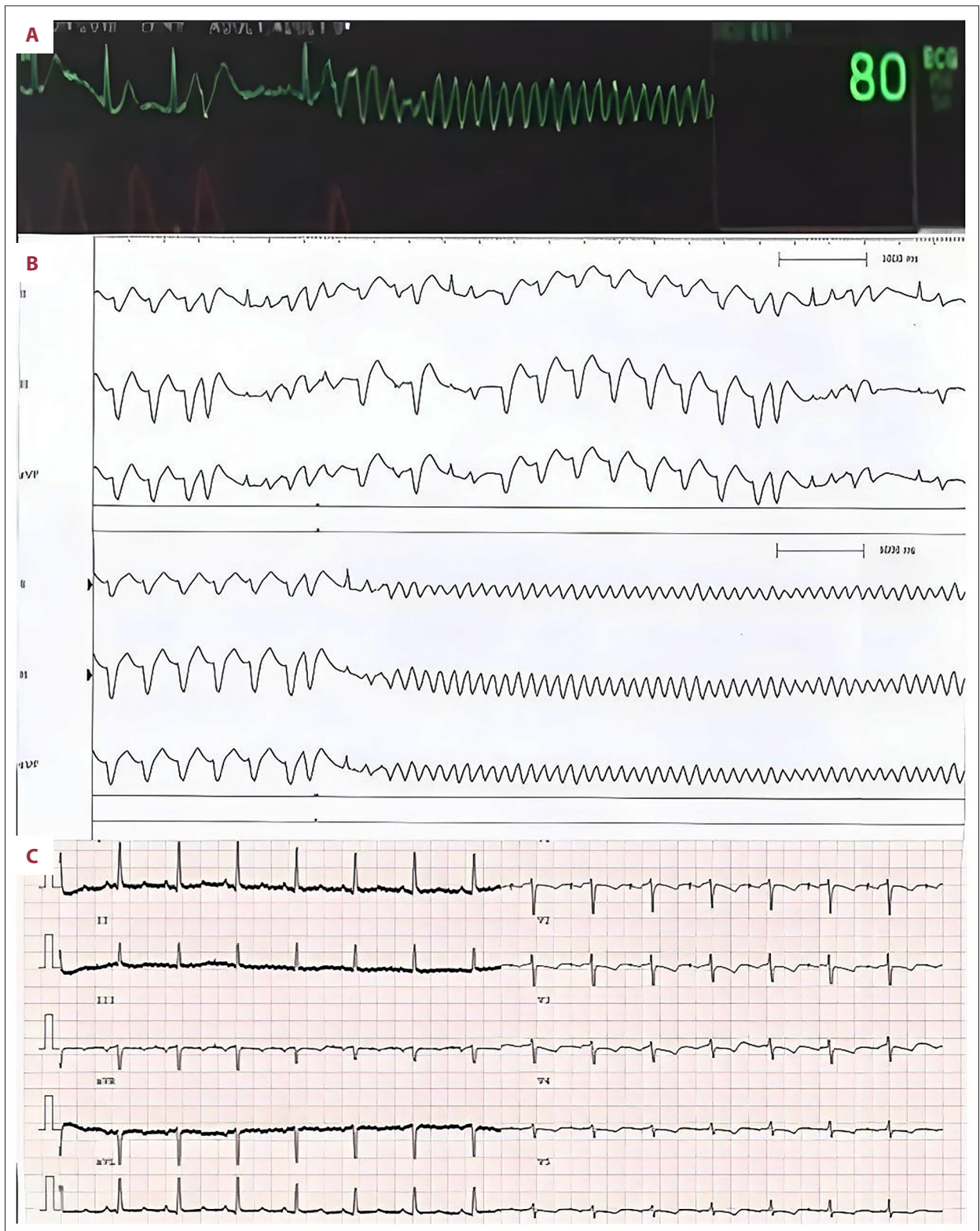


Figure 2. (A) Short-coupled premature ventricular contraction (coupling interval 200 ms) triggering torsades de pointes (TdP). (B) TdP persisting despite ventricular pacing at 140 beats per minute. (C) Atrial demand pacing rhythm at 90 beats per minute following dual-chamber implantable cardioverter-defibrillator implantation, recorded after intravenous lidocaine had successfully controlled the arrhythmia.

