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Commotio cordis (cardiac concussion) in a child. A case report

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ABSTRACT

Commotio cordis or cardiac concussion is a rare and fatal mechano-electric arrhythmogenic syndrome. It is the second most common cause of sudden cardiac death in young athletes. It is most commonly associated with a sports-related injury, wherein, there is a high-velocity impact between a projectile and the precordium, causing arrhythmia that leads to the immediate death of the individual without cardiac resuscitation.

On autopsy, the heart is structurally normal.

With increasing awareness of this condition and community training in cardiopulmonary resuscitation, survival rates have been improving.

The objective of this study is to describe the case of a patient who arrived at our hospital with *commotio cordis* and his course, emphasizing the importance of prevention and training of the population in cardiopulmonary resuscitation techniques and the use of the automated external defibrillator for the survival of patients suffering from *commotio cordis*.

Key words: commotio cordis*; pediatrics; arrhythmias; sports.*

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INTRODUCTION

Commotio cordis (CC) is a fatal mechano-electric syndrome occurring in a normal heart; it is a ventricular fibrillation caused by a blunt trauma to the chest.[1,2] The English meaning of the term, "agitation of the heart," describes the mechanism whereby significant distortion of the myocardium creates enough mechanical energy to cause inappropriate depolarization, resulting in an unstable dysrhythmia.[2,3]

It is a major cause of sudden death in young athletes.

CASE REPORT

This was a healthy, 11-year-old boy who suffered a blunt trauma to the chest due to the impact of a soccer ball in the precordial region while playing sports, with sudden loss of consciousness. He was assisted by a teacher, who verified the boy was in cardiopulmonary arrest and performed basic cardiopulmonary resuscitation (CPR) without the use of an automated external defibrillator (AED) because it was not readily available on site.

The patient was transferred to a hospital, where advanced CPR maneuvers were continued; defibrillation was performed and the cardiopulmonary arrest was reversed after 45 minutes. He was transferred to a pediatric intensive care unit (PICU), where he was admitted in an unstable condition with cardiogenic shock. He required high-dose vasoactive drips and mechanical ventilation (MV).

He had elevated cardiac enzymes (*Table 1*); the electrocardiogram showed sinus rhythm; the echocardiogram did not show structural heart disease with a 26% fractional shortening without pericardial effusion.

The computed tomography of the brain, cervical spine, chest, abdomen, and pelvis were normal. Improvement was observed in the cardiac control at 48 hours.

The electroencephalogram (EEG) performed on day 2 of hospitalization showed medium-voltage brain activity; and improvement was observed on day 7 of hospitalization.

As of day 10 of hospitalization, in terms of neurological condition, the child did not require analgesia or sedation; he presented with motor involvement, left brachiocrural hemiparesis, generalized hypotonia, and weak cough reflex; therefore, a tracheostomy was performed to improve his comfort and advance with motor and functional rehabilitation.

On day 15, a brain magnetic resonance imaging with gradient echo and diffusion sequences was performed; it showed an increased bilateral and symmetrical signal intensity of both globi pallidi in T2 and FLAIR sequences, with restriction images in diffusion sequences compatible with hypoxic-ischemic lesion (*Figure 1*).

The patient was weaned from ventilation and positive pressure was discontinued at 35 days of hospitalization.

He was transferred to the pediatric ward without oxygen requirement. He was wakeful, reactive, connected, with left upper limb monoparesis, normal gait, and good verbal communication, with persistent dysarthria.

He was receiving enteral feeding by nasogastric tube and, on day 42 of hospitalization, after a normal swallowing study, he started oral feeding.

The tracheostomy decannulation was performed because he showed a good respiratory and neurological course.

The patient was discharged after 47 days of hospitalization, with follow-up and rehabilitation.

Twenty months after *commotio cordis*, the

Table 1. Cardiac enzyme values

Hospitalization day	Troponin (ng/L)	CK (IU/L)	CK-MB (UI/L)
1	11 653	615	213
2	70 155	2360	291
3	32 154	994	122
4	10 623	528	85
6	2239	171	48
8	82	97	-
10	22	74	-

CK: creatine kinase. Values expressed in IU/L. Normal value: 26–140 IU/L.

CK-MB: isoenzyme of creatine kinase.

Troponin: values expressed in ng/L. Normal value: < 30 ng/L.

patient does not have motor deficit, neurological deficit, or cognitive and learning disorders; he continues receiving the corresponding therapies. All cardiac controls after discharge were normal.

