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From Innocuous Contact to Sudden Death in Sports: An Overview of Commotio Cordis

Mohammed Shaban[*1], Franklin Sosa[1], Miguel Rodriguez-Guerra[2], Muhammed Saad[3], Timothy J Vittorio[3]

[1]*Department of Medicine, BronxCare Hospital Center, Icahn School of Medicine at Mt. Sinai, Bronx, NY, USA.*

[2]*Department of Medicine, Albert Einstein College of Medicine, Montefiore Medical Center, Bronx, NY, USA.*

[3]*Division of Cardiology, BronxCare Hospital Center, Icahn School of Medicine at Mt. Sinai, Bronx, NY, USA.*

[*]Corresponding Author

Mohammed Shaban, Department of Medicine, BronxCare Hospital Center, Icahn School of Medicine at Mt. Sinai, Bronx, NY, USA.

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Abstract

Commotio Cordis (CC) is a rare but potentially fatal condition that occurs when a blunt impact to the chest causes ventricular fibrillation (VF) and sudden cardiac death. CC is most common in teenage males who participate in sports, but it can occur in people of all ages. The mechanical distortion of the myocardium caused by trauma can lead to inappropriate depolarization and myocardial firing, which can cause arrhythmias. Immediate identification and defibrillation are crucial to reducing mortality rates.

Currently available screening tools for CC, such as the electrocardiogram (EKG), stress EKG, or echocardiogram, lack sensitivity for primary prevention. The pathogenesis of CC is influenced by a variety of factors, including the location, velocity, shape, and hardness of the impact object, as well as biological characteristics like gender, chest wall flexibility, and genetic susceptibility. The ion channels involved in arrhythmia generation in CC are thought to be stretch-activated channels (SACs).

Diagnosis is based on a history of chest wall blunt trauma, and treatment includes immediate supportive care and defibrillation according to BLS and ACLS protocols. The currently available chest wall protectors have not been shown to prevent CC, and improving sports equipment may help reduce the incidence of CC.

A famous American football player incident in which he collapsed after making a tackle during the first quarter of the Bills' game against the Cincinnati Bengals on January 2, 2023, is a notable case of commotio cordis (CC) in a football player. This incident is unusual because helmets are not typically associated with CC, which is usually caused by external blunt force trauma to the chest with small balls, hockey pucks, or even a blow. The player, who fortunately survived the event, was relatively older than the recorded mean age for CC and has a more rigid chest wall. It is unclear whether this case is an exception or if helmets can pose a risk for CC in certain circumstances.

The case highlights the need for continued research and awareness of CC to improve prevention and management strategies for sudden cardiac arrest in sports. Despite being an ancient condition, CC remains a tragic event, and further research is needed to improve preventive measures and reduce mortality rates.

Keywords: Commotio Cordis, Ventricular fibrillation, Sudden cardiac death, Blunt trauma, Stretch-activated channels, Sports equipment, Football player, Sport trauma

Introduction

Trauma remains a leading cause of morbidity and mortality, particularly among young people. In the US, approximately 30,000 blunt cardiac trauma patients are reported annually [1]. Commotio Cordis (CC) is a word in Latin that means "agitation of the heart or disturbance of the heart." It is commonly used for ventricular fibrillation (VF) precipitated by blunt trauma to the heart [2]. The non-penetrating trauma sparks mechanical distortion of the myocardium, creating inappropriate depolarization, which in turn initiates myocardial firing [3].

The sudden cardiac death of a young athlete is the most tragic event in sports and the local community. Unfortunately, cardiac arrest is the first cardiac sign in up to 80% of asymptomatic young athletes because of the limited screening modalities and the lack of guidance in this fatal condition [4]. The rapid identification of the condition in the field, with early defibrillation, has been the only way to cut down on the high mortality and improve the outcome [5]. This review will discuss the available historical records, epidemiology, pathophysiology, acute management, and current preventive measures to improve outcomes in CC.

