

Torsade de Pointes Following Myocarditis; A Case Report

Miyokarditi Takiben Gelişen Torsade de Pointes: Olgu Sunumu

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Abstract

Myocarditis is defined as myocardial edema associated with non-ischemic necrosis in myocardial tissue. Acute viral myocarditis may cause several electrocardiographic changes. These include conduction disturbances, disorders of repolarization, ventricular ectopic beats, and ventricular tachycardia. "Torsade de pointes" is a malignant ventricular arrhythmia associated with prolongation of the QT interval. In this report, we present a case with myocarditis that developed Torsade de pointes and sudden cardiac arrest. (*Sakarya Med J* 2019, 9(2):356-360)

Keywords Ventricular tachycardia; sudden death; electrocardiography; Torsade de pointes; myocarditis.

Öz

Miyokardit, miyokard dokusunda iskemik olmayan nekroz ile ilişkili miyokard ödemi olarak tanımlanır. Akut viral miyokardit, çeşitli elektrokardiyografik değişikliklere neden olabilir. Bunlar, iletim bozuklukları, repolarizasyon düzensizlikleri, ventriküler ekotopik atımlar ve ventriküler taşikardidir. "Torsade de pointes", QT aralığının uzamasıyla ilişkili malign ventriküler bir aritmidir. Bu yazıda Torsade de pointes ve ani kalp durması gelişen miyokardit olgusu sunulmaktadır. (*Sakarya Tıp Dergisi* 2019, 9(2):356-360)

Anahtar kelimeler Ventriküler taşikardi; ani ölüm; elektrokardiyografi; Torsade de pointes; miyokardit

INTRODUCTION

Myocarditis is a disease characterized by acute or chronic inflammation of the heart muscle. It is usually nonischemic and the result of exposure to different infective agents, systemic diseases, and/or toxic substances.¹ Myocarditis is classified as acute or chronic based on the chronology and the type of inflammatory substance (e.g., lymphocytic, neutrophilic, eosinophilic, or giant cells) detected in the myocardial fibers.^{2,3} Patients with myocarditis often present with chest pain, transient electrocardiographic changes, and even, life-threatening cardiogenic shock or ventricular arrhythmia.

Here we have described the clinical process of a patient with myocarditis who was admitted to the emergency department with complaints of palpitations and chest pain.

CASE REPORT

A 16-year-old female was admitted to the county state hospital with complaints of chest pain and palpitations that persisted for 2 days. The electrocardiography (ECG) showed a normal sinus rhythm, right axis deviation, and premature ventricular complexes (PVCs) [nonsustained polymorphic ventricular tachycardia (VT) and ventricular couplets]. Additionally, the corrected QT (QTc) interval was slightly prolonged (QT/QTc=320/480 ms) (Fig. 1-a). Because the patient was hemodynamically stable, an intravenous infusion of 300 mg of amiodarone over 30 minutes was started, and the patient was referred to our institution. In the intensive care unit, her general condition was good and her vital signs were stable. There were no clinical signs of heart failure, and she had no complaints of chest pain. Her temperature was 37.3°C. The ECG showed sinus tachycardia (120 beats/min), PVCs, and a QT/QTc of 380/535 ms (Fig. 1-b). The transthoracic echocardiography revealed that her left ventricular diameters were near the upper limit, and that she had a hypokinetic interventricular septum and mild mitral valve regurgitation. Her left ventricular ejection fraction (LVEF) was 52%. Upon admission, her cardiac troponin T level was 1,630 pg/ml (normal ran-

ge<100 pg/ml) and her creatine phosphokinase muscle/brain isoenzyme level was 8.2 ng/ml (normal range<4.0 ng/ml). However, patient's electrolytes were within the normal limits. Her white blood cell count was 17x10⁹/l, her erythrocyte sedimentation rate and C-reactive protein level were slightly increased, and her procalcitonin level was normal. Based on the clinical examination, we suspected that this patient had viral myocarditis.

A β -blocker (100 mg of metoprolol in the morning and 50 mg in the evening) and an angiotensin converting enzyme inhibitor (ACEI) (5 mg of perindopril) were started. On the first day, she exhibited nonsustained VT and bigeminy PVCs (Fig. 1-c). Additionally, her amiodarone infusion was discontinued due to QT interval prolongation. The coronary angiography showed that there were no stenoses or obstructive lesions in the coronary artery. Moreover, the QT and QTc intervals measured from the daily ECG samples were significantly longer (400/515 ms) (Fig. 2-a). On the fifth day of her hospitalization, sudden cardiac arrest (SCA) developed during sleep. Ventricular fibrillation (VF) could be seen on the monitor, and she was defibrillated using a single 200J shock. Her circulation was successfully restored. In the monitor's records, we saw that she developed VF following polymorphic VT [torsade de pointes (TdP)] triggered by a PVC (Fig. 2-b). On the ECG obtained 1 hour later, the QT/QTc was 480/610 ms (normal sinus rhythm and no PVCs) (Fig. 2-c). Her medical treatment (β -blocker and ACEI) was continued, and there was no decrease in her LVEF. The QT and QTc intervals returned to normal on day four, after her cardiac arrest. One month later, her ECG was normal and the echocardiography showed that this patient had normal ventricular wall movement (Fig. 2-d).

