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A Sudden Complication: Electrocute-Induced Atrial Fibrillation

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Abstract

The most serious complications developing after an electric shock are cardiac arrhythmias, skin lesions, and multiple organ injuries after a fall from heights. Although mortality due to high-voltage electric shocks is higher, low-voltage electrical shocks can cause cardiac complications. Of the cardiac complications due to electric shock, myocardial necrosis, and ventricular arrhythmias are more common. Although rarer than ventricular arrhythmias, supraventricular arrhythmias can also occur. However, the mechanism of developing arrhythmias after electric shocks cannot be completely explained. In this present case report, AF with rapid ventricular response which developed after a 380-volt electrical shock in a 42-year-old male patient with no risk factors for AF will be discussed.

Keywords: Arrhythmia, Atrial Fibrillation, Electrocute

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INTRODUCTION

Today, the number of patients admitted to emergency services due to electrical shocks is increasing. In the United States, 17 thousand people a year are admitted to emergency services due to electric shocks, and about 500 to 1000 of them lose their lives due to complications. (1) Deaths occurring in the early stages are usually due to ventricular fibrillation.

(2) Although mortality due to high-voltage electric shocks is higher, low-voltage electrical shocks can cause cardiac complications (3).

The most serious complications developing after an electric shock are cardiac, skin lesions, and multiple organ injuries after a fall from heights (4). Of the cardiac complications due to electric shock, myocardial necrosis, and ventricular arrhythmias are more common. Although rarer than ventricular arrhythmias, supraventricular arrhythmias can also occur (5). However, the mechanism of developing arrhythmias after electric shocks cannot be completely explained (6).

Atrial fibrillation (AF) after electrical shock is a rare case. In a study performed with 182 patients who developed complications after an electric shock, AF was reported only in two of the patients. The risk of developing AF in low-voltage electric shocks is even lower. AF is the most common chronic cardiac arrhythmia. Its incidence increases with age. Among the other known risk factors are hypertension (HT), heart valve diseases, heart failure, ischemic heart disease, pulmonary embolism, and thyrotoxicosis (3).

In this present case report, AF with rapid ventricular response which developed after a 380-volt electrical shock in a 42-year-old male patient with no risk factors for AF will be discussed.

CASE

A 42-year-old man who worked in a factory was brought to the emergency room by ambulance after he got a 380-volt electric shock from the power cable. The patient who lost consciousness for a short time and fell to the ground when he got the electric shock recovered his consciousness while he was transported in the ambulance. After the initial intervention, the patient was given oxygen support and monitored.

The patient described pain at the junction of the left 12th costa and midclavicular line. He also had palpitations. The physical examination of the patient who had no cardiac risk, did not use any substance, never drank alcohol or smoked, and had no previous palpitation complaints revealed a heart rate of 163 beats per minute and a blood pressure of 130 / 90mmHg. His oxygen saturation level was 98%. A second-degree burn was observed on the second and third fingers of his right hand where he was exposed to an electrical current (Figure 1A). The exit wound (0.5x0.5cm) was at the junction of the left 12th costa and midclavicular line. No extra sounds were heard on auscultation during the cardiovascular system examination. Heartbeats were considered arrhythmic. The patient's electrocardiogram (ECG) demonstrated atrial fibrillation (AF) with a rapid ventricular response rate was 163 beats per minute (Figure 1B). Because the patient was hemodynamically stable, electrical

cardioversion was not considered first. In the emergency room, he was intravenously (IV) administered 25 mg diltiazem twice at an interval of 30 minutes. Thirty minutes after the second administration of diltiazem, the patient's heart rhythm was considered atrial fibrillation. However, his heart rate dropped to 144 beats per minute. The patient was transferred to the coronary intensive care and his medical follow-up continued there.

The patient's cardiac enzymes, hemogram, and biochemistry tests were taken. To rule out multiple organ traumas, postero-anterior chest X-ray, thoraco-abdominal, and cerebral computed tomography imaging were taken. His blood test results showed complete blood count, kidney functions, electrolytes, troponin, and myoglobin levels within normal limits. The patient's posteroanterior chest X-ray and computed tomography revealed no pathological findings. The patient who did not recover sinus rhythm within two hours in the intensive care unit started on amiodarone infusion (1 gr in 24 hours). In addition, enoxaparin at a 0,6mg/kg dose was administered subcutaneously (2x1). At the third hour of amiodarone infusion, the patient recovered the sinus rhythm (Figure 1C). His vital signs were stable. Cardiac enzymes checked three times at three-hour intervals were negative.

The patient's echocardiographic evaluation revealed that the left ventricular diameter and wall motions were normal, the ejection fraction (EF) was 60%, the first-degree mitral and aortic insufficiency was present, and the size of the right atrium and ventricle were in normal limits.

During the follow-up, the patient remained in sinus rhythm. 24-hour troponin and thyroid function tests were within normal limits. The patient whose Chadsvasc score was 0 was not given anticoagulant therapy. On the second day of his hospitalization, the patient was prescribed diltiazem tablets (90 mg,1x1) and then discharged.

