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Case Report / Olgu Sunumu

Moxifloxacin dependent torsades de pointes in a bradycardic patient with multiple risk factors

Çoklu risk faktörleri olan bradikardik hastada moksifloksasine bağlı torsades de pointes

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ABSTRACT

Torsades de pointes (TdP), which is associated with a prolonged QT interval, is a rare but potentially fatal arrhythmia. The most common cause of drug-induced QT prolongation is inhibition of the rapidly activating component of the delayed rectifier potassium current. Rapidly activating component of the delayed rectifier potassium current inhibition delays repolarization by blocking the potassium in myocytes. In this article, we present a patient who had TdP risk factors including female sex, organic cardiac disease, and bradycardia. On the fifth day of 400 mg/day oral moxifloxacin administration, the patient had an episode of TdP, which progressed to ventricular fibrillation, and was successfully defibrillated.

Keywords: Cardiac surgery; moxifloxacin; torsades de pointes.

ÖZ

Uzamış QT aralığı ile ilişkilendirilen Torsades de pointes (TdP) nadir fakat ölümcül bir aritmidir. İlaçla indüklenmiş QT uzamasının en yaygın nedeni gecikmiş düzeltici potasyum akımlarının hızlı komponentinin inhibisyonudur. Gecikmiş düzeltici potasyum akımlarının hızlı komponentinin inhibisyonu miyositlerdeki potasyumu bloke ederek repolarizasyonu geciktirir. Bu yazıda; kadın cinsiyeti, organik kardiyak hastalık ve bradikardiyi içeren TdP risk faktörleri olan bir hasta sunuldu. 400 mg/gün oral moksifloksasin uygulamasının beşinci gününde hasta ventriküler fibrilasyona ilerleyen bir TdP epizodu geçirdi ve başarıyla defibrile edildi.

Anahtar sözcükler: Kardiyak cerrahi; moksifloksasin; torsades de pointes.

Ventricular tachyarrhythmias can occur after cardiac surgery. Following such events, several possible triggers should be ruled out immediately, primarily electrolyte abnormalities, cardiac ischemia, and drug therapies.[1] Fluoroquinolones are frequently prescribed antibiotics that are clinically important. Although generally well tolerated with safety profiles similar to those of other antimicrobial drugs, patients who take fluoroquinolones may experience significant adverse reactions, for example a prolonged QT interval, which can lead to potentially life-threatening arrhythmias such as torsades de pointes (TdP).

CASE REPORT

A 37-year-old woman who had undergone mechanical mitral valve replacement, tricuspid ring annuloplasty, and radiofrequency ablation two weeks prior to being admitted to our outpatient clinic with a sternal wound infection. Her preoperative left ventricular end-diastolic diameter and left ventricular end-systolic diameter were 5.6 cm and 3.8 cm respectively, and her left ventricular wall motion was normal. In addition, her preoperative corrected QT (cQT) interval was 42, and her postoperative cQT interval was 46 on an electrocardiogram (ECG). In the patient's first examination, the wound was hyperemic with no purulent discharge, and her white blood count (WBC) was slightly elevated. She was consulted with the infection committee, and the decision was made to obtain cultures and begin the administration of empiric moxifloxacin (Bayer Pharma AG., Wuppertal, Germany) 400 mg daily. In her second control five days after the drug was first administered, the inflammation

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Table 1. Laboratory findings at the time of hospitalization

Laboratory tests	
Serum magnesium	2.4 meq/Lt
Serum potassium	4.4 mmol/L
Serum creatinine	0.89 mg/dL
Alanine transaminase	23 U/L
Serum aspartate amino transferase	27 U/L
Leukocyte count	16.000 µL
Sedimentation rate	45 mm.
C-reactive protein	67 mg/L

had regressed, but there still was very mild exudation from the wound and no changes in the blood count results (Table 1). After these evaluations, the patient was hospitalized and started on oral moxifloxacin. The wound was also dressed. Three hours after being hospitalized, the patient had an episode of hypotension. Although she was on sinus rhythm at the time, monitorization showed atrial fibrillation (AF) with a QT interval of 0.66 seconds (cQT interval was 54.8) as well as an episode of TdP (Figure 1). This subsequently progressed to ventricular fibrillation (VF), and the patient was then successfully defibrillated. Afterwards, she experienced AF with a heart rate of 50 beats/min, and the cQT interval was 39.7 (Figure 2). Furthermore, cardioversion occurred two times, but the sinus rhythm could not be sustained. As a result, a temporary transvenous cardiac pacemaker with a rate of 110 beats/min was implanted in the patient.

The moxifloxacin was then discontinued, and piperacillin-tazobactam was initiated that resulted in a regression in the inflammation. By the end of the first week, the patient was free of infection, and after her cultures came back negative, a pacemaker in VVI mode (Medtronic, Minneapolis, MN, USA) was implanted due to AF caused by a low heart. She was then discharged on the 12th day after her admission.