DISCUSSION

Myocarditis is a common inflammatory disease of the myocardium. Its diagnosis is often based on the clinical scenario and supportive imaging evidence, such as echocardiography and magnetic resonance imaging. Although

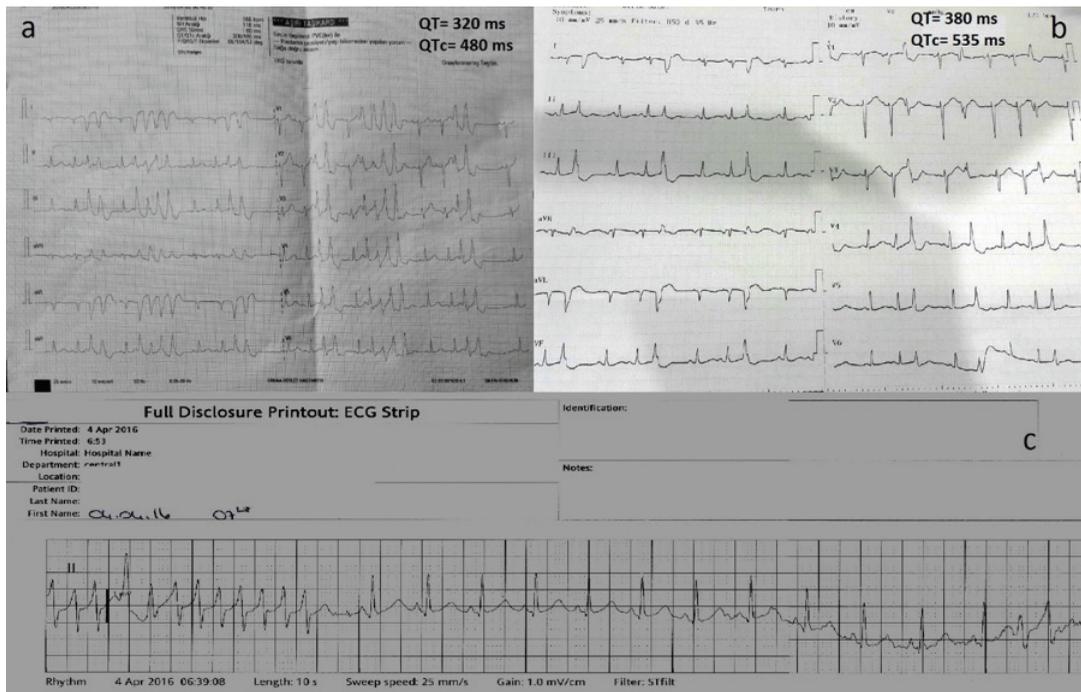


Figure 1. (a) The electrocardiogram (ECG) obtained at the external center; (b) The ECG obtained upon presentation to our institution; (c) The example of nonsustained ventricular tachycardia from the recording on the first day.

