Çağdaş Tıp Dergisi 2015;5(3): 196-198
DOI: 10.16899/ctd.36735

Case Report / Olgu Sunusu

A Case of Ventricular Tachycardia From Use of Clomipramine at Therapeutic Dosage

Tedavi Dozunda Klomipramin Kullanımına Bağlı Gelişen Ventriküler Taşikardi Olgusu

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Başvuru Tarihi/Received :

17-04-2014

Kabul Tarihi/Accepted:

13-06-2014

ABSTRACT

A 57-year-old housewife was admitted to the emergency department with a 2-hour history of palpitations and acute dyspnea. Upon detailed examination of her medical history, it was found that she had been suffering from tachycardia attacks lasting 10-15 minutes for six months and been receiving anti-depressant therapy for ten years. Her electrocardiogram (ECG) indicated ventricular tachycardia (VT), which rapidly changed to sinus rhythm with cardioversion. Clomipramine therapy was ceased as QTc was calculated as 600 ms. At the initial 3-day follow-up, the rhythm of the patient who had three times attacks was converted to sinus rhythm with cardioversion. Later, the QTc value of the patient who had no VT attack was evaluated as 420 ms in her ECG which was analyzed at the end of the first week of her hospitalization. The patient was discharged from the hospital on the fifteenth day of her hospitalization. We present a case of VT from use of clomipramine at therapeutic dosage.

Key Words: Klomipramin, long QT syndrome, ventricular tachycardia

ÖZET

57 yaşında kadın 2 saattir devam eden çarpıntı ve nefes darlığı şikayeti ile başvurdu. Hastanın hikayesi sorgulandığında, çarpıntı ataklarının 6 aydır olduğu, 10-15 dakika sürüp geçtiği ve on yıldır antidepresan tedavi aldığı öğrenildi. Hastanın elektrokardiyogramındaki ventriküler taşikardi (VT) mevcuttu ve kardiyoversiyonla hızlı bir şekilde sinüs ritmine döndürüldü. QTc mesafesi 600 ms olarak ölçülmesi üzerine Klomipramin tedavisi kesildi. Takibinin ilk 3 gününde 3 kez oluşan ventriküler taşikardi atağı kardiyoversiyon ile sinüs ritmine döndürüldü. Hastanın yatışının ilk haftasının sonunda QTc mesafesi 420 ms olarak ölçüldü ve sonrasında hiç VF atağı olmadı. Hasta yatışının onbeşinci günü hasteneden taburcu edildi. Biz tedavi dozunda klomipramin kullanımına bağlı bir VT olgusunu sunmak istedik

Anahtar Kelimeler: Klomipramin, long QT sendromu, ventriküler taşikardi

INTRODUCTION

Many drugs can prolong the QT interval of the electrocardiogram (ECG). This effect is important as it is associated with ventricular tachycardia and possible sudden cardiac death. Clomipramine is a tricyclic antidepressant for psychiatric disorders that can induce QT prolongation, which may lead to ventricular tachycardia. Herein, we describe the case of a patient with a history of depression who presented with a prolonged QT interval and episodes of ventricular tachycardia while taking therapeutic doses of clomipramine.

Case report

A 57-year-old housewife was admitted to the emergency department with a 2-hour history of palpitations and acute dyspnea. Upon detailed examination of her medical history, it has been found that she had been suffering from tachycardia attacks lasting 10-15 minutes for six months and been receiving anti-depressant therapy for ten years. Her blood pressure was 120/80 mm Hg, and heart rate was 167/min. Her electrocardiogram (ECG) indicated ventricular tachycardia (VT) (Figure 1),

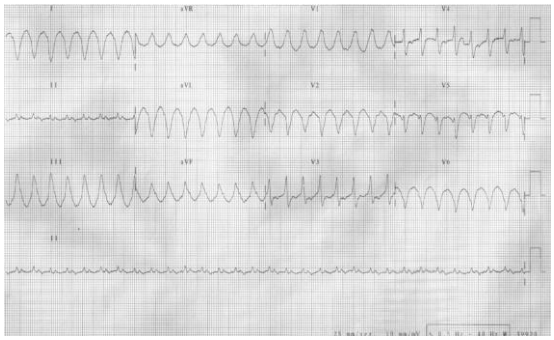

Figure 1 Electrocardiography revealing ventricular tachycardia on admission.

which rapidly changed to sinus rhythm with cardioversion (Figure 2).

