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Case Report

Commotio cordis during prolonged cardiac ventricular repolarization due to exercise-induced hypokalemia: A case report

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Abstract

Background: Commotio cordis is ventricular fibrillation after a direct precordial blow without mechanical damage to organs. Early recognition and prompt cardiopulmonary resuscitation with defibrillation save lives. According to previous reports, the major determinants of commotio cordis include location and timing of the blow. Other risk factors are young age, a thin and undeveloped chest cage, and a small hard spherical projectile. The pathophysiology of commotion cordis remains uncertain, such as individual susceptibility to prolonged repolarization and long QT.

Case report: A 32-year-old man suddenly collapsed after a direct blow to the anterior chest from an elbow during a basketball game. A bystander called an ambulance and activated the emergency medical service immediately. Dispatch assisted the bystander with cardiopulmonary resuscitation via telephone. An automated external defibrillator was applied when emergency medical technicians arrived, which showed ventricular fibrillation. The patient regained a pulse after the first electrical shock and five-cycle-chest compression. Further examinations revealed a long QT on electrocardiogram and hypokalemia. The QT interval was within normal limits without any ventricular arrhythmia after the potassium level had been normalized.

Conclusion: This case reminds physicians of the risk of commotio cordis associated with exercise-induced hypokalemia. An electrocardiogram should be checked for QT prolongation immediately after return of spontaneous circulation in patients of sudden cardiac arrest. Supplying adequate potassium for hypokalemia related QT prolongation should be considered as treatment and primary prevention in such case.

Keywords: cardiac arrhythmia; cardiac concussion; commotio cordis; sudden cardiac death; ventricular fibrillation

1. Introduction

Commotio cordis is a ventricular fibrillation after direct precordial blow without mechanical damage of organs.[1] The mortality rate of commotio cordis is high.[2] Early recognition, and prompt cardiopulmonary resuscitation (CPR) with defibrillation saves lives.[3] According to previous reports, there are two major determinants of commotio cordis: the location and the timing of the blow.[4] Other risk factors include young age, thin and undeveloped chest cage of the patient, and a small, hard, spherical projectile.[5,6] However, the details of pathophysiology remains uncertain, such as individual susceptibility with prolonged repolarization and long QT.

We report a case of ventricular fibrillation after a blow to the chest during a basketball game. The patient developed return of spontaneous circulation after defibrillation. Further examinations revealed a long QT on electrocardiogram (ECG) and hypokalemia. The QT interval was within normal limits without any ventricular arrhythmia after a normalized potassium level. This case reminds us of the potential risk of

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prolonged repolarization due to exercise-induced hypokalemia in commotio cordis.

2. Case Report

A 32-year-old man, who was previously healthy without a medical history, suddenly collapsed after a direct blow to the anterior chest from an elbow during a basketball game. According to a witness' statement, a bystander called an ambulance and activated the emergency medical service immediately. Dispatch assisted the bystander with CPR via telephone. An automated external defibrillator was applied when emergency medical technicians arrived, which showed ventricular fibrillation (Figure 1). The patient regained a pulse after the first electrical shock and five-cycle chest compression. He was transported to our hospital.

The patient was clear and oriented in the emergency department, but he could not recall what happened. There were sequelae of dull headache, chest tightness, and muscle soreness. In addition, none of the patient's family members had suffered from sudden cardiac death. Initial vital signs were body temperature of 37.5°C, pulse rate of 129 beats/min, and blood pressure of 128/60 mmHg. A physical examination was unremarkable. No jugular vein engorgement or peripheral edema was detected. No bruises or wounds were found on the patient's anterior chest. ECG revealed sinus tachycardia with a heart rate of 104 beats/min and QT prolongation with a corrected QT interval of 510 ms by Bazett's[7] formula (Figure 2). Chest radiography showed a normal heart size, a clear lung field, and an intact thoracic bony cage structure. A hemogram showed a white cell count of 17,300/dL and hemoglobin levels of 15.0 g/dL. Blood biochemistry values were as follows (normal reference range): sodium, 144 (134−148) mEq/L; potassium, 2.7 (3.6−5.0) mEq/L; creatinine, 1.34 (0.4−1.0) mg/dL; glucose, 187 (70−105) mg/dL, aspartate transminase, 121 (≤ 34) U/L; alanine transaminase, 68 (≤ 36) U/L; myoglobin, 66.3 (17.4−105.7) ng/mL; and troponin I, 0.129 (< 0.05) ng/mL.

