

## Frequently occurring Torsades de pointes attacks in an old patient on solifenacin therapy and management strategy

To the Editor,

The most frequent reason for the acquired long QT syndrome and associated Torsades de pointes (TdP) is drugs. Solifenacin, an antimuscarinic drug, causes QT prolongation by decreasing the activity of potassium channels in phase 3 of the action potential (1). In these patients, temporary pacemaker (PM) implantation is a life-saving therapeutic approach (2).

An 84-year-old male patient was admitted to the emergency department with complaints of short-term episodes of loss of consciousness and cyanosis in the hands. The patient has been on treatment with 10 mg/day solifenacin for 15 days because of the urinary incontinence. In addition, he has been taking metformin (2000 mg/day) and atorvastatin (10 mg/day) for type 2 diabetes mellitus and hyperlipidemia. The patient's consciousness was clear in the first examination in the emergency department. Moreover, it was determined that the patient had a blood pressure of 120/80 mm Hg, heart rate of 72 bpm, and blood glucose of 140 mg/dL. During evaluations, he developed a sudden loss of consciousness. A sustained ventricular tachycardia (VT) attack was observed in the electrocardiogram (ECG) monitor, and synchronized cardioversion was applied with 50 J. After cardioversion, a normal sinus rhythm was established, and the patient's consciousness normalized again. A 12-lead ECG was obtained just after the VT episode. There were no ischemia-related alterations in 12-lead ECG; however, QT prolongation was determined. The corrected QT (QTc) interval was calculated as 548 ms. Cardiac enzymes and electrolytes were found to be in normal ranges. Despite the fact that the level of K<sup>+</sup> was 4 mE/L, parenteral K<sup>+</sup> and peroral magnesium treatment were provided to the patient because of the existence of VT resulting from QT prolongation. Echocardiographic examination of the patient demonstrated normal echocardiographic findings with an ejection fraction of 63%.

Frequent VT attacks reappeared after approximately 4h. Short-term episodes of a loss of consciousness accompanied those attacks. Parenteral magnesium therapy was initiated. Because of the unresponsiveness of VT attacks to the parenteral magnesium treatment, synchronised cardioversion was applied 8 times. Sustained VT episodes were reappearing in almost 5-10 min after cardioversion. A temporary VVI-PM was implanted in the patient. The heart rate was set as 110 bpm in PM. The VT attacks did not recur after PM implantation. In addition, coronary angiography was performed to rule out coronary artery disease. The angiography demonstrated no significant obstructive lesion in epicardial coronary arteries.

When PM was stopped after 8 h, QTc was determined to be 450 ms. Serum electrolytes were also in normal ranges at that moment. On the next day, solifenacin therapy was discontinued. TdP attacks did not recur. Temporary PM was removed after 24 h of observation. The patient was discharged after 3 days of hospitalization. He had no complaints in the outpatient controls, and the QT interval was measured as 420 ms.

In conclusion, patients taking QT prolonging drugs should be monitored in the hospitals for few days in case of TdP development. After documenting the first TdP attack, temporary PM should be immediately inserted with a ventricular rate of 110-120 bpm to shorten the QT interval.

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