

[ CASE REPORT ]

## Torsade de Pointes Due to QT Prolongation after Pulmonary Vein Isolation for Persistent Atrial Fibrillation

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### Abstract:

We herein report a 60-year-old woman with long-standing persistent atrial fibrillation (AF) who developed QT prolongation and torsade de pointes (TdP) after pulmonary vein isolation (PVI). When electrical cardioversion was performed three months before PVI, prominent QT prolongation was not observed. QT prolongation emerged after PVI and was sustained until AF recurrence on the third day after ablation, and TdP disappeared along with AF recurrence. PVI affects the ganglionated plexi around the atrium, leading to modification of the intrinsic cardiac autonomic system. This case indicates that PVI has the potential risk of inducing lethal ventricular arrhythmias due to QT prolongation.

**Key words:** torsade de pointes, QT prolongation, pulmonary vein isolation

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### Introduction

Catheter ablation is a highly effective therapeutic strategy for atrial fibrillation (AF) (1, 2). Pulmonary vein isolation (PVI) is a cornerstone of AF ablation. The ablation line of PVI transects the sites where the major atrial ganglionated plexi (GP) exists. GP ablation carries a risk of increasing atrial and ventricular vulnerability to arrhythmias, and GP ablation induced QT prolongation after myocardial infarction in a canine model (3, 4, 5). QT prolongation and lethal ventricular arrhythmias related to PVI and antiarrhythmic drugs have also been reported (6).

We herein report a patient who developed QT prolongation and torsade de pointes (TdP) after PVI for persistent AF without an antiarrhythmic drug.

### Case Report

A 60-year-old woman with long-standing persistent AF was referred to our hospital for catheter ablation. She had no structural heart disease or inherited arrhythmias. Her prescribed medication included bisoprolol fumarate 1.25 mg

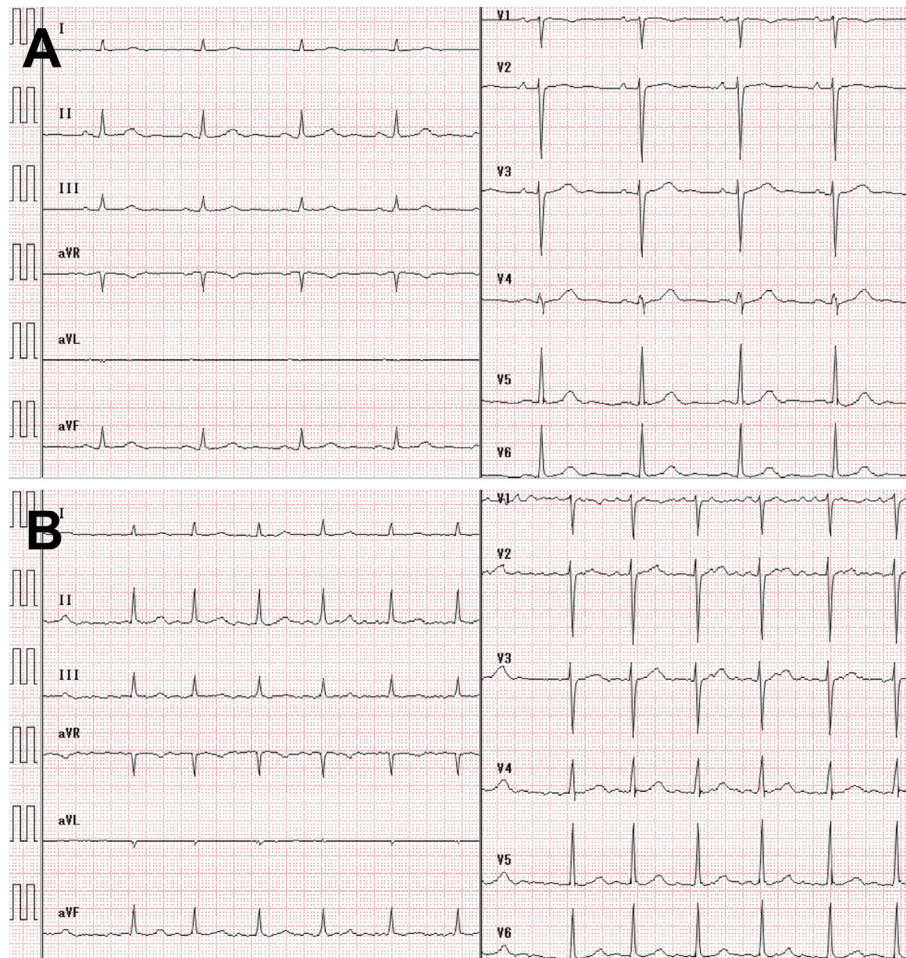
daily; edoxaban tosilate hydrate 30 mg daily; olmesartan medoxomil 20 mg daily; sodium valproate 200 mg twice daily; and ifenprodil tartrate 20 mg three times a day. Bisoprolol fumarate was discontinued two days before the ablation procedure. We performed electrical cardioversion three months before the ablation procedure, and prominent QT prolongation was not observed early after cardioversion. Intravenous injection of 150 mg thiopental sodium was performed before electrical cardioversion (Fig. 1A).