Left ventricular systolic function was fair with an ejection fraction of 55% derived from bedside M-mode ECG. No structural heart disease was detected.

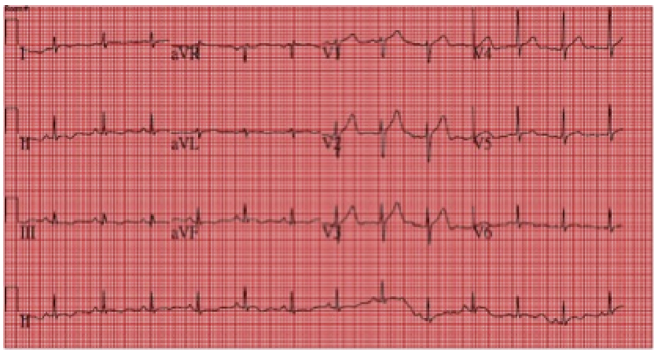

Figure 2. Initial electrocardiogram of our patient in the emergency department.

The initial impression was ventricular fibrillation due to a chest contusion and exercise-induced hypokalemia. The patient was on a continuous ECG monitor with potassium supplied by intravenous potassium chloride solution. Cardioangiography was performed and demonstrated patent coronary arteries. Further electrophysiological studies showed no inducible sustained cardiac arrhythmia. Serial serum troponin I and potassium levels were within normal limits. A follow-up ECG showed a corrected QT (QTc) interval of 470 ms (Figure 3). The patient was transferred from the intensive care unit to the general ward, in a stable condition, on Day 3, and was discharged 10 days later without neurological or psychological deficits.

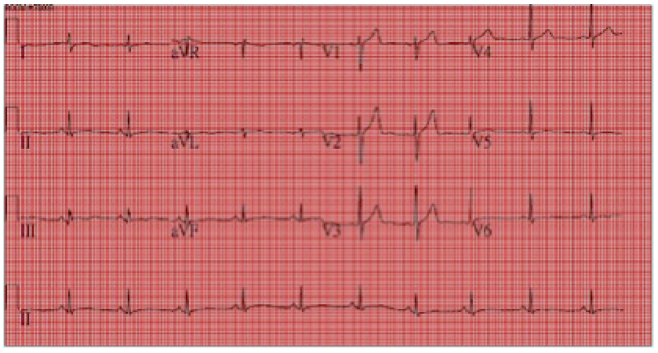

Figure 3. Electrocardiogram after serum potassium level was normalized.

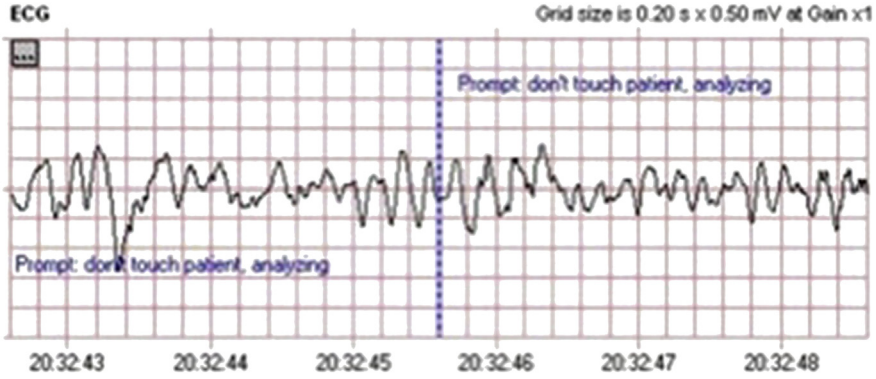

Figure 1. Ventricular fibrillation recorded from an automated external defibrillator. ECG = electrocardiogram.