CASE REPORT

QT Prolongation and T-Wave Alternans during Catheter Ablation of Atrial Fibrillation: A Case Report

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We report a case of a 63-year-old woman who developed profound QT prolongation, T-wave alternans, and spontaneous ventricular fibrillation during catheter ablation of atrial fibrillation. A thorough search into the possible mechanisms identified the use of sevoflurane, an inhalational gas anesthetic as the culprit. The patient was converted to propofol anesthesia and her QT interval normalized promptly.

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The effect of medications on the ventricular repolarization time and the clinical consequence of a prolonged QT interval by surface electrocardiogram (ECG) have been extensively studied.^{1,2} We report a case of a 63-year-old woman with paroxysmal atrial fibrillation (AF) who developed significant QT prolongation and spontaneous ventricular fibrillation during catheter ablation of AF. After successful defibrillation, a thorough search into the possible mechanisms leading to this complication identified the use of sevoflurane, an inhalational gas anesthetic as the culprit. The patient was converted to propofol anesthesia and her QT interval normalized promptly.

CASE REPORT

A 63-year-old Caucasian woman with a history of paroxysmal AF maintained on dofetilide for 2 years was admitted for AF ablation because of recent increases in symptoms. Her cardiac workup had included an echocardiogram (Vivid 7, General Electric, Fairfield, CT, USA) showing normal left atrial size and normal left ventricular size and systolic function, a nuclear stress test showing normal myocardial perfusion, and a left heart catheterization which revealed only mild luminal

irregularities in the left anterior descending artery and a 30%–40% stenosis in the right coronary artery.

The morning of the procedure, the patient's ECG showed normal sinus rhythm with a corrected QT interval (QTc) of 477 ms. In the electrophysiology laboratory, she was placed under general anesthesia with sevoflurane and was noted to have frequent premature ventricular contractions (PVCs) and prolongation of the QT interval. The patient underwent a double transeptal catheterization of the left atrium for planned pulmonary vein isolation. With only two transeptal sheaths in the left atrium and no other catheters inside the heart, the patient spontaneously went into ventricular fibrillation and was promptly defibrillated with a 200 J biphasic shock. She continued to have frequent PVC's and her QT prolongation worsened reaching close to 850 ms with evidence of T-wave alternans (Fig. 1). Immediately, a transthoracic echocardiogram (Vivid e, General Electric) was obtained which demonstrated normal ejection fraction, no significant wall motion abnormalities, and no pericardial effusion. Arterial blood gas showed normal oxygenation. Plasma electrolytes levels were normal. Ischemic etiology was suspected and a coronary angiogram was performed revealing no critical occlusions.

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Figure 1. Surface ECG tracings demonstrating the progressive QTc prolongation in our patient over the course of the AF ablation with the use of sevoflurane, leading to a ventricular fibrillation arrest. Note the presence of significant QTc prolongation with T-wave alternans in panel (e). Also note the progressive shortening of the QTc interval back to baseline levels after the discontinuation of sevoflurane.

* Corrected QT interval (QTc) calculated using Bazett's formula

Figure 2. Plot demonstrating the rise and fall of the QT (blue) and QTc (red) interval as a function of the use of sevoflurane. Note that the QT interval increased by a factor of 1.73 during exposure to sevoflurane. QTc interval was calculated using Bazett's formula: $QTc = QT/(RR)^{1/2}$.

In the absence of other etiologies, the possibility of an interaction between the patient's dofetilide and the inhaled sevoflurane was suspected and the patient was switched to total intravenous anesthesia with propofol. Within 10 minutes all PVC's disappeared and the QT interval started normalizing (Fig. 2). At the conclusion of a successful isolation of all pulmonary veins, the QT interval had shortened back to its baseline value. Dofetilide was discontinued and replaced by a β -blocker.

The effects of prolonged use of inhalational anesthetic agents on QT interval causing torsades de pointes in the setting of major noncardiac surgery have been reported.^{3,4} Inhibition of the repolarizing K⁺ channels in the ventricles has been implicated as the main mechanism of this complication. Physicians should recognize this life-threatening complication of inhaled anesthesia

particularly when combined with other QT prolonging drugs. If faced with this situation, a search for other reversible causes of QT prolongation should be undertaken.

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