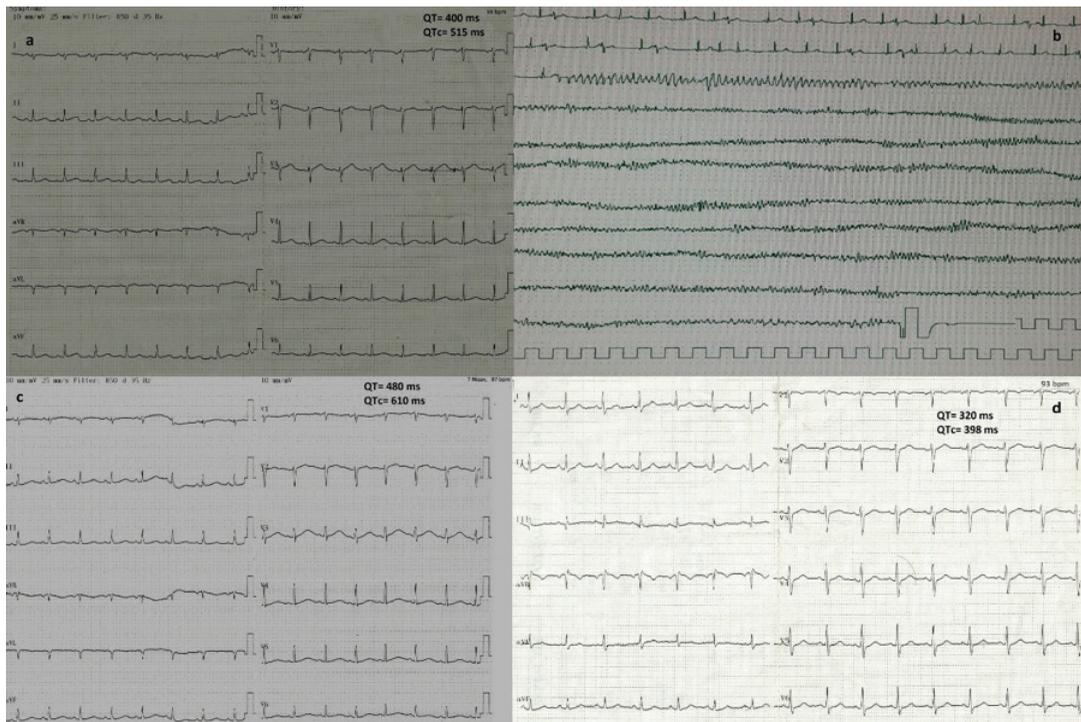


Figure 2. (a) The electrocardiogram (ECG) obtained at day 4th of follow-up; (b) ventricular fibrillation after Torsade de Pointes; (c) The ECG obtained at 1st hour after ventricular fibrillation; (d) The ECG obtained at 1st month after discharge.

an endomyocardial biopsy (EMB) is the gold standard diagnostic criterion, it is not performed routinely. Infections, drugs, toxins, and systemic diseases can all cause myocarditis; however, the Coxsackie B virus is the most common cause of viral myocarditis.⁴

Viral myocarditis can manifest itself with cardiovascular complaints, such as palpitations, syncope, chest pain, and shortness of breath, and the presentation of these complaints is based on the phase and severity of the disease.⁴ The progression of viral myocarditis is best explained using murine models.⁵ According to these, three phases can be recognized: an acute phase resulting from direct cellular damage by the infectious agent, a subacute phase involving an innate host immune response against the virally-infected cells, and a chronic phase that leads to dilated cardiomyopathy. A fourfold increase in the titers of the neutralizing antibodies against the Coxsackie B virus is considered to be evidence of a new infection.⁶ Our patient presented during the acute phase, complaining of palpitations accompanied by a subfebrile body temperature. Although most patients with viral myocarditis exhibit a silent disease process, some patients may precipitate major cardiac arrhythmias (e.g., PVCs, VT, VF, and TdP), a complete atrioventricular block, and sudden cardiac death.^{7,8}

Myocarditis usually heals with supportive therapy and without sequelae. The treatment of myocarditis involves specific treatments for the special clinical manifestations, such as heart failure and arrhythmias. After the acute phase, standard heart failure medications, such as β -blockers, ACEIs or angiotensin receptor blockers, and aldosterone antagonists, can be initiated if the patient shows symptoms of heart failure.⁹ However, the cause and severity of the disease play important roles in the myocarditis prognosis.⁶ Although the prognosis of acute myocarditis is good, the prognosis of giant cell myocarditis is poor. A cardiac transplantation is the only feasible option for patients who do not respond to medical treatment.¹⁰

This patient was administered a β -blocker, ACEI, and amiodarone (for frequent ventricular extrasystole), which make up the standard myocarditis treatment in the intensive care unit. During this time, there was a QT prolongation. On the first day of treatment, we stopped the amiodarone due to an increase in the QT interval. On the fifth day of treatment, this patient was asymptomatic with supportive therapy; however, VF developed while she was sleeping, followed by defibrillation. There was no decrease in the LVEF. The QT and QTc intervals returned to normal on the fourth day following this patient's SCA. An EMB and immunosuppressive therapy were not planned because this patient did not show signs and symptoms of cardiac dysfunction. A high dose β -blocker was given as an anti-arrhythmic drug for her PVCs and TdP, and an ACEI was given for the septal hypokinesia, and these were continued after her discharge. One month later, her ECG was normal, and there were no signs of septal hypokinesia on her echocardiography.

CONCLUSIONS

Myocarditis should be diagnosed after excluding other diseases such as coronary artery disease. When myocarditis is diagnosed, the etiology should be defined quickly. It should first be suspected of myocarditis caused by viruses. After defining the pathogen, definitive treatment can be conducted as early as possible to improve the prognosis.

Asymptomatic myocarditis should not be taken too lightly. It may cause an increased QT interval and lead to unexplained sudden cardiac death. Therefore, these patients should be followed closely and about a week in terms of early or late arrhythmic complications.

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