Recurrence of AF occurred, and a 12-lead electrocardiogram (ECG) showed no QT prolongation before ablation (Fig. 1B). Wide antral ipsilateral PVI using radiofrequency (RF) ablation was performed under the guidance of three-dimensional (3D) ultrasound geometries and 3D merged computed tomography (CT) using the CARTO 3 system (Biosense Webster, Diamond Bar, USA). RF ablation was performed under deep sedation with noninvasive positive pressure ventilation support. Deep sedation was initiated with 6 µg/kg/h dexmedetomidine for 10 min and fentanyl citrate 0.1 mg. Subsequently, it was maintained with 0.6 µg/kg/h dexmedetomidine. The total dexmedetomidine dose was 150 µg. First-pass isolation was achieved in both PVs, and electrical cardioversion was performed to restore sinus

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**Figure 1.** (A) A 12-lead electrocardiogram acquired soon after electrical cardioversion 3 months before pulmonary vein isolation. The HR was 54 bpm. The raw QT interval was 487 ms, and the Fridericia formula [ $QT_c = QT / (RR^{1/3})$ ]-corrected  $QT_c$  was 470 ms. (B) A 12-lead electrocardiogram showing atrial fibrillation before catheter ablation. The raw QT was 427 ms, and the Fridericia-corrected  $QT_c$  was 449 ms. The raw QT was measured using the tangent method as an average of six or more beats.

rhythm.

We performed right PVI first to avoid the vagal reflex, followed by left PVI (Fig. 2). Intravenous injection of 50 mg thiopental sodium was performed before electrical cardioversion. The bidirectional block was confirmed, and a dissociated potential was observed in both PVs. No complications were observed during the procedure, and the patient returned to the general ward in sinus rhythm. The total procedure time was 104 minutes.

Seventeen hours after the procedure, TdP suddenly occurred and spontaneously terminated. A 12-lead ECG revealed prominent QT prolongation. Laboratory data showed a slightly low serum magnesium (Mg) level of 1.6 mg/dL. Creatine phosphokinase levels were within the normal limits (85 IU/L). T wave inversions were found in I, aVL, and V2, but there were no obvious local wall motion abnormalities on bedside echocardiography. QT prolongation persisted, and TdP was not suppressed despite normalization of the serum Mg level using intravenous administration of Mg sulfate

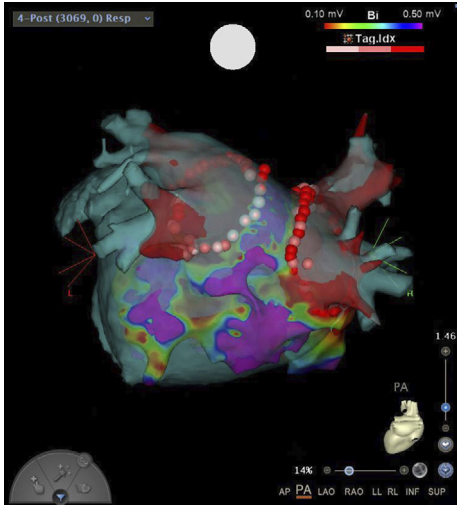
hydrate (Fig. 3). TdP appeared occasionally but resolved spontaneously and did not progress to ventricular fibrillation. TdP disappeared together with AF recurrence three days after ablation. QT prolongation and negative T waves were also improved (Fig. 4).

A genetic test using the TruSight Cardio sequencing panel (Illumina, San Diego, USA) detected no known gene mutations related to long QT syndrome. On the sixth day after the procedure, coronary angiography showed no obstruction or stenosis, and left ventriculography revealed no local wall motion abnormalities. She did not take any antiarrhythmic drugs before or after ablation.

## Discussion

The QT interval is affected by various factors, including the heart rate, electrolyte disorders, autonomic nervous system, and anesthesia. In this case, the QT interval was prolonged after PVI, and the prolongation was sustained despite

the normalization of electrolyte disorders. Exaggerated QT prolongation after cardioversion has been reported (7); however, in the present case, QT prolongation was not observed when electrical cardioversion was performed three months before the ablation. Her prescribed medications did not



