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ADVANCED

CASE REPORT: CLINICAL CASE

Cardiac Arrest in a Softball Player Following a Collision

Catching the Correct Diagnosis

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ABSTRACT

Commotio cordis is a rare cause of sudden cardiac arrest from blunt chest trauma; however, it is a diagnosis of exclusion. We present a case of sudden cardiac arrest in a collegiate athlete initially attributed to commotio cordis but in whom further history and workup revealed another rare condition. (**Level of Difficulty: Advanced.**) (J Am Coll Cardiol Case Rep 2022;4:1070–1073) © 2022 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

HISTORY OF PRESENTATION

A 21-year-old woman experienced a sudden cardiac arrest (SCA) during a collegiate softball game. While running to first base, she was tagged forcibly in the chest by a competitor and 5 seconds later collapsed face down. She was revived with manual compressions and a shock from an automated external defibrillator for an unspecified shockable rhythm after 4 minutes. She was taken to an outside hospital in a neurologically intact condition. Her temperature was 36.7 °C, blood pressure 138/89 mm Hg, respiratory rate 18 breaths/min, oxygen saturation 98% on room air. Physical examination was unrevealing. Her electrocardiogram (ECG) suggested left ventricular hypertrophy (LVH), first-degree atrioventricular block, and left anterior fascicular block. Transthoracic echocardiogram (TTE) by report showed a left ventricular ejection fraction (LVEF) of 66% and mild concentric LVH but was otherwise unremarkable. Her estimated pulmonary arterial systolic pressure was 15 mm Hg. Cardiac magnetic resonance suggested an LVEF of 48%, mildly dilated LV (end-diastolic volume index 114 mL/m^2), normal right ventricle and valves, and no late

LEARNING OBJECTIVES

- To generate a differential diagnosis of sudden cardiac arrest in young athletes and recognize specific atypical features that argue against exercise-induced cardiac remodeling and warrant further evaluation.
- To review the mechanisms, pathophysiology, and workup of suspected commotio cordis.
- To describe the malignant nature of anomalous left coronary artery from the pulmonary artery.

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gadolinium enhancement. She was discharged with an implantable loop recorder and a diagnosis of suspected commotio cordis. She presented to our institution for evaluation before returning to play.

MEDICAL HISTORY

The patient had experienced exercise-induced chest tightness since childhood. This was diagnosed as exercise-induced asthma and treated with inhaled beta-agonists, which did not improve her symptoms. Two weeks before the SCA the patient had a non-COVID-19 upper respiratory infection treated with azithromycin. She had no prior syncope or family history of SCA.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis for SCA in this athlete included commotio cordis, postviral myocarditis, arrhythmogenic cardiomyopathy, hypertrophic cardiomyopathy, long QT syndrome, Brugada syndrome, Wolff-Parkinson-White syndrome, and anomalous coronary arteries.

INVESTIGATIONS

Repeated ECG at our institution showed delayed R-wave progression, LVH, and left anterior fascicular block (**Figure 1**). The result of a repeated TTE was similar to the prior report, although LVEF by visual estimation was 50% to 55%, similar to that seen on cardiac magnetic resonance. Genetic testing for long QT syndrome was unrevealing. She completed 12 minutes (13 METs) of a standard Bruce protocol stress echocardiogram with appropriate hemodynamic response. The test was stopped because of chest tightness and increasing ventricular ectopy. She had premature ventricular contractions, ventricular couplets, and mid-distal anteroseptal hypokinesis with stress (Video 1). A subsequent coronary computed tomographic angiogram (CCTA) showed an anomalous left coronary artery (LCA) from the pulmonary artery (ALCAPA) with a large right coronary artery (RCA) supplying right-to-left collaterals and retrograde filling of the left anterior descending artery (**Figure 2**).

MANAGEMENT

After the CCTA, a discussion including congenital cardiology and cardiothoracic surgery favored attempting translocation of the LCA onto the aorta. She underwent ALCAPA repair with left coronary button translocation from the pulmonary artery (PA) to the aorta by use of a button tubularization technique. She was discharged without complications.

