

CASE REPORT

BEGINNER

CLINICAL CASE

Commotio Cordis

Indeed?



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ABSTRACT

We discuss an unusual case of a patient who initially presented with apparent commotio cordis but returned several months later with spontaneous wide complex tachycardia. This case highlights the importance of investigation of survivors of commotio cordis, especially if atypical, to determine if there is underlying cardiac disease. (**Level of Difficulty: Beginner.**) (J Am Coll Cardiol Case Rep 2019;1:597-601) © 2019 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 35-year-old professional soccer player was struck in the chest by a ball from a close distance while playing in a practice game. He experienced dizziness but remained conscious for several minutes before he collapsed. A staff member immediately started cardiopulmonary resuscitation until an emergency medical team arrived. The first documented rhythm

was sustained monomorphic ventricular tachycardia (VT), which deteriorated to ventricular fibrillation (VF) (**Figure 1**). Four electrical shocks were delivered with restoration of sinus rhythm and full neurological recovery.

On admission to the intensive cardiac care unit, the patient was alert and oriented. His physical examination and vital signs were normal.

MEDICAL HISTORY

The patient is a professional healthy athlete, without any significant medical history and no family history of cardiac disease or sudden cardiac death. A routine echocardiography performed 2 years earlier was within normal limits.

DIFFERENTIAL DIAGNOSIS

The differential diagnoses are commotio cordis, contusio cordis, and other causes of sudden cardiac death among athletes, including hypertrophic cardiomyopathy, arrhythmogenic right ventricular dysplasia, ischemic heart disease, anomalous coronaries, myocarditis, and inherited arrhythmia

LEARNING OBJECTIVES

- To understand the importance of performing a comprehensive evaluation for underlying cardiac pathology in survivors of apparent commotio cordis, including CMR and EPS if a scar exists.
- To recognize an additional possible mechanism for atypical commotio cordis, in which a blunt trauma to the chest triggers ventricular arrhythmia within an existing myocardial scar. In patients presenting with this type of mechanism, ICD implantation may be required.

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Informed consent was obtained for this case.

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ABBREVIATIONS AND ACRONYMS

CMR = cardiac magnetic resonance

EPS = electrophysiological study

ICD = implantable cardioverter-defibrillator

LGE = late gadolinium enhancement

LV = left ventricular

VF = ventricular fibrillation

VT = ventricular tachycardia

disorders (e.g., congenital long QT syndrome, Brugada syndrome, Wolff-Parkinson-White syndrome).

INVESTIGATIONS

An electrocardiogram showed sinus rhythm, incomplete right bundle branch block, and ST-T changes in the inferior and precordial leads that normalized in the following days. Results of blood tests revealed elevated high-sensitivity cardiac troponin-T of 283 ng/l (normal range, 0 to 14 ng/l). Transthoracic echocardiography demonstrated moderate left ventricular (LV) systolic dysfunction with an ejection fraction of 40%, minimal segmental variation, mildly hypokinetic right ventricular apex, and minimal pericardial effusion around the right ventricular apex. Coronary angiography revealed normal coronary arteries.

Cardiac magnetic resonance (CMR) performed 5 days later revealed mild biventricular dilatation, normal LV wall thickness with mild systolic dysfunction (LV ejection fraction 49%), and inferolateral wall hypokinesis. The right ventricle was mildly hypokinetic, with no evidence of dyskinesia or aneurysm. CMR criteria for arrhythmogenic right ventricular dysplasia were not fulfilled. Confluent subepicardial late gadolinium enhancement (LGE) was noted in the lateral and inferolateral walls of the

left ventricle, corresponding with the wall motion abnormality.