DISCUSSION

Commotio cordis occurs as ventricular fibrillation caused by a blunt trauma to the left side of the chest, not attributable to structural damage of the heart and its surrounding structures.[1,2] It is one of the leading causes of sudden death in adolescents and young adults. It is the second most common cause of cardiac death in young athletes, predominantly in males, following hypertrophic cardiomyopathy.

The number of reported cases is less than 30 annually. The United States National *Commotio Cordis* Registry (Minneapolis) has been established to facilitate the aggregation of information.[3,4]

In a study published in *JAMA* in 2002 that confirmed 128 cases of CC, 95% of the patients had a mean age of 14 years. Of these cases, 62% occurred during sporting events. The fatal event was dependent on the range of impact velocity and the time until assisting the victim.[5]

The reported survival rate among African Americans is 4% versus 33% among Caucasians. This may be due to a higher rate of late resuscitation (44% versus 22%) and a lower AED use (4% versus 8%).[6]

CC is caused by an impact to the precordial region with a hard ball, at a vulnerable moment of cardiac repolarization, which triggers a cardiorespiratory arrest due to ventricular fibrillation. It is a direct, non-penetrating, involuntary trauma to the chest, without injury to the ribs or sternum and in the absence of an underlying cardiovascular disease.

The absence of a structural cardiac injury distinguishes CC from cardiac contusion (*contusio cordis*), where high-impact blows cause a traumatic injury to the myocardial tissue and the chest.

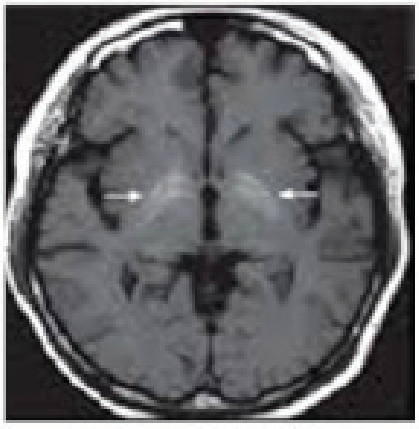
CC events are observed during sports activities involving children, adolescents, and young adults. The injury occurs when the mechanical energy caused by a blow, limited to the precordial area, alters the electrical stability of the myocardium and causes ventricular fibrillation. The energy of the impact must be sufficient to cause ventricular depolarization, estimated at about 50 joules, e.g., a thrown baseball.[7] Smaller balls may have a higher risk of CC because the impact is concentrated on a smaller surface area.[8]

CC is usually fatal, with a very low survival rate, although it has increased thanks to awareness of CC, the rapid establishment of basic life support, and access to AED in the shortest possible time.

For this to occur, several factors must be combined, such as the object characteristics, velocity, and direction; the individual's physique and susceptibility; the location of the blow; and the fact that, at the moment of impact, the myocardium is electrically vulnerable (elevation of T wave) and the ion channels are activated so that ventricular fibrillation occurs.

The importance for diagnosis is to exclude

Figure 1. Brain magnetic resonance imaging with increased bilateral and symmetrical signal intensity of both globi pallidi in T2 and FLAIR sequences, with restriction images in diffusion sequences compatible with hypoxic-ischemic lesion



major chest trauma inducing structural cardiac injury. Children with long QT intervals are susceptible to CC, making electrical conversion the best immediate treatment.[9]

The American Heart Association (AHA) and the American College of Cardiology (ACC) provide a strong recommendation, based on moderate-quality evidence, that after resuscitation patients with CC should undergo "a comprehensive evaluation for underlying cardiac pathology and susceptibility to arrhythmias."[10]

An ECG may reveal evidence of myocardial injury, being it difficult to establish whether it occurred primary or secondary to cardiac arrest.

Troponin levels and an echocardiogram are useful to determine the presence of myocardial contusion. An echocardiogram identifies any underlying structural abnormalities present. A stress test or cardiac catheterization may be considered to assess for coronary artery disease, while pharmacological tests may be done to look for Brugada and long-QT syndromes.

The survival rate has improved thanks to the accessibility to immediate defibrillation and rapid bystander response. Every minute lost before defibrillation reduces the survival rate.