DISCUSSION

This case highlights the need for a thorough evaluation of the causes of SCA in athletes before they return to play and the importance of differentiating exercise-induced cardiac remodeling from pathologic conditions. In this case, the patient's LV dilation and borderline LVEF would be more characteristic of endurance athletes and thus suggest pathologic changes.[1] Therefore, a stress echocardiogram was performed to evaluate for augmentation in LVEF, which, if present, would favor exercise-induced cardiac remodeling. Furthermore, although isolated LVH can be a normal ECG finding in athletes, the presence of left axis deviation is considered a borderline finding.[2] Finally, exertional chest pain and dyspnea in an otherwise conditioned athlete are abnormal according to the American Heart Association 12-element screening recommendation for competitive athletes and should have prompted further workup such as an ECG and echocardiogram that might have identified the aforementioned abnormalities.[3]

Commotio cordis is a cause of SCA in athletes defined by ventricular fibrillation and SCA from blunt, nonpenetrating trauma, often from small objects traveling at high speeds such as a ball or a puck. The impact often occurs directly over the heart and occurs within 10 to 20 milliseconds on the upstroke of the T-wave; importantly, the affected individual must not have pre-existing cardiac disease or resulting damage to the ribs, sternum, or heart.[4] A comprehensive evaluation for cardiac pathology and susceptibility to arrhythmias should be performed (Class I; Level of Evidence: B, per the 2015 American Heart Association/American College of Cardiology Scientific Statement), and if no underlying pathologic conditions are identified, then individuals can return to play, given no evidence of increased risk for subsequent arrhythmic events (Class IIa; Level of Evidence: C).[5] If underlying cardiac disease is absent, implantable cardioverter defibrillators are not recommended.[5]

Although the diagnosis of commotio cordis is rather obvious in sporting events, unusual circumstances such as this require a thorough diagnostic workup. Though possible, SCA from a slow, softer object, such as a softball mitt, is atypical. Therefore, in this athlete with exertional symptoms and pathologic findings on

ABBREVIATIONS AND ACRONYMS

ALCAPA = anomalous left coronary artery from the pulmonary artery

CCTA = coronary computed tomographic angiogram

ECG = electrocardiogram

LCA = left coronary artery

LVEF = left ventricular ejection fraction

LVH = left ventricular hypertrophy

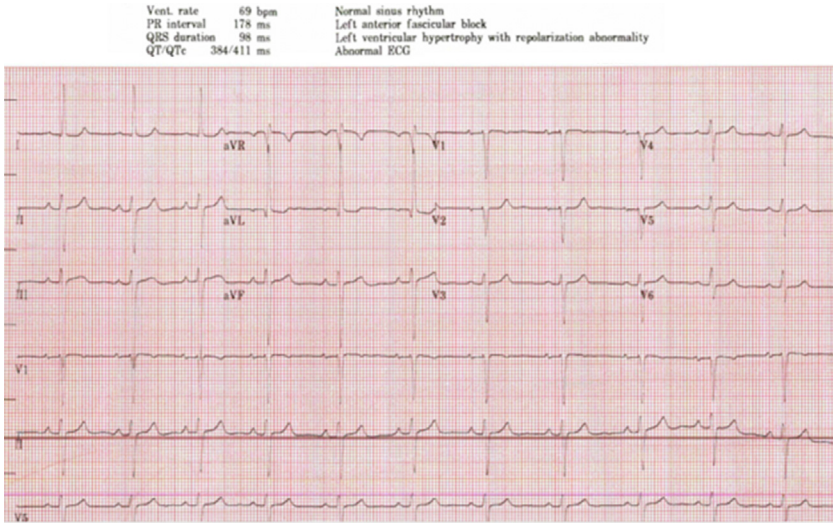
PA = pulmonary artery

RCA = right coronary artery

SCA = sudden cardiac arrest

TTE = transthoracic echocardiogram

FIGURE 1 Initial Outpatient Electrocardiogram

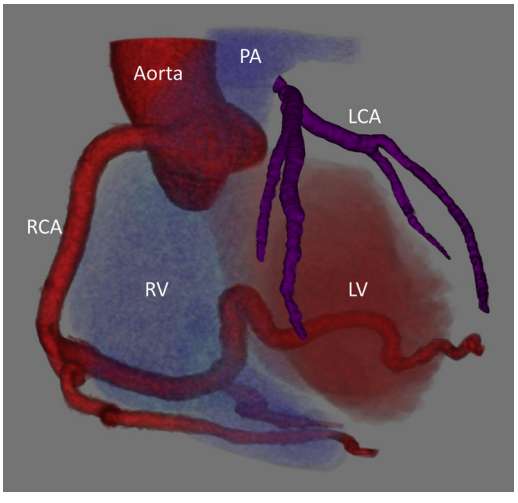


Initial outpatient electrocardiogram after cardiac arrest demonstrating delayed R-wave progression, left ventricular hypertrophy, and left anterior fascicular block.