Historical Records

Despite the term Commotio Cordis being first used in the 19th century, cases likely attributable to CC have been described for centuries. An ancient Chinese martial art technique, Dim Mak, was reported to cause death by a carefully directed blow, christened "the touch of death" [3].

The earliest documentation so far was by Giovanni Lancisi, the papal physician, in De Subitaneis Mortibus's book when a man died after receiving a fist under the xiphoid cartilage [6]. In sports, the earliest case in the newspaper was a teenage cricket ball player in 1898 after a moderate velocity impact of a cricket ball to his chest [7].

Few similar unexplained death scenarios were found in archival baseball records of the early 1900s [8]; however, the exact mechanism of CC was unclear. The high-velocity (projectile) objects, such as baseball and hockey purks, were thought to be the initiative etiology [9]. To uncover the CC pathology, George Ralph Mines was one of the first cardiac electrophysiologists who conducted many animal experiments to induce cardiac fibrillation. Ultimately, he passed away at the age of 28 years during a self-experiment after he decided it was time to begin work on human subjects [10]. Another famous George victim of CC was Captain George Boiadri, who lost his life during a baseball match in 2004 [11]. More recently, a Helmet caused the same pathology in a famous American football player, who luckily was able to recover due to early intervention by CPR & Automated External Defibrillation (AED) [12].

Epidemiology

Since the United States National Commotio Cordis Registry was started in 1995, approximately 10 to 20 cases have been added to the registry yearly. The mean age in the registry is 15 years, with less than 10% older than 25 years. The male gender counts for 95% of cases, with 75% of athletic body build [13].

Cooper et al. [14] conducted a retrospective analysis of a database of six thousand cases of sudden cardiac death in the United Kingdom's popular Sports such as football, cricket, and rugby. They identified 17 cases of CC. Sixteen were male, and 11 were 18 years old or younger. Eleven occurred while playing sport (mainly by a ball in the chest), while six involved physical interaction, including assault. Although the true incidence of CC was not precisely calculated, they estimated that UK's CC cases follow the same circumstantial and age profile as in the US. [14]. More recently, in 2022, FIFA Sudden Death Registry (FIFA-SDR) tracked the sudden death in worldwide football (soccer) from 2014 to 2018. The registry collected data on 617 sudden deaths of soccer players from 67 countries. The mean age of the victims was 34, with a wide spectrum of standard deviation up to 16 years. Similar to prior registries, the vast majority were male gender (96%). Of those 617 players, only 142 (23%) have survived, and 211 cases had diagnostic autopsies. In players younger than 35 years, CC occurred in 5% [15].

According to most registries, CC occurs mainly in teenage males, who represent the highest participation rates in sports. It could also be explained by the incomplete chest wall development, allowing external forces transmission to the heart [16]. Fortunately, cardiac resuscitation and automated external defibrillator (AED) improved the survival rate to 85% (compared to 35% without), reflecting the urged need for immediate access to resuscitation capabilities, especially AED devices at competition sites [15].

Pathophysiology

The action potential (AP) is voltage changes across the cell membrane caused by ions movement intra and extracellular through ion channels. According to the presence of various ion channels, AP varies in different tissues, such as contractile myocytes, pacemaker cells, and conductive pathways [17]. AP starts with depolarization (phase 0) mainly due to the opening of sodium (Na) channels that allow Na+ to flow into the cell. The AP repolarization (Phase 1, 2, and 3) depends on potassium (K) channels opening, allowing K to leave the cell, and calcium (Ca) channels, increasing the intracellular Ca [18] (Figure 1, Table 1)