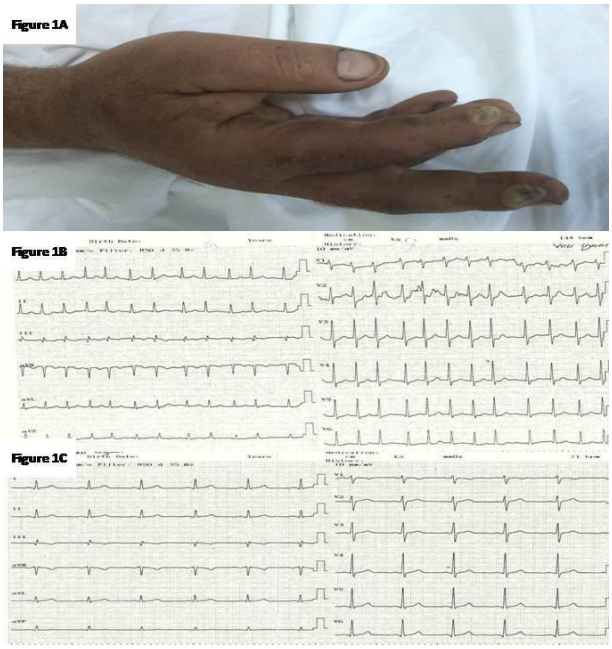

Figure 1A: right hand where he was exposed to an electrical current 1B: electrocardiogram (ECG) demonstrated atrial fibrillation, Figure 1C: sinus rhythm

DISCUSSION

The heart is one of the organs most vulnerable to electric shocks. Although the exact mechanism is not known, the reason for the heart to be vulnerable to electric shocks is that blood, a very good conductor of electricity, continuously circulates in the chambers of the

heart and the heart has a widespread neural network (5,7).

Among the electric shock-related cardiac complications are myocardial infarction, left ventricular dysfunction, cardiac rupture, and arrhythmias. Although electric shocks frequently lead to ST-segment changes and sinus tachycardia, among the other arrhythmias are ventricular extrasystoles, ventricular tachycardia, ventricular fibrillation, atrial tachycardia, atrial fibrillation, atrioventricular block. (8) Although arrhythmias, as in our case, usually occur within the first day after the electric shock, they may occur within the first few days as well (9).

The majority of sudden deaths due to electric shocks are caused by asystole and ventricular fibrillation. While ventricular fibrillation is common in low-voltage electrical injuries, asystole is more common in high-voltage injuries. In low-voltage electrical injuries, the rate of cardiac side effects is lower (1).

Atrial fibrillation due to electric shocks is a rare condition (10). According to studies in the literature, while the occurrence of high-voltage electric shock-related AF is more likely, low-voltage electric shock-related AF is rare. As in our case, electric shocks require a multidisciplinary approach (8). Detection of internal organ traumas should be started as soon as the patient is admitted to the emergency room. Although indications for cardiac monitoring due to electric shocks cannot be fully explained, if the patient demonstrates such symptoms as loss of consciousness, abnormal ECG, high voltage exposure, and if the patient has cardiac disease history, particularly a history of cardiac arrhythmias, it is recommended to monitor the patient for 24 hours (10).

Because AF after an electrical injury is a rare case, there are no clear guidelines on managing AF. While electrical cardioversion is recommended for hemodynamically unstable patients, different approaches have been reported for hemodynamically stable patients, like our case.

Zihni et al. reported that in their case, the patient who developed AF after an electric shock spontaneously recovered sinus rhythm within 24 hours after he was intravenously administered 5 mg metoprolol to control heart rate. Because they could not recover the patient's sinus rhythm with 150 mg flecainide and amiodarone infusion within 24 hours, Alex and Mark returned the patient to sinus rhythm with the electrical cardioversion method. Because their patient was hemodynamically unstable, Akdemir et al. reported that they preferred electrical cardioversion as the first choice. In their patient, Ercan et al. managed to control heart rate with intravenously administered digoxin, and the patient spontaneously returned to the sinus rhythm. In their case, Mitrakrishn et al. reported that the patient spontaneously returned to the sinus

rhythm 6 hours after they managed to control heart rate with intravenously administered atenolol.

In our case, because the patient had the symptoms of loss of consciousness and abnormal ECG findings (AF with rapid ventricular response), he was closely monitored. Because he was hemodynamically stable, he was intravenously (IV) administered diltiazem twice at an interval of 30 minutes in the emergency room.

The patient's heart rate dropped from 163 beats per minute to 144 beats per minute.

Because the patient did not return to sinus rhythm spontaneously during the two-hour follow-up in the intensive care unit, amiodarone infusion was started. The patient returned to the sinus rhythm 3 hours later and remained in the sinus rhythm during the 24-hour monitoring.

CONCLUSION

Our case showed that although the patient had no risk factor for AF, he was exposed to low voltage, and AF after the electric shock is not common, he developed AF. In the management of AF, electrical cardioversion should be the first choice in hemodynamically unstable patients. However, there is no consensus on the treatment of hemodynamically stable patients. On the other hand, in cases reported, heart rate control was often established, patients spontaneously returned to sinus rhythm, and thus aggressive interventions were not needed.

Ethics Committee Approval: The presented study is qualitative and consent was obtained by giving information about the study by one-to-one interviews with the subjects who agreed to participate. The study was carried out by paying attention to the Declaration of Helsinki.

Peer-review: Externally peer-reviewed

Author Contributions: Concept: UO, AF, Design: UO, AF, Data Collection and Processing: UO, AF, Analysis and Interpretation: GA Writing: GA

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