DISCUSSION

Ventricular tachyarrhythmias can occur after cardiac surgery and are generally seen in the early postoperative period. In this case, the arrhythmia was observed on the 19th postoperative day. Our patient's serum electrolytes were normal, and there was no cardiac ischemia on her ECG. However, she had risk factors for TdP such as female gender, extreme bradycardia, and previous cardiac surgery and medication. As in our case, sometimes patients who have been prescribed flouroquinolones are genetically susceptible to drugs that cause a prolonged QT interval, so this should be kept in mind when deciding on the treatment regimen. The safety profile for fluoroquinolones is equal to that of other antibiotics, but they can have serious adverse effects, and because moxifloxcacin blocks the rapid component of the delayed rectifier potassium current (IKr), it can cause the prolonged QT interval.

In patients with low cardiac rates, a less potassium is released outside the cell due to reduced cardiac repolarization and extracellular potassium concentration. This decrease in potassium concentration then increases the IKr blockage level. Thus, the use of moxifloxacin provides a base for the development of TdP, especially when bradycardia is present.[2]

In the literature, there are five reports of TdP associated with moxifloxacin, but only one patient was

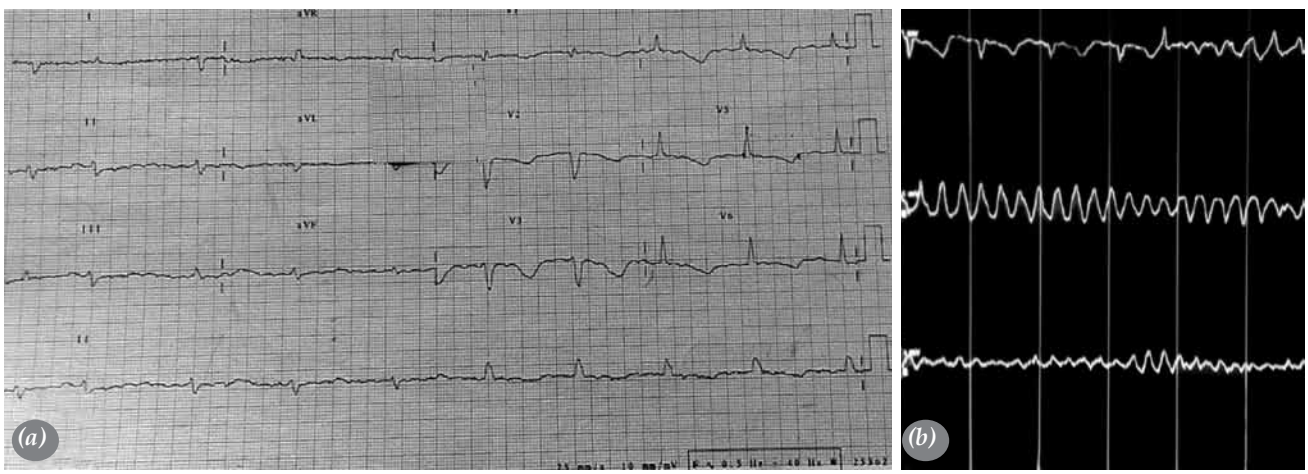

Figure 1. (a) The electrocardiogram on the fifth day of moxifloxacin treatment showed a heart rate of 60 beats/min and a QT interval of 0.66 seconds. (The paper speed was 25 mm/s). **(b)** The monitor reading showed the presence of TdP.

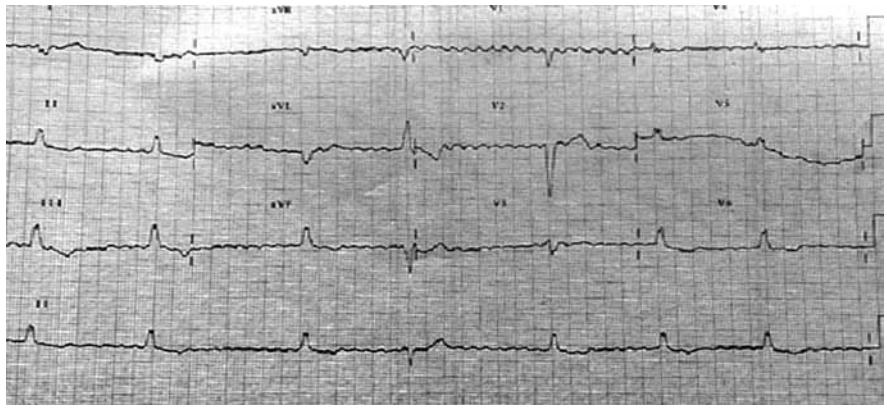

Figure 2. Readout showing the atrial fibrillation rhythm with a rate of 50 beats/min. (The paper speed was 25 mm/s).

converted to VF,[3-5] and cardiac pacing was needed in three of the others.

Our case had a prolonged QT interval followed by a TdP attack which then triggered VF. During all of these fatal arrhythmias, the electrolyte and arterial blood gas levels were normal. Our findings led to the hypothesis that moxifloxacin was responsible for the prolonged QT interval. However, this drug is not the only source of this condition and torsades de pointes. Radiofrequency ablation, as reported by Grimm et al.,[6] can also cause these arrhythmias.

In conclusion, if a patient has multiple risk factors for TdP, the risk/benefit ration must be carefully assessed before prescribing drugs which are known to cause a prolonged QT interval, and this should be kept in mind during the patient follow-up.

Declaration of conflicting interests

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