An Unusual Case of Commotio Cordis Resulting in Ventricular Flutter

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Abstract

A 16-year-old male developed palpitations immediately following chest impact with a soccer ball. The patient was noted to have ventricular flutter in a delayed presentation that was successfully treated. While ventricular fibrillation is the predominant arrhythmia following commotio cordis, ventricular flutter may occur as well. Ventricular flutter may be better tolerated in a young athletic individual with structurally normal heart and may lead to a delayed presentation.

Keywords: Commotio cordis, palpitations, ventricular flutter

Introduction

Commotio cordis refers to sudden cardiac arrest after direct chest impact.[1,2] Most impacts occur in competitive sports, with some reports during recreational activities and violent altercations.[2,3] Although early recognition and cardiopulmonary resuscitation improve survival, commotio cordis remains a potentially fatal condition. This report describes an interesting case of an adolescent male with commotio cordis resulting in ventricular flutter with delayed presentation to medical attention.

Case Report

A previously healthy 16-year-old male was hit in the center of his chest with a soccer ball from 20 feet away while playing competitive soccer. He immediately developed palpitations and stopped play for several minutes to recover. He returned to play despite persistent palpitations and made a penalty kick, but did not return to baseline. He subsequently developed diaphoresis, shortness of breath, lethargy, nausea, and emesis and then presented to the emergency department 8 h after the event.

The patient had no prior history of syncope, palpitations, exercise intolerance, or recent illness. His family history was negative for congenital heart disease, arrhythmias, cardiomyopathy, and sudden death. He denied alcohol and cocaine exposure, but reported marijuana use 3 weeks prior. He took a friend’s lisdexamfetamine pill that morning to concentrate on studying for a test.

Physical examination revealed an ill, pale-appearing patient with normal mental status and no external signs of trauma. He had “cyanotic lips” and felt cool and clammy. His heart rate (HR) was 225–240 beats per minute (bpm) and blood pressure (BP) was 160/100 mmHg with 100% oxygen saturation. An electrocardiogram (ECG) demonstrated ventricular flutter at 227 bpm, prompting administration of amiodarone 150 mg intravenous over 10 min [Figure 1]. Hemodynamic instability with a hypotension to 95/64 mmHg and altered mental status prompted rapid sedation and analgesia with midazolam and fentanyl and then external direct current cardioversion with 125 Joules. Sinus rhythm returned with a HR of 86 bpm and BP of 106/67 mmHg.

An amiodarone drip of 1 mg/min was initiated with transfer to the Connecticut Children’s Medical Center. Transthoracic echocardiography demonstrated normal ventricular systolic function, no pericardial effusion or regional wall motion abnormalities, and normal coronary artery origins. No further

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ectopy or tachyarrhythmia was appreciated by telemetry and subsequent Holter monitoring of amiodarone. A follow-up ECG demonstrated sinus arrhythmia with no ventricular preexcitation and normal QTc interval [Figure 2]. An exercise stress test demonstrated no arrhythmia at peak exercise or during recovery. The patient was permitted to return to sports with a chest protector. There were no further events or symptoms at more than 1 year of follow-up. He was strongly discouraged to use any stimulant medications.

**Discussion**

Commotio cordis is the second most common cause of sudden death in young male athletes after hypertrophic cardiomyopathy.[2,4] The most frequent arrhythmia described is ventricular fibrillation leading to sudden death on initial presentation.[5,6] Victims typically collapse upon impact; 20% of patients remain conscious for several seconds and very few present minutes or hours after impact.[7-9]

Increased chest wall compliance and altered repolarization patterns play a role in commotio cordis pathophysiology. Determinants of injury include precordial location, repolarization phase, projectile density, and impact velocity (30–50 miles/h). [4,6] The vulnerable phase of repolarization occurs 10–20 ms before peak of the T wave[2] [Figure 3]. Studies suggest that transmitted impact force causes myocardial ion channel upregulation and massive ion influx leading to arrhythmias such as ventricular fibrillation. Poor cerebral and myocardial perfusion cause immediate collapse. Dense-core, hard and small projectiles such as baseball, hockey and lacrosse balls have a higher incidence of ventricular fibrillation and increased morbidity compared to hollow objects.

Ventricular fibrillation is the most common mechanism of cardiac arrest following commotio cordis, although other arrhythmias including ventricular tachycardia, complete heart block, asystole, supraventricular tachycardia, and atrial fibrillation are reported.[5,9,10] Delayed presentation of commotio cordis is uncommon.[8] While our patient experienced palpitations immediately after impact, he did not demonstrate hemodynamic compromise until receiving amiodarone. This may be because ventricular flutter at a relatively slower rate of 225–240 bpm is more stable compared to ventricular fibrillation.

Stimulant medications have been attributed to sudden cardiac arrest.[11] Lisdexamfetamine has not been specifically reported with cardiac arrest; however, it carries an Food and Drug Administration warning regarding such.[12] It may potentiate arrhythmia mechanisms in patients with previously undiagnosed arrhythmias. A primary role of lisdexamfetamine in this patient’s arrhythmia cannot be excluded, but is unlikely with no risk factors and the temporal association of symptoms to chest impact.

The gold standard for primary prevention of commotio cordis is avoidance of chest wall impacts. Although chest protectors can lower the risk of ventricular fibrillation compared to impacts without chest protectors, the quality of the product varies and the use does not eliminate the risk of commotio cordis.[13] In
addition, softer equipment such as “safety baseballs” also lower the risk of ventricular fibrillation but fail to eliminate the risk.[14] Early access to automated external defibrillators improves survival rates, but survival remains as low as 25% despite resuscitation within 3 min.[15]

**Conclusion**

Commotio cordis is a rare, dreadful event following precordial impact with a high velocity projectile, typically resulting in ventricular fibrillation and immediate collapse. Rapid identification and resuscitation are the key to survival; nonetheless, the survival rate is low. Only 4% of projectile-related deaths are associated with air-filled balls. In this unique case, an air-filled ball induced an arrhythmia which allowed for preserved perfusion with delayed presentation to medical attention. Any palpitations following chest injury require immediate medical attention.

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**Conflicts of interest**
There are no conflicts of interest.

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