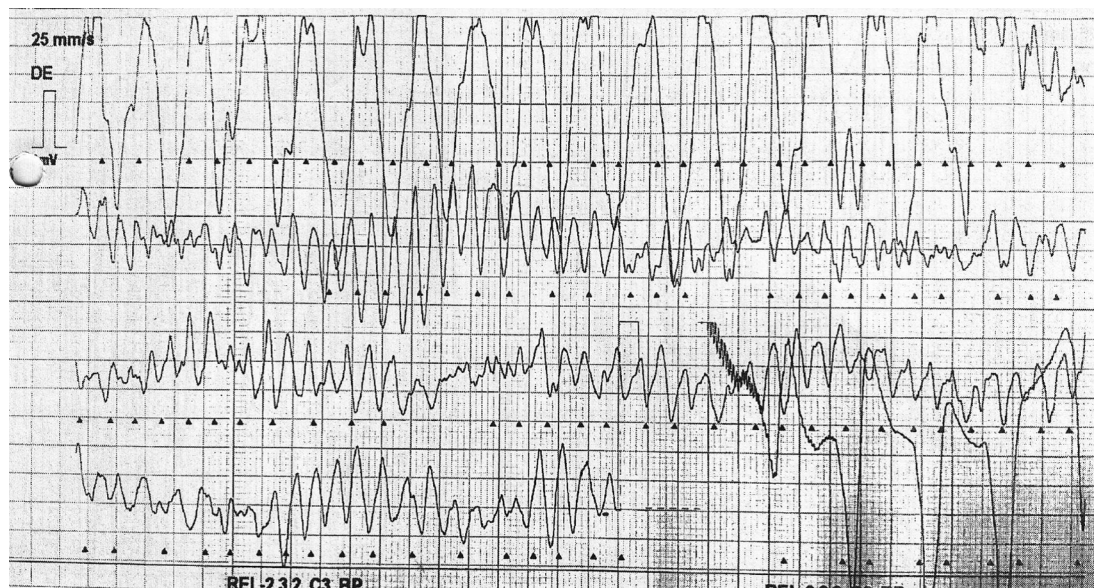
MANAGEMENT

During hospitalization, the patient remained asymptomatic with no further documented arrhythmias. Implantable cardioverter-defibrillator (ICD) implantation was considered but was ultimately not performed due to the apparent clinical diagnosis of commotio cordis, despite the evidence of LGE. The patient was instructed to avoid intense exercise and refrain from competitive sports. Upon discharge, he began a cardiac rehabilitation program.

HISTORY OF PRESENTATION-CONTINUED

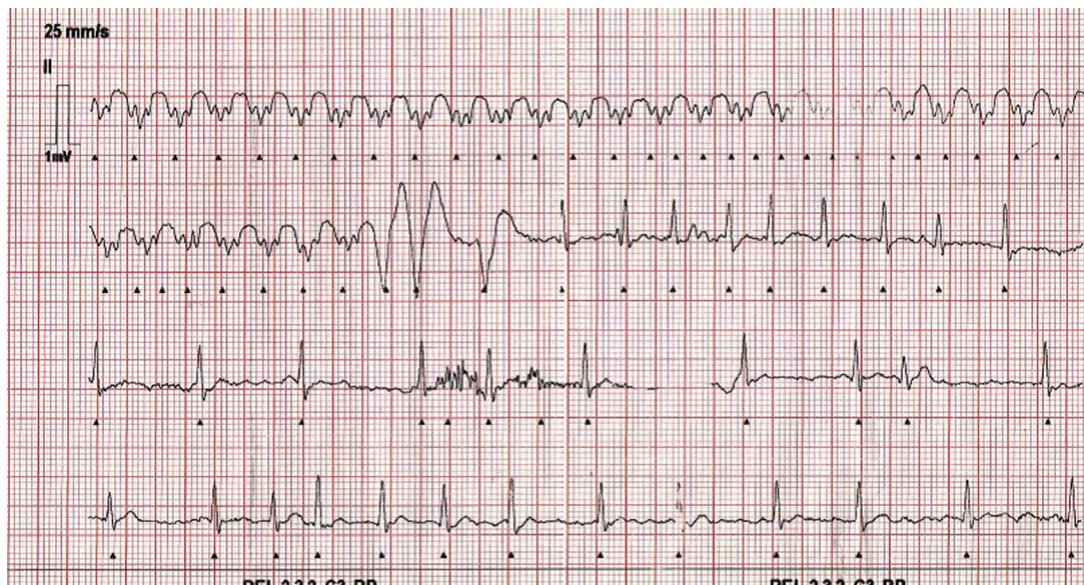
Nine months after the initial presentation, while the patient was walking on the soccer field as an assistant coach, he suddenly felt palpitations, along with weakness and dizziness. An emergency medical team was immediately called, documenting regular wide complex tachycardia of 230 beats/min that terminated after 3 consecutive rapid premature ventricular contractions, followed by atrial fibrillation and spontaneous conversion to sinus rhythm (Figure 2). A 12-lead electrocardiogram was normal, and results of blood tests revealed a mildly elevated high-sensitivity cardiac troponin T level of 36 ng/l.

