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CRITICAL CARE AND RESUSCITATION

CASE REPORT: CLINICAL CASE

# Blunt Chest Wall Trauma Leading to Sudden Cardiac Arrest

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## ABSTRACT

A 54-year-old hockey player survived sudden cardiac arrest after a chest slapshot, receiving immediate resuscitation and defibrillation of ventricular fibrillation. Examinations revealed chest trauma and subclinical single-vessel disease; a coronary dissection could not be ruled out. The patient recovered without complications, underscoring the importance of rescue equipment in sports facilities. (JACC Case Rep. 2024;29:102504) © 2024 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 54-year-old man was admitted to the emergency department after surviving sudden cardiac arrest (SCA). While playing ice hockey with his senior team, he received a slapshot to the chest without wearing a chest protector. Immediately, he lost consciousness and collapsed onto the ice. The hockey team promptly initiated cardiopulmonary resuscitation and used an automated external defibrillator (AED). Ventricular fibrillation (VF) was detected, and the patient received a shock, restoring sinus rhythm (SR). Upon the arrival of the rescue team, the patient was awake and in stable hemodynamic condition. The clinical examination in the emergency department revealed a rectangular red skin mark (from the ice hockey puck) on the precordial chest wall (**Figure 1**). The patient's blood pressure was 145/95 mm Hg, heart rate was 82 beats/min, and oxygen saturation was 94% with 2 L of oxygen given over the nose. He was drowsy, experienced retrograde amnesia, and complained of chest discomfort after resuscitation.

### LEARNING OBJECTIVES

- To be able to recognize the mechanisms behind commotio cordis and its potential to induce ventricular fibrillation.
- To consider coronary artery disease in athletes, even among those with a seemingly low cardiovascular risk profile.
- To identify the importance of prompt emergency response and widespread availability of AEDs in sports facilities to enhance outcomes during sudden cardiac events.

## PAST MEDICAL HISTORY

Apart from occasional cigarette smoking, he had no other cardiovascular risk factors. Reporting no incidents of angina during sports, he never underwent a cardiac stress test or a resting electrocardiogram (ECG). He regularly participated in ice hockey games at a local club.

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The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the Author Center.

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**ABBREVIATIONS AND ACRONYMS**

**AED** = automated external defibrillator

**CAD** = coronary artery disease

**ECG** = electrocardiogram

**LAD** = left anterior descending artery

**SCA** = sudden cardiac arrest

**SCD** = sudden cardiac death

**TTE** = transthoracic echocardiogram

**VF** = ventricular fibrillation

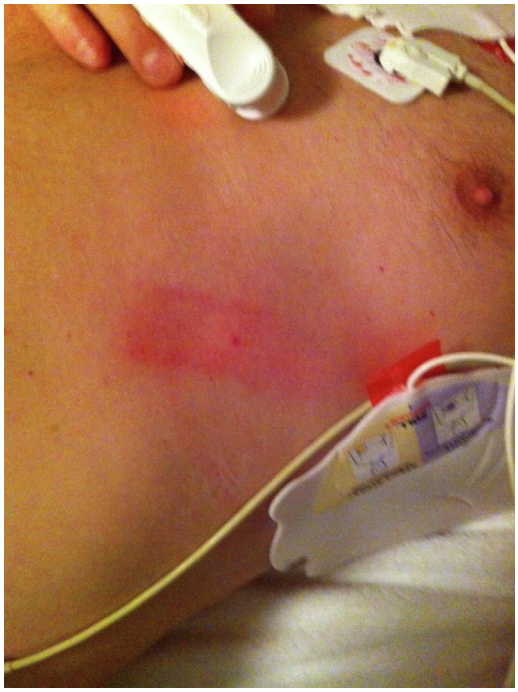
## DIFFERENTIAL DIAGNOSIS

The differential diagnosis of sudden cardiac death (SCD) and SCA during sports activity in middle-aged sportsmen includes acute coronary syndromes with myocardial infarction caused by coronary artery disease (CAD), cardiomyopathies such as hypertrophic cardiomyopathy, arrhythmic disorders or channelopathies, or blunt chest trauma leading to commotio cordis.