ECG and TTE, a coronary evaluation by CCTA showed the true diagnosis of ALCAPA.

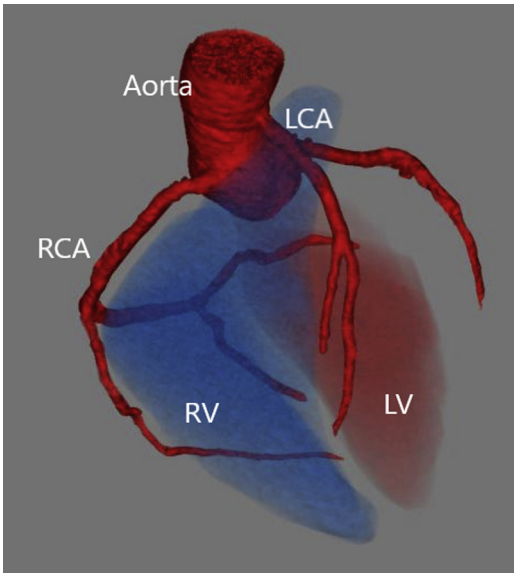
ALCAPA is a rare coronary anomaly occurring in 1 in 300,000 births.[6] Although ALCAPA can be well tolerated in the prenatal and early neonatal period because of similar aortic and PA pressures, its hemodynamic effects arise as the PA pressure decreases after birth. Consequently, antegrade flow into the LCA decreases, and the higher-pressure, fully oxygenated RCA flow leads to retrograde flow through the LCA into the lower-pressure PA, bypassing the higher-pressure coronary microcirculation. This "coronary steal" leads to subsequent LV overload, ischemia, and heart failure. The degree of collateralization that develops between the RCA and the LCA determines the extent of ischemia, age at presentation, and prognosis. In individuals without collaterals the infant type develops, characterized by heart failure, ischemic mitral regurgitation, and death in 90% in the first few months of life if unrepaired.[6,7] Those with well-developed collaterals present with the adult type. Chronic subendocardial ischemia is present and, as was the case in this athlete with superimposed chest wall trauma, can lead to ischemic

FIGURE 2 Initial 3-Dimensional Reconstruction of Native Coronary Anatomy



3-dimensional reconstruction demonstrating anomalous left coronary artery **(purple vessel)** from the pulmonary artery **(blue vessel)**. LCA = left coronary artery; LV = left ventricle; PA = pulmonary artery; RCA = right coronary artery; RV = right ventricle.

FIGURE 3 Postoperative 3-Dimensional Reconstruction of Coronary Anatomy



3-dimensional reconstruction demonstrating re-implanted left coronary artery onto the aorta. LCA = left coronary artery; LV = left ventricle; RCA = right coronary artery; RV = right ventricle.

cardiomyopathy, malignant arrhythmias, and sudden death in adolescence or early adulthood.[6,7] The combination of chronic ischemia and chest wall trauma was the likely mechanism of this SCA. The goal of surgical correction is to restore a 2-coronary-artery system and is usually done by coronary button translocation from the PA to the aorta. In rare cases, a baffle from the PA wall to tunnel the LCA through the main PA into the left coronary ostia, or coronary artery bypass grafting with ligation of the ALCAPA, is performed.

FOLLOW-UP

A stress echocardiogram showed improvement in baseline LVEF, normal augmentation with improved exercise capacity, and no chest pain or arrhythmias. Repeated CCTA showed the LCA attached to the left side of the aorta and a smaller RCA (**Figure 3**). Interrogation of the implantable loop recorder yielded unremarkable results. Given these findings, the woman enrolled in cardiac rehabilitation with plans to return to play.

CONCLUSIONS

This case highlights the importance of a broad differential diagnosis and complete evaluation in cases of SCA among athletes, especially when commotio cordis is suspected based on the initial event. This case offers the opportunity to review commotio cordis and ALCAPA, particularly in athletes.

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KEY WORDS congenital heart defect, coronary vessel anomaly, exercise, sports cardiology, sudden cardiac arrest, ventricular fibrillation

APPENDIX For a supplemental video, please see the online version of this article.