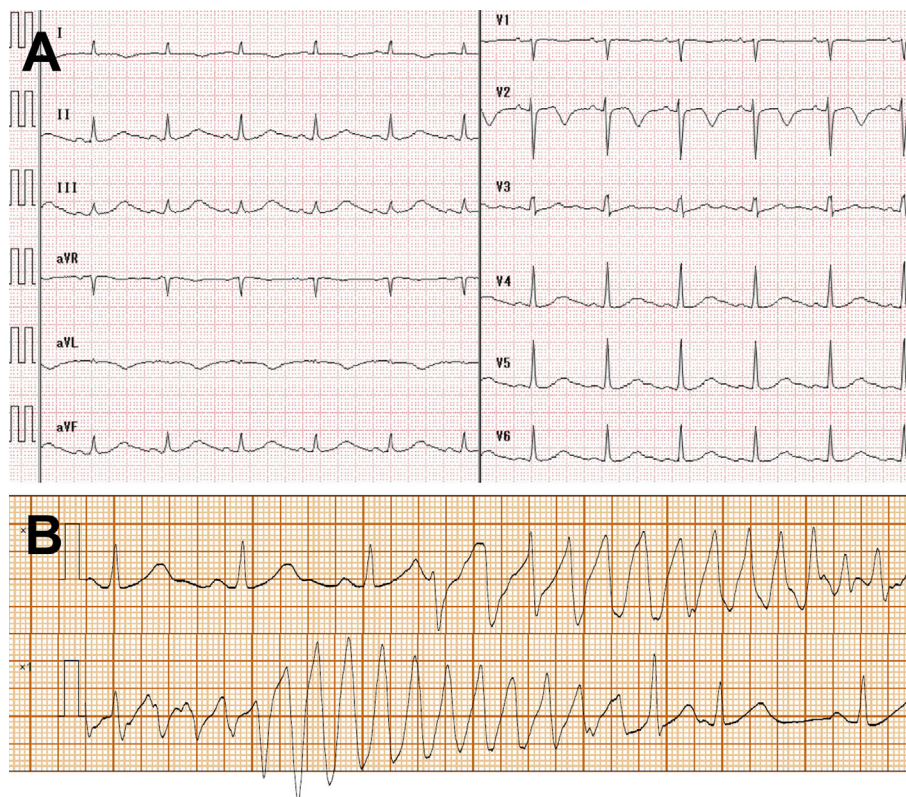
**Figure 2.** Three-dimensional computed tomography merged voltage map after pulmonary vein isolation. Each tag shows an ablation point.

change after the ablation procedures. These observations suggest the involvement of the PVI procedure in QT prolongation.

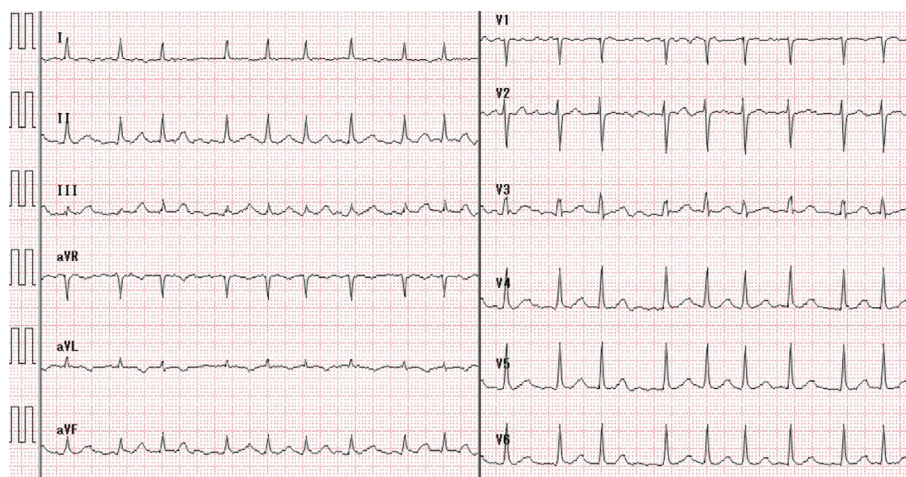
The ablation line of PVI for AF transects the sites where the major atrial GP exists; therefore, PVI inadvertently modifies the intrinsic cardiac autonomic nervous system, which is a determinant of the QT interval (8). In animal models, GP ablation is associated with a prolonged QT interval and increased atrial/ventricular vulnerability to arrhythmias (3, 4). QT prolongation has also been observed after PVI, especially in the acute phase, in humans (9). Although TdP associated with QT prolongation after PVI with antiarrhythmic drugs has been reported (6), no antiarrhythmic drugs were used in the present case. Approximately 30% of acquired long QT syndrome cases have a known clinical long QT syndrome gene mutation (10), but no relevant genetic background was detected in this case; nevertheless, discontinuation of the beta-blocker two days before the procedure may have been involved in QT prolongation after the ablation procedure.

This case indicates the potential risk of lethal arrhythmia induced by QT prolongation due to PVI, even in patients without a known genetic background of long QT syndrome or antiarrhythmic drug prescription.

The authors state that they have no Conflict of Interest (COI).



**Figure 3.** QT prolongation was observed two days after pulmonary vein isolation. The HR was 71 bpm. The raw QT was 540 ms, and the Fridericia-corrected QTc was 549 ms. T wave inversions were found in I, aVL, and V2. Torsade de pointes (TdP) occurred despite the normalization of serum Mg levels. TdP disappeared with the recurrence of atrial fibrillation.



**Figure 4.** A 12-lead electrocardiogram was acquired on the third day after the procedure, showing atrial fibrillation. The raw QT was 362 ms, and the Fridericia-corrected QTc was 440 ms. QT prolongation and negative T waves were improved.

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