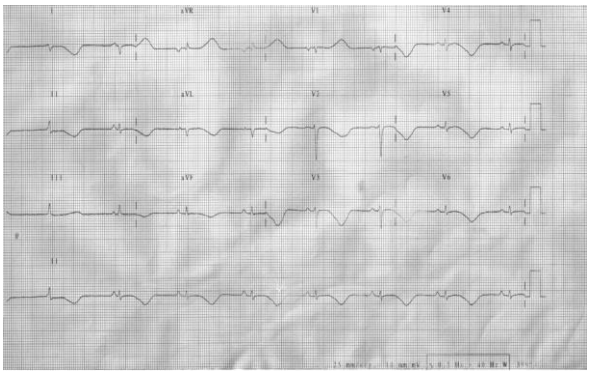

Figure 2 Electrocardiography showing long-QT (QTc: 600ms) after DC.

Following this, she was started on prophylactic amiodarone infusion with the dosage of 5 mg/kg in the form of a short term infusion within 20 minutes, followed by a continuous intravenous infusion of 1100 mg over 24 hours. Clomipramine therapy was ceased as QTc was calculated as 600 ms. At the initial 3-day follow-up, rhythm of the patient who had ventricular tachycardia attacks three times was converted to sinus rhythm with cardioversion. Later, the QTc value of the patient who had no VT attack was evaluated as 420 ms in her ECG (Figure 3)

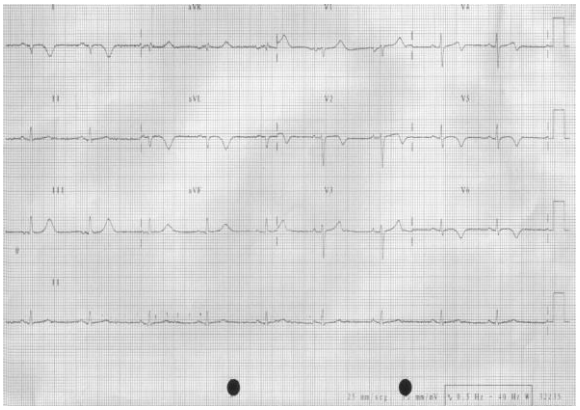

Figure 3 Electrocardiography showing normal-QT (QTc: 420ms) at the end of the first week of her hospitalization which was analyzed at the end of the first week of her hospitalization.

The patient was discharged from the hospital on the fifteenth day of her hospitalization. During her follow-up examination, she had been applied electrophysiologic study (EPS) and coronary angiography in another medical center. Arrhythmias were not induced and her coronary angiography was normal.

DISCUSSION

Potential arrhythmia due to tricyclic antidepressants was reported in 1970s. (1). It has been observed that ceasing antidepressant therapy caused arrhythmias to resolve. The results of the studies, aimed at determining have researched whether the findings of patients basic electrocardiography were the predictors of serious arrhythmias, which might have occurred after the usage of tricyclic antidepressants, contradict each other. Boehnert et al, in their study indicated that determination of the maximal limb-lead QRS duration predicts the risk of seizures and ventricular arrhythmias in acute overdose with tricyclic antidepressants (2). In addition, this study indicated serum drug levels are not of predictive value. In their study, Buckley et al did not support the view that QRS duration predicts arrhythmias in acute overdose with tricyclic antidepressants (3). This study concluded that the R/S ratio in aVR>0.7 was most strongly related to arrhythmia but had estimated positive and negative predictive values of only 41 % and 95 %, respectively. In addition, this study indicated QTc>500ms is not of predictive value. Therefore, it is hard to predict arrhythmia, which may occur after the usage of tricylic antidepressants.

Tricyclic antidepressants are widely used for the treatment of depression. However, even under a clinical dosage regimen, they occasionally induce QT prolongation. In a study in which the where arrhythmogenic potency of antidepressants, imipramine (tricyclic antidepressant agent) and fluvoxamine (selective serotonin reuptake inhibitor agent) were compared, it was found that the potency for QTc prolongation of imipramine was 1.7-fold higher than that of fluvoxamine; fluvoxamine's clinical arrhythmogenic risk in therapeutic plasma concentrations was five times higher than that of imipramine (4).

CONCLUSION

Accurate identification of patients at risk of QT_c prolongation and ventricular tachycardia is a difficult. It is important to assess each patient before prescribing an implicated drug and then to closely monitor them afterwards. Clinicians should be alert to the increasing list of drugs causing QT_c prolongation and to the presence of predisposing conditions.

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