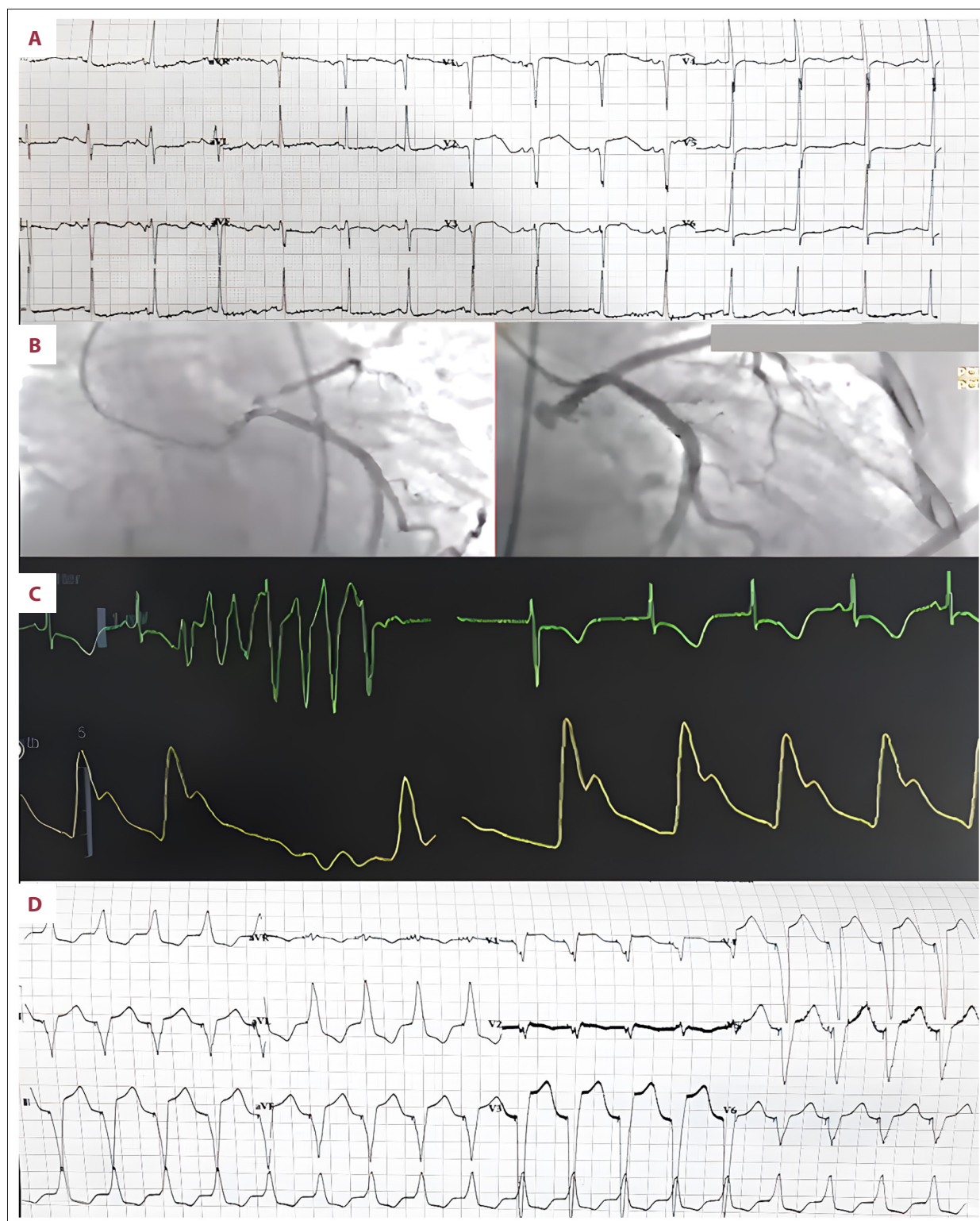


Figure 3. (A) Electrocardiogram at presentation demonstrating anterior ST-elevation myocardial infarction. (B) Coronary angiography demonstrating sub-total occlusion of the proximal left anterior descending artery with Thrombolysis in Myocardial Infarction (TIMI) grade II flow (left) and successful TIMI grade III flow restoration following provisional stenting (right). (C) R-on-T phenomenon triggering torsades de pointes (TdP), with the corrected QT interval measured at 708 ms at this time. (D) Ventricular overdrive pacing at 100 beats per minute successfully abolishing TdP.