FIGURE 1 Initial Cardiac Rhythm Upon Arrival of the Emergency Medical Team



Sustained monomorphic ventricular tachycardia that deteriorated into ventricular fibrillation.

FIGURE 2 Initial Electrocardiogram of the Second Event



Transthoracic echocardiography showed normal biventricular systolic function with mild hypokinesis of the posterior wall.

Given a wide complex tachycardia and the patient's history, an electrophysiological study (EPS) was performed, with induction of sustained monomorphic VT by double extrastimuli delivered from the right ventricular apex at a basic cycle length of 500 ms and 2 extrastimuli of 230 and 220 ms, terminated by right ventricular overdrive pacing. The arrhythmia was consistent with the clinical arrhythmia documented by the emergency medical team (Figure 3). Subsequently, a dual chamber ICD was implanted concomitant with beta-blocker treatment. During 9 months of follow-up, he remained asymptomatic with no evidence of further arrhythmias.

DISCUSSION

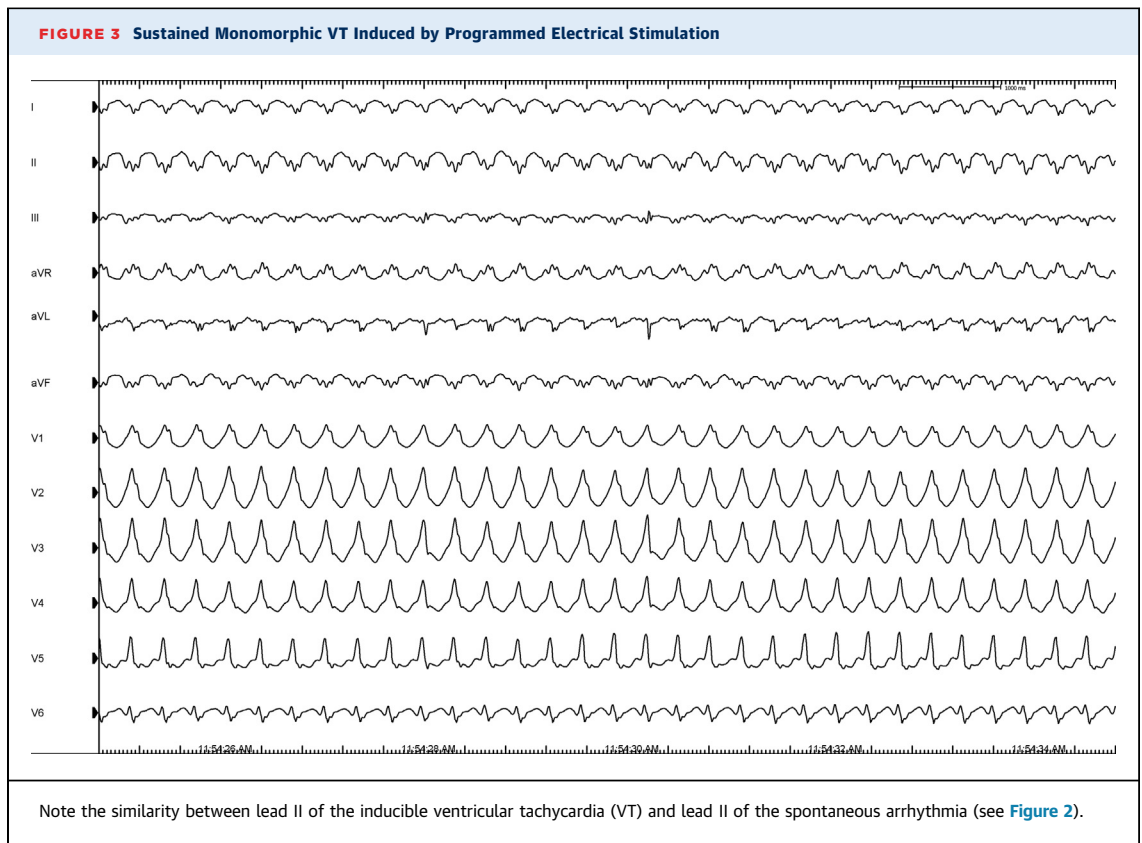
The rare phenomenon, in which a nonpenetrating blow to the chest results in sudden cardiac death in the absence of cardiac damage, is known as commotio cordis. It is a significant cause of sudden cardiac death in athletes, mainly in adolescent male subjects (1).