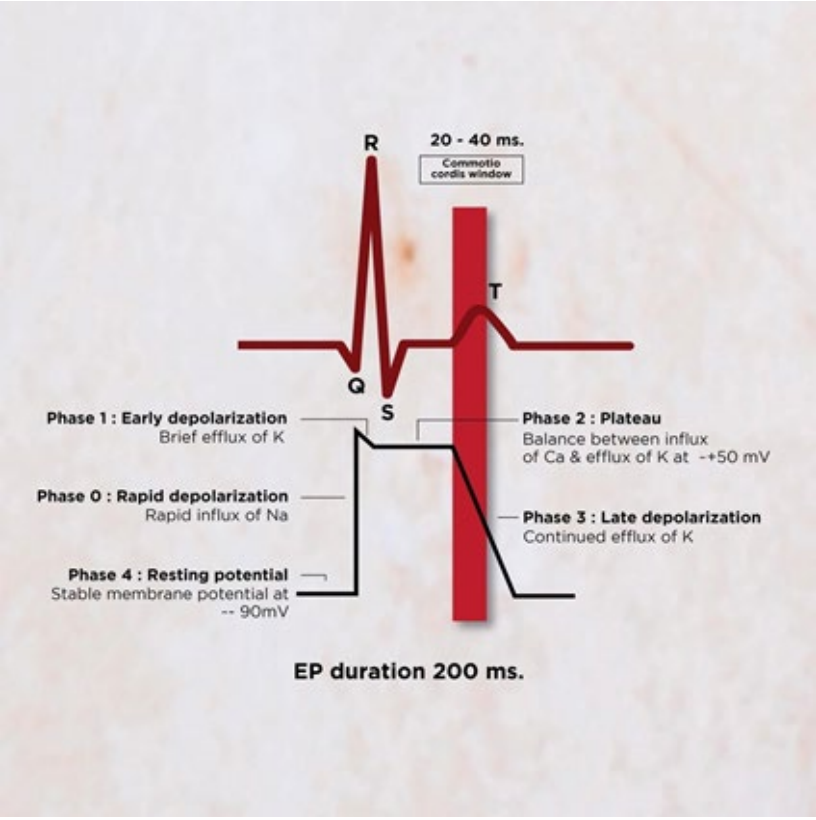

Figure 1: Cardiac action potential physiology with corresponding QRS and T waves showing commotio cordis window.

Cardiac arrhythmias are variable syndromes caused by functional and structural defects in ion channels or electrical pathways. Some ion channel defects interfere with the AP stability and duration, causing excitability abnormalities. Abnormal excitations in the early part of phase 3 repolarization may lead to inactivated Na/Ca channel reopening. The Na/Ca channel reopening provides extra current for depolarization that may trigger polymorphic ventricular tachycardia, torsades de pointes, and even VF [19]. In CC, the left ventricular pressure rise after an impact would likely activate ion channels via mechano-electric coupling. The generation of inward current via mechano-sensitive ion channels likely results in a non-uniform myocardial activation, causing premature ventricular depolarization and VF [20].

Mechanical Component

Mechanical stimulation initiates the electrical cardiac activation in CC. The absence of structural cardiac injury distinguishes CC from cardiac contusion [21]. Myocardial mechanosensitivity is involved in arrhythmogenesis in various ways; It can start a life-threatening VF, as in CC, or contrarily terminate VF, as in precordial thump [22]. Precordial thump had been thought to be a life-saving procedure in VF when a defibrillator was not immediately available [23]; however, it is only equivalent to approximately 5-10 joules of mechanical energy to the heart, i.e., shallow compared to the needed dose to break most of VFs. Nowadays, there is a growing concept that precordial thump is rarely of value [24].

The pathogenesis of CC is also affected by the impact object's location, velocity, shape, and hardness. In addition, biological characteristics such as gender, the flexibility of the chest wall, and genetic susceptibility are important determinants (Figure 2) [25]. Link et al. [26] used 30-mph baseball impacts on pigs to study CC. They could generate VF (with T-wave strikes) and ST-segment elevation (with QRS strikes) [26]. Dickey, Bian, Khan, et al. [27] performed 128 chest impacts to evaluate the resultant rib deformation with LV pressure. They used 16 impact locations with variable baseball stiffness levels. They found the highest LV strain with 17.9 m/s velocity and directly over the LV. Interestingly, the rib deformations, not the impact force metric, correlated to LV strain and pressure [27].