Case 2

A 79-year-old woman was admitted to the coronary care unit with an anterior ST-elevation myocardial infarction (STEMI) complicated by cardiogenic shock and acute pulmonary edema; the admission ECG demonstrated an already-prolonged QTc of 518 ms (**Figure 3A**). She was a non-smoker with no history of alcohol or substance use. Emergency coronary angiography demonstrated a sub-total occlusion of the proximal left anterior descending (LAD) artery with Thrombolysis in Myocardial Infarction (TIMI) grade II flow (**Figure 3B**). Primary percutaneous coronary intervention (PPCI) was performed using the provisional technique under angiographic guidance, successfully restoring TIMI grade III flow (**Figure 3B**). Transthoracic echocardiography after primary percutaneous coronary intervention demonstrated severely impaired left ventricular systolic function (ejection fraction 33%), which improved to 48% before discharge.

Serial ECGs in the days following PPCI demonstrated progressive development of reperfusion T-waves and worsening QTc prolongation, reaching a maximum of 708 ms (**Figure 3C**). Serum electrolytes remained within normal limits throughout. The patient's medications during the peri-infarction period comprised ticagrelor, aspirin, atorvastatin, an intravenous beta-blocker, guideline-directed medical therapy for heart failure, and a loop diuretic; no agent with recognized QT-prolonging potential was administered. The intravenous beta-blocker and lidocaine were selected specifically because the presenting QTc was already prolonged. On this basis, acquired long QT related to the acute infarction and reperfusion was favored as the most likely mechanism, although the available evidence cannot fully exclude other unrecognized contributors. Beginning on the fourth day after PPCI, the patient deteriorated acutely, with escalating PVC frequency precipitating recurrent TdP requiring repeated defibrillation, with more than 50 episodes within a single 24-hour period (**Figure 3C**). Serum electrolytes were confirmed normal at the onset of torsades de pointes and were subsequently maintained at the upper limit of normal until the arrhythmia resolved. Given that the common acquired causes—electrolyte imbalance and QT-prolonging drug exposure—were absent, and that the deep, prolonged reperfusion T-wave changes were localized to the left anterior descending (anterior) territory with onset following reperfusion, an infarction- and reperfusion-related acquired long QT mechanism was favored. Nonetheless, this mechanism cannot be established with certainty: congenital or genetic long QT syndrome (no genetic testing was performed), thyroid dysfunction (thyroid function was not assessed), and neurological contributors (which the electrocardiogram can suggest but not exclude) were not formally ruled out.

The patient was sedated, mechanically ventilated, and treated with intravenous potassium, intravenous magnesium, an

intravenous beta-blocker, and a continuous intravenous lidocaine infusion. Despite these measures, the QTc failed to shorten and TdP persisted. A temporary transvenous pacemaker was inserted and ventricular overdrive pacing at 100 beats per minute was initiated; TdP resolved completely and promptly (**Figure 3D**). Serial monitoring over 7 days, including deliberate pacemaker deactivation, confirmed sustained suppression. The patient was extubated and discharged on oral beta-blocker therapy.

At 3-month follow-up, no arrhythmia recurrence was reported, and repeat ECG confirmed normalization of the QTc interval to 461 ms. No treatment complications were observed in either patient.

Discussion

These 2 cases illustrate that TdP can arise through opposing electrophysiological mechanisms that may respond differently to rescue therapy. In short-coupled variant TdP, episodes are initiated by short-coupled PVCs that occur independently of bradycardia or QTc prolongation [2], rendering the standard pause-elimination rationale for overdrive pacing insufficient as the sole mechanism. Ventricular overdrive pacing can paradoxically worsen scTdP by increasing repolarization heterogeneity [7]. Chiladakis et al documented that ventricular pacing at 90 beats per minute not only failed to suppress TdP but appeared to exacerbate it, whereas atrial pacing—which preserves the normal ventricular activation sequence and limits repolarization dispersion—was associated with sustained suppression [7]. In Case 1, arrhythmia cessation was temporally associated with initiation of lidocaine after magnesium, esmolol, verapamil, and escalating ventricular pacing had failed to achieve evident control. Because these interventions were administered sequentially and without controls, delayed or combined effects of the preceding measures, or spontaneous fluctuation of the arrhythmia, cannot be excluded; the sequence therefore cannot establish lidocaine as the sole cause of suppression or demonstrate a specific substrate-stabilizing effect. Nonetheless, this response is notable: across pooled cases of short-coupled malignant ventricular arrhythmias, lidocaine achieved a success rate of only 4.2%, the lowest of any reported antiarrhythmic agent [4].