An experimental model of commotio cordis has shown that the typical arrhythmia induced by a chest

blow is VF. Self-limited VF and transient heart block were also observed. Transient heart block is typically seen with higher velocity impacts that cause structural damage (2). There are several critical variables in the induction of the arrhythmia, including the location, velocity, and shape of the impact object. The most important variable is the timing of impact relative to the cardiac cycle. Only impacts occurring during the 20- to 40-ms window on the upslope of the T-wave cause VF (1,2). In a suggested mechanism, the adenosine triphosphate-sensitive potassium channel is activated by the chest blow, causing alteration in repolarization and eventually VF. It is debatable if there is a component of individual susceptibility to commotio cordis (3).

Commotio cordis is to be distinguished from contusio cordis, in which a significant blunt chest trauma causes structural cardiac damage such as contusion of myocardial muscle, rupture of a cardiac chamber, or disruption of a heart valve, mostly due to motor vehicle accidents.

In the current case, there was no significant structural cardiac or chest injury. The transient ventricular dysfunction could be consistent with post-resuscitation myocardial dysfunction that develops after successful resuscitation from cardiac arrest as previously described. Furthermore, CMR performed a



few days later did not show any evidence of acute myocardial changes that represent cardiac contusion, such as myocardial edema or focal delayed hyper-enhancement (4).

In this unique case, the initial documented arrhythmia was sustained monomorphic VT that deteriorated into VF ([Figure 1](#)), followed by a second episode of spontaneous sustained monomorphic VT 9 months later ([Figure 2](#)). The negative QRS complexes in the inferior and lateral leads during the VT, are indicative of the VT origin, which is consistent with the areas of LGE seen in the CMR. Given a monomorphic VT, inducibility by ventricular extrastimuli in EPS, and evidence of LGE, it seems that the mechanism of arrhythmia in the current case is re-entrant arrhythmia within an old myocardial scar, rather than a typical commotio cordis.

Notably, there are also several clinical features that are not consistent with typical commotio cordis. The patient was 35 years old, which is older than the common age range in commotio cordis (1). Furthermore, whereas the classic presentation in commotio cordis is immediate collapse, this patient remained conscious for several minutes before

collapsing, which is consistent with the presenting arrhythmia that was not VF. Ultimately, although first presented with a ventricular arrhythmia following a direct hit to the chest, he experienced a second spontaneous ventricular arrhythmia several months later (5).

Because the initial documented arrhythmia was sustained monomorphic VT that deteriorated to VF, the authors hypothesize that the mechanism of the arrhythmia in this case is a re-entrant VT originating from an old myocarditis-related scar, triggered by a blunt hit to the chest.

FOLLOW-UP

The patient had no further arrhythmias over the 1-year follow-up.

CONCLUSIONS

The current case shows another possible mechanism for the phenomenon in which a nonpenetrating blow to the chest results in sudden cardiac arrest. The mechanism involved a re-entrant VT originating from an old myocardial scar, triggered by a blunt hit to the chest.

It also highlights the importance of performing a comprehensive evaluation for underlying cardiac pathology in survivors of commotio cordis, especially if atypical, including EPS if there are signs of myocardial scar. ICD implantation should be considered in these cases.

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