Timing is very crucial in the pathogenesis of CC. The animal models revealed that only mechanical impacts during a narrow window of repolarization would cause VF [25]. The impact must be before the peak of the T wave, called a "supernormal or vulnerable window," causing membrane stretch and ion channel activation [16] (Figure 1).

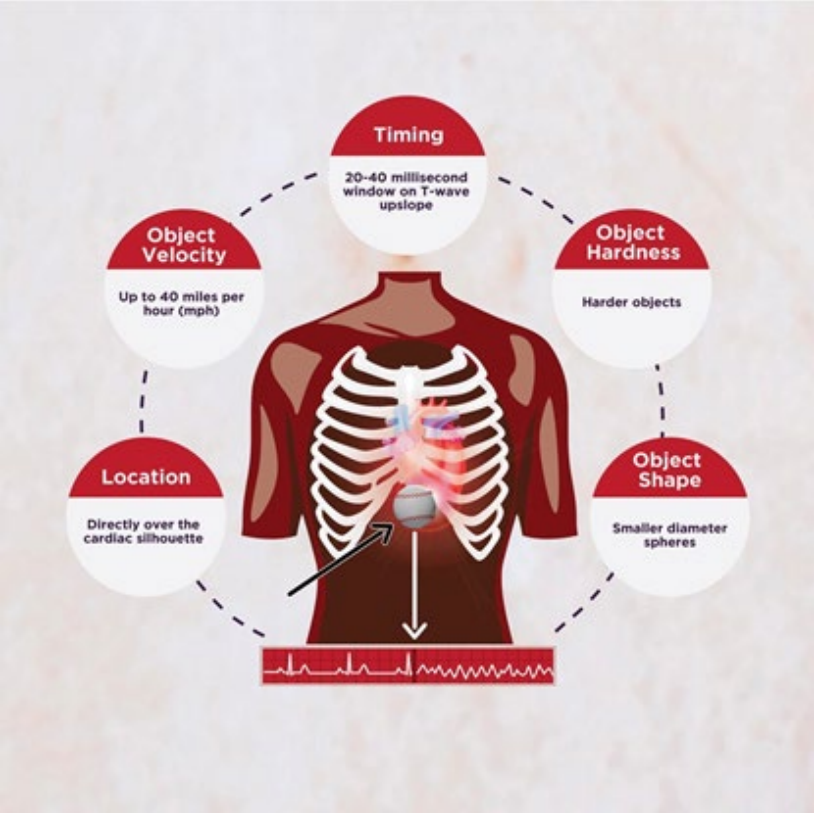

Figure 2: Mechanism of CC.

Electric Component

Cardiac K channels are membrane proteins that allow the passive movement of K+ ions across the cell membrane along its electrochemical gradient. They regulate the resting membrane potential, the frequency of pacemaker cells, and the shape and duration of the cardiac action potential [28] K channels are the main channels in the cardiac cell action potential repolarization, particularly phase 3, when a "supernormal or vulnerable window" occurs [28,29]. Link et al. [26] hypothesized that K channels are responsible for ventricular arrhythmias in the CC. They experimented on pigs after being given 0.5 mg/kg IV glibenclamide, a selective inhibitor of the K channel. They found that K channel blockade reduced the incidence of VF to 4% compared to 33% in the control group [26]. A similar experiment with different outcomes was repeated on an isolated rabbit heart by Quinn et al. [30]. They performed a pretest pharmacological block of ATP-inactivated K channels or stretch-activated channels (SAC) followed by mechanical stimulation to generate premature ventricular complex or VF. They found that the K channel blocker did not affect the frequency of premature ventricular complex; however, it delayed the narrow "vulnerability window" by delaying T wave repolarization and prolonging refractoriness [30].