Catheter ablation targeting an identifiable short-coupled triggering premature ventricular contraction—typically arising from the Purkinje network—is a recognized option for recurrent, drug-refractory PVC-triggered polymorphic ventricular tachycardia or ventricular fibrillation, and the 2022 European Society of Cardiology guidelines state that it should be considered when episodes are triggered by a consistent PVC and are non-responsive to medical treatment or coronary revascularization (Class IIa) [8]. Ablation is most effective when a single, unifocal trigger can be mapped.

In Case 1, ablation was not pursued: the acute electrical storm was managed pharmacologically and with pacing, no electrophysiology study or trigger mapping was performed, and a defibrillator was implanted for secondary prevention.

In contrast, the post-MI setting generates heterogeneous action potential duration recovery, creating the substrate for early afterdepolarizations and the characteristic pause-dependent pattern of TdP initiation [6]. Halkin et al established the incidence of this form of TdP at approximately 1.8% of MI admissions, with maximal QT prolongation and TdP occurring 3 to 11 days after infarction [5]. In Case 2, QTc prolongation peaked on day 4 after PPCI, within this window. Overdrive pacing suppresses this mechanism directly, and pacing at 100 beats per minute achieved immediate and complete abolition of TdP, with durable suppression confirmed on pacemaker withdrawal.

Isoproterenol, which accelerates heart rate without increasing repolarization dispersion, is an established alternative in scTdP electrical storms [7] but was not available in Case 1 and was contraindicated in Case 2 given the early post-infarction setting. The transient LV systolic dysfunction in Case 1, which resolved completely with no obstructive coronary disease identified, likely reflected reversible dysfunction secondary to hemodynamic compromise during repeated cardiac arrests. The principal limitations of this report are the absence of genetic confirmation in Case 1, where testing was unavailable, and the inherent constraints of drawing mechanistic inferences from 2 uncontrolled cases. In Case 1, cardiac magnetic resonance imaging and an electrophysiology study were not performed, further limiting mechanistic characterization.

Conclusions

These cases suggest that features such as QT behavior, PVC coupling interval, pause dependence, and clinical context can help guide the selection of rescue therapy in refractory TdP. Overdrive pacing was effective in the reported acquired long

QT case but ineffective in the reported short-coupled case, in which pharmacological rescue was required; lidocaine may therefore be considered when pacing does not control short-coupled TdP, recognizing that its efficacy here was an unusual observation. As a broader, guideline-based consideration, implantable cardioverter-defibrillator implantation may be appropriate for high-risk survivors on the basis of individual risk assessment [9], and genetic evaluation should be considered when an inherited channelopathy is suspected.

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Patient Permission/Consent

Written informed consent for publication of this case report and the accompanying images was obtained from both patients.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

References:

- Dessertenne F. La tachycardie ventriculaire à deux foyers opposés variables. *Arch Mal Coeur Vaiss.* 1966;59:263-72
- Leenhardt A, Glaser E, Burguera M, et al. Short-coupled variant of torsade de pointes: A new electrocardiographic entity in the spectrum of idiopathic ventricular tachyarrhythmias. *Circulation.* 1994;89:206-15
- Chokr MO, Darrieux FC, Hardy CA, et al. Short-coupled variant of "torsades de pointes" and polymorphic ventricular tachycardia. *Arq Bras Cardiol.* 2014;102:e60-64
- Belhassen B, Tovia-Brodie O. Short-coupled idiopathic ventricular fibrillation: A literature review with extended follow-up. *JACC Clin Electrophysiol.* 2022;8:918-36
- Halkin A, Roth A, Lurie I, et al. Pause-dependent torsade de pointes following acute myocardial infarction: A variant of the acquired long QT syndrome. *J Am Coll Cardiol.* 2001;38:1168-74
- Drew BJ, Ackerman MJ, Funk M, et al. Prevention of torsade de pointes in hospital settings: a scientific statement from the American Heart Association and the American College of Cardiology Foundation. *Circulation.* 2010;121:1047-60
- Chiladakis JA, Spiroulas G, Koutsogiannis N, et al. Short-coupled variant of torsade de pointes as a cause of electrical storm and aborted sudden cardiac death: Insights into mechanism and treatment. *Hellenic J Cardiol.* 2008;49:360-64
- Zeppenfeld K, Tfelt-Hansen J, de Riva M, et al. 2022 ESC Guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death. *Eur Heart J.* 2022;43:3997-4126
- Priori SG, Wilde AA, Horie M, et al. HRS/EHRA/APHRS expert consensus statement on the diagnosis and management of patients with inherited primary arrhythmia syndromes. *Heart Rhythm.* 2013;10:1932-63