## INVESTIGATIONS

A pneumothorax and rib fractures were ruled out. The neurologic examination yielded normal results. During the stay in the intensive care unit, 2 brief episodes of ventricular runs were recorded. The ECG can be interpreted as indicating a global ischemic injury after cardiac arrest, potentially affecting distal branches of the left bundle and Purkinje fibers. The S-wave notch in $V_2$, the presence of an S-wave in $V_6$, and the QRS "J-wave" slurring in leads I and aVL all suggest an injury of the left bundle branch. So far, there have been no suspicions of channelopathies but rather suspicions of non-ST-segment elevation myocardial infarction (**Figure 2**). The high-sensitivity troponin T levels were elevated at 0.143 μg/L (normal: <0.014 μg/L) and increased to 0.510 μg/L in the following hours. A subsequently conducted coronary angiography revealed a single-vessel disease with multiple serial lesions in the left anterior descending artery (LAD) (**Figure 3**), including a proximal stenosis (90%) and a distal lesion of 99% where dissection could not be ruled out. A congenital coronary artery anomaly was excluded. The ECG for days after percutaneous coronary intervention (**Figure 4**) showed a completely different picture, indicating normal left bundle conduction. The R-wave notch in $V_3$ hints at a nonspecific conduction abnormality, potentially midmyocardial or midseptal, which correlates with the apical akinesia noted on the transthoracic echocardiogram (TTE) at the same time. The TTE did not reveal any signs of cardiomyopathies.

**FIGURE 1 Clinical Examination**



Rectangular red mark on the chest wall caused by the impact of an ice hockey puck.

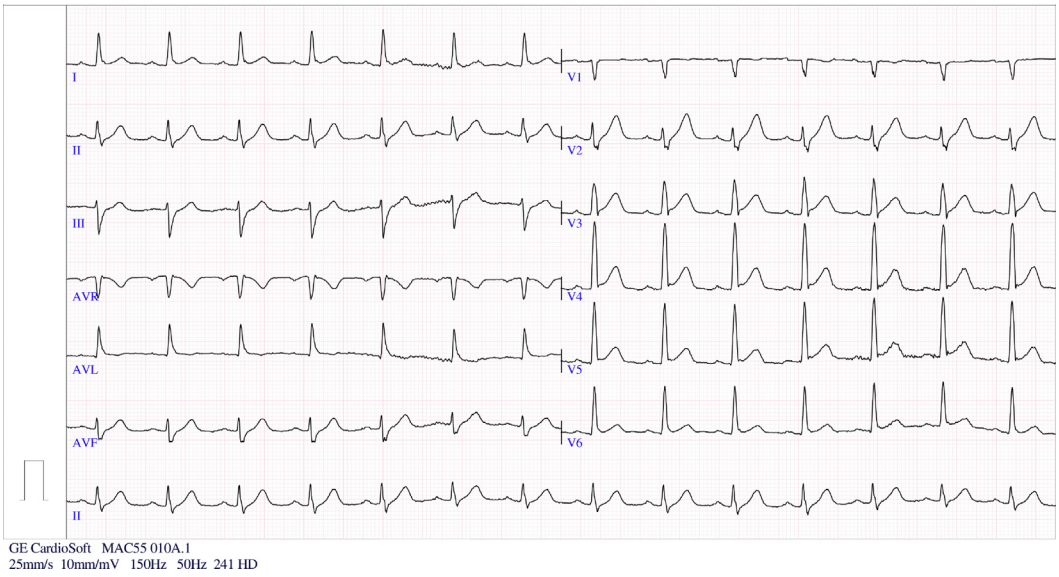
## MANAGEMENT

The proximal lesion of the LAD was successfully treated with stenting. Fractional flow reserve, which is commonly used today for evaluating such lesions, was not performed. However, attempts to perform percutaneous coronary intervention on the distal doubtful dissected part were unsuccessful. The subsequent clinical course of the patient remained uneventful. A cardiac computed tomography angiography or a follow-up angiography was not initiated. Upon discharge, the patient was prescribed dual antiplatelet therapy (aspirin and clopidogrel), β-blocker, and statin therapy.