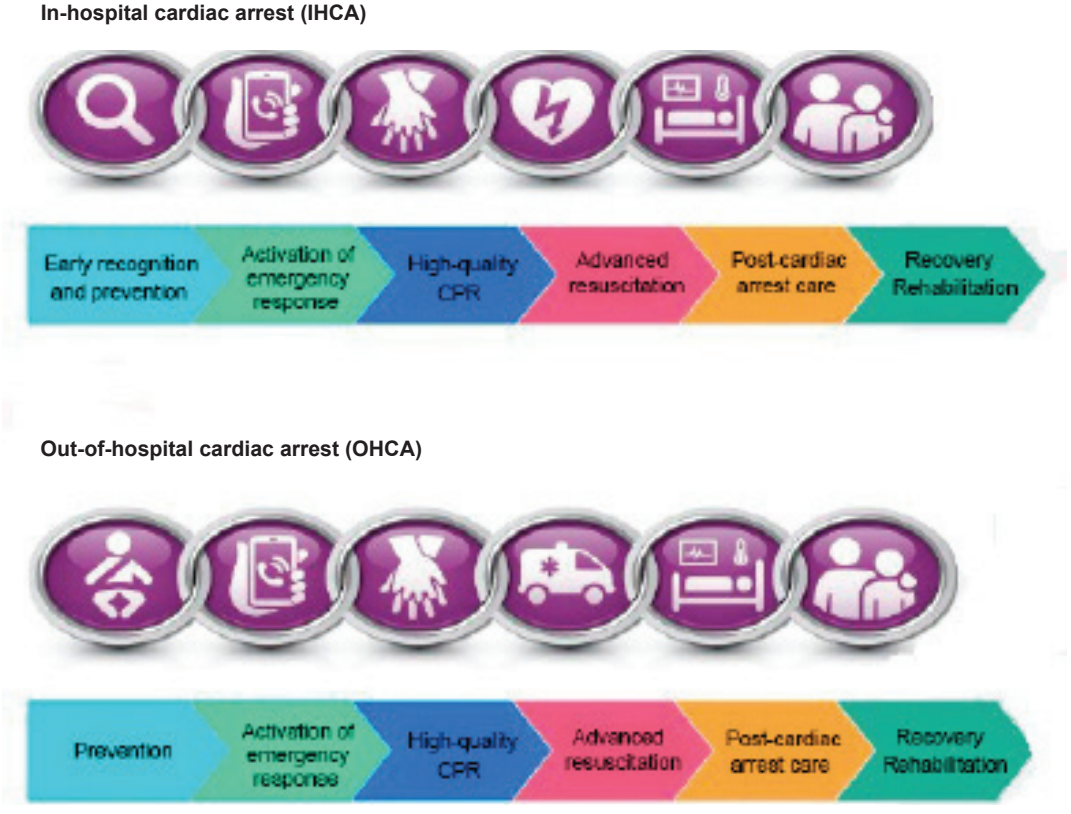
Prevention remains a major factor, with primary and secondary strategies.

Primary strategies include raising awareness to avoid precordial blows, considering ball material, wearing chest vests (although 37% of reported cases occurred with chest protectors in place).[6–11]

Secondary strategies include training the population in CPR techniques and AED availability. The AHA[10–12] provides the following recommendations:

1. "Measures should be taken to ensure

Figure 2. Pediatric chain of survival proposed by the American Heart Association for in-hospital cardiac arrest (IHCA) and out-of-hospital cardiac arrest (OHCA)



Source: Pediatric Basic and Advanced Life Support. In Highlights of the 2020 American Heart Association guidelines for CPR and ECC. Dallas: American Heart Association; 2020;14-22.
CRP: cardiopulmonary resuscitation.

successful resuscitation of *commotio cordis* victims, including training of coaches, staff, and others to ensure prompt recognition, notification of emergency medical services, and institution of cardiopulmonary resuscitation and defibrillation." (Strong recommendation, based on moderate-quality evidence).

2. "Use age appropriate safety baseballs to reduce the risk of injury and *commotio cordis*." (Moderate recommendation, based on moderate-quality evidence).
3. "Rules governing athletics to reduce chest blows can be useful to decrease the probability of *commotio cordis*." (Moderate recommendation, based on limited evidence).

Survival has improved with increased awareness and access to medical care and public defibrillators.[13–15]

The key reasons for the mortality are the failure of timely resuscitation and the presence of congenital heart disease.

CONCLUSION

Commotio cordis is a condition that can be fatal if no immediate action is taken; the prognosis is better if the chain of survival is activated in a timely manner and basic CPR with the use of AED is performed. It is important to provide training to teachers and to have AEDs available at all sports competitions.

The standardization of resuscitation out of the hospital has led to a steady increase in survivors from all types of cardiac arrest.[13–15]

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