## DISCUSSION

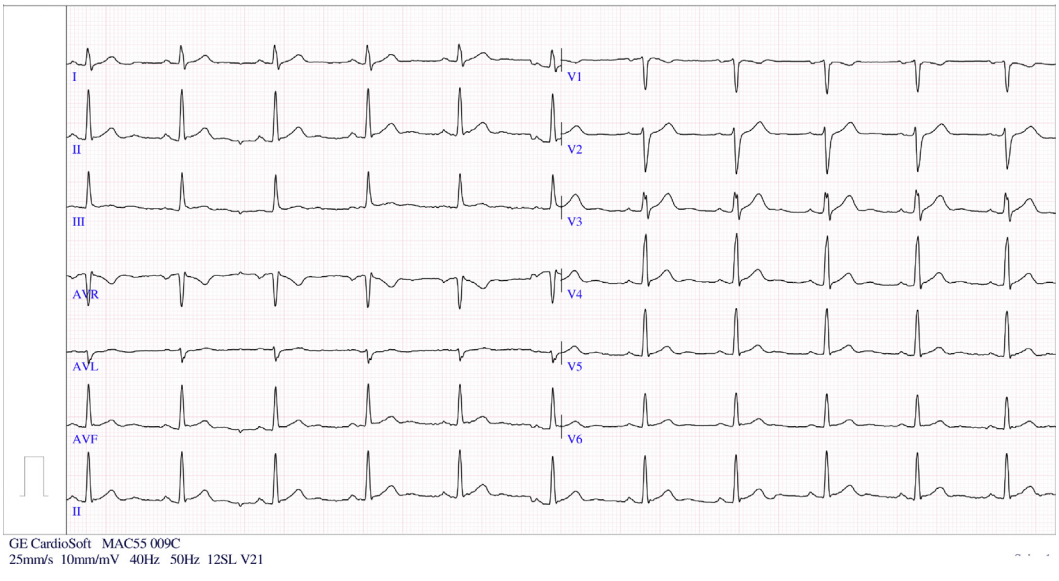
This case raises several questions. First, the patient did not use a chest protector to safeguard the heart against blunt trauma. Instances of heart concussion (commotio cordis) leading to SCA and SCD have been documented in children and adolescent ice hockey players who lacked adequate chest protection.[1] Research into the mechanisms of commotio cordis and SCD during sports involved

**FIGURE 2** **Electrocardiogram on Emergency Department Admission**



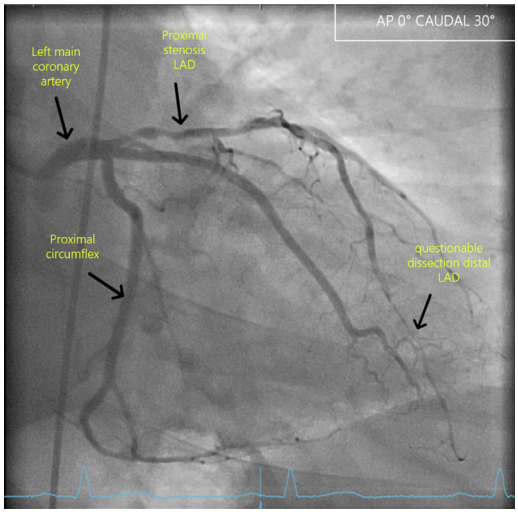

Sinus rhythm, left axis deviation, heart rate of 100 beats/min, nonsignificantly elevated ST-segment in $V_3$-$V_6$, S-wave notch in $V_2$, and QRS "J-wave" slurring in leads I and aVL.

**FIGURE 3** **Electrocardiogram 4 Days After Percutaneous Coronary Intervention**




Sinus rhythm, heart rate of 70 beats/min, normal axis, notch in $V_3$, and normal repolarization.

**FIGURE 4 Left Coronary Angiogram**



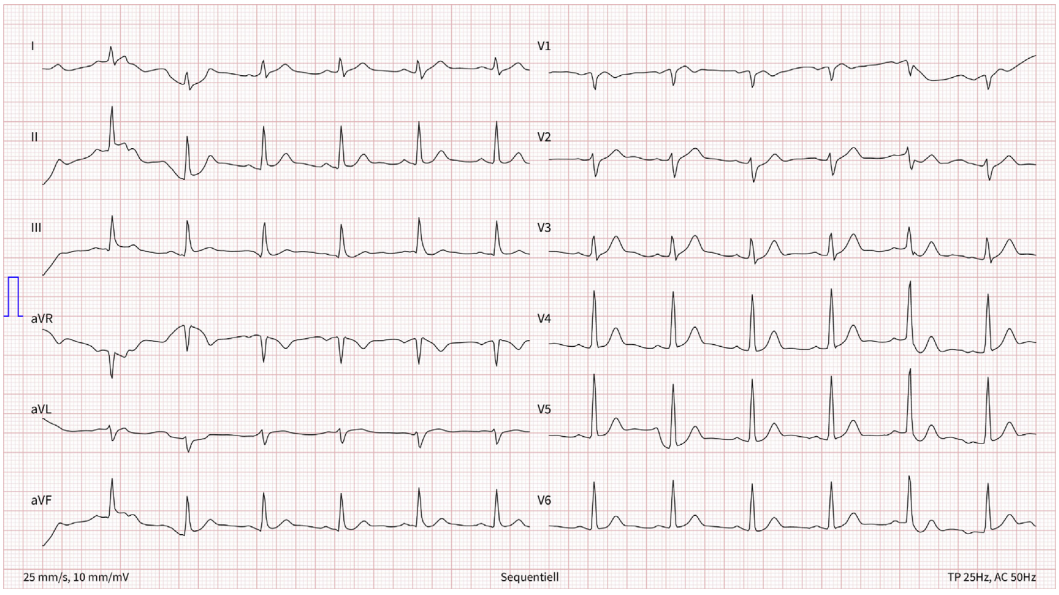

Tight proximal stenosis of the LAD and questionable coronary artery dissection of the distal part of LAD. LAD = left anterior descending artery.

experimental models using ice hockey pucks or baseballs on anesthetized juvenile pigs. These studies demonstrated that VF induction is possible during chest wall impacts without underlying structural cardiovascular disease. VF induction occurs within a vulnerable 20-ms window before the T-wave peak on the ECG. Determinants such as the impact's location, velocity, and object hardness play critical roles. The rapid rise in left ventricle pressure following a chest impact activates ion channels via mechanoelectric coupling, resulting in nonuniform myocardial depolarization that triggers VF.[2,3] This contributed to the development of current chest wall protectors in ice hockey.

However, the effectiveness of these protectors in preventing commotio cordis is questionable, as illustrated by SCA experienced by football player Damar Hamlin despite wearing a chest protector. Although common in young athletes, such events are unusual in adult players, ranking third (3%) in occurrence after congenital coronary artery anomalies (17%) and hypertrophic cardiomyopathy (36%) for sudden deaths in young competitive athletes. The highest susceptibility is found in children and adolescents (mean age: 15 ± 9 years), primarily male athletes engaged in sports like hockey, baseball, lacrosse, or football.[4,5]

Isolated coronary artery injury is rare in blunt chest traumas, with the LAD most affected (76% of cases). Causes include rupture, compression, plaque disruption, or artery dissection.

**FIGURE 5 Electrocardiogram 10 Years Later**




Motion artifact in the first 2 beats. Sinus rhythm, heart rate of 75 beats/min, normal axis, and normal conduction.

Acute dissection most commonly mimics acute coronary syndrome, requiring diagnosis through coronary angiography.[6,7] In our patient, the possibility of coronary artery dissection could neither be ruled out nor proven. Published cases with coronary artery dissection after blunt chest traumas typically involve massive impacts from motorcycle and car collisions, falls, or brawls resulting in multiple body injuries.[8]

Our patient had never experienced angina pectoris or undergone a myocardial stress test, but he had significant CAD, with multiple serial lesions in the LAD, potentially inducing silent ischemia during physical activity. The clinical relevance of silent ischemia in this situation for VF induction and SCD remains questionable.[9] CAD has been identified as the most common cause of SCA in middle-aged senior athletes. Almost one-fifth of individuals with a low cardiovascular risk profile harbor significant occult CAD.[10] Given the patient's immediate collapse after the blunt impact to the cardiac region (**Figure 1**), without experiencing any chest pain before, commotio cordis appears to be the dominant mechanism for the fatal arrhythmia.

## FOLLOW-UP

Although there were no additional physical or anatomic tests (such as stress imaging or cardiac computed tomography angiography) regarding clinical implications, the clinical course of the 54-year-old individual remained uneventful over the subsequent decade, without symptomatic arrhythmias or suspicious syncope characteristic of channelopathies.

Cardiovascular risk factors are well managed, with normal blood pressure and the absence of diabetes requiring medication. The patient is prescribed statin/ezetimibe and aspirin. His physical fitness is excellent, and he has resumed participation in senior ice hockey. Both the ECG (**Figure 5**) and the treadmill test yielded normal results, and the TTE revealed normal findings with no regional wall motion abnormalities, except for a negligible sclerosis of the aortic valve.

## CONCLUSIONS

Blunt chest trauma can lead to commotio cordis, including VF. Although well documented in children and adolescents engaged in competitive sports like ice hockey and baseball, it is considered uncommon in adults. An experimental model has elucidated the underlying mechanism, significantly affecting the development of the protective gear used in these sports today. The presented case raises the question of whether a chest impact from an ice hockey puck on the unprotected chest of a 54-year-old man led to commotio cordis with VF as the sole mechanism. The presence of previously unknown significant CAD with serial lesions in the LAD, potentially involving coronary artery dissection and silent ischemia, suggests more than coincidental factors. All these mechanisms are potential contributors to VF induction. The prompt efforts of the team players to resuscitate the patient using a locally installed AED are commendable. Early cardiopulmonary resuscitation is pivotal, with a 40% survival rate within 3 minutes, contrasting with a mere 2% survival rate after 3 minutes.[8] The increased availability of AEDs and heightened awareness of commotio cordis have contributed to improved survival trends. The excellent 10-year follow-up findings for our patient highlight the importance of well-managed cardiovascular risk factors.

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The authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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**KEY WORDS** **blunt chest trauma by ice hockey puck, commotio cordis, coronary artery dissection, silent coronary artery disease, sudden cardiac death/arrest during sports**


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