The stretch-activated channels (SACs) are the ion channel thought to play the main role in arrhythmia generation in CC. The SACs activation alters AP repolarization, triggering ventricular depolarization and a spiral wave that quickly breaks down into VF [25]. The scroll wave re-entry is thought to underlie the VF in CC [31]. Trayanova, Constantino, and Gurev [32] initially proved the mechano-electric coupling through SACs' activation in 2010 using a computerized rabbit model. They also hypothesize that the exact mechanism contributes to arrhythmias termination with a precordial thump or during acute regional ischemia [32]. Colli Franzone, Pavarino, and Scacchi [33] used another computerized 3D human model in 2017 to study the reentrant wave dynamics and mechano-electric coupling. They have proven that VF can be triggered by the low reversal potential of non-selective SACs [33]. More recently, in 2022, Lee, Cansız, and Kaliske [34] used another human ventricular model to evaluate three different models for SACs during Mechano-Electric Feedback (MEF). They found that each model of SACs has regionally variable effects on the pathology of CC, precordial thump, or, even more interesting, blood pressure fluctuation associated with ventricular arrhythmias [34].

Till now, there is not much data suggesting genetic susceptibility to CC. However, Creta, Hanington, and Lambiase [35] reported a CC case with a heterozygous mutation of the CACNA1C gene [35]. CACNA1C gene encodes for the L-Type cardiac calcium channel; its variants follow the autosomal dominant inheritance pattern and cause gain-of-function mutation, manifested with long QT syndromes [36]; however, Creta, Hanington, and Lambiase's case had normal QT duration [35].

Clinical Scenarios

The severity of cardiac trauma varies widely in their condition on presentation. Although some may present mild sternal bruising, others may present with acute valvular rupture or malignant arrhythmia [37].

CC is the VF caused by a blunt, non-penetrating, and often innocent-appearing unintentional blow to the chest without damage to the ribs, sternum, or heart [3]. VF is the most dangerous arrhythmia that commonly results in a sudden loss of consciousness, absence of pulse, and gasping for breathing. Respiratory arrest and sudden cardiac death will occur if the VF is not reverted [2]. CC is diagnosed when there are no structural abnormalities in the heart, ribs, or sternum [7].

The earliest sports documentation of CC in the newspaper was in 1898 [7]. Despite CC cases in archival baseball records of the early 1900s, limited medical literature records about CC were published before the 1990s, probably as cases were not noticed or explained. This lack of records raises another theory to suggest that these events represent a novel evolving cardiac pathology in the younger population [8].

Differential Diagnosis

The analysis of 617 mortalities in the FIFA-SDR registry has revealed that traumatic deaths such as traumatic intracranial hemorrhage, carotid artery rupture, abdominal bleeding, and neck fracture were responsible for deaths in 8% of total cases, and 11% of people younger than 35 years old. Other mortalities were caused by non-traumatic causes, valvular heart disease, congenital heart disease, unspecified cardiovascular diseases, non-traumatic intracranial bleeding, and arrhythmogenic cardiac syndromes. In South America, cardiomyopathy syndromes such as hypertrophic cardiomyopathy, ischemic cardiomyopathy, or arrhythmogenic right ventricular cardiomyopathy counted for 42% of the total cardiac mortalities in players older than 35 years old. Worldwide, coronary artery disease is the leading cause of sudden death in players older than 35 (76%). In players younger than 35 years, whereas CC occurred only in 5%, cardiomyopathy was prevalent in 18%, coronary artery disease in 9%, coronary artery anomalies in 9%, and myocarditis in 5% [15].

Conduction system disorders have been reported in relation to blunt chest trauma [38]. Atrial fibrillation (AF) is frequently associated with atrial dilatation caused by pressure or volume overload. SACs in the atrial myocardial cells are thought to mediate the AF in dilated atria [39]. There are limited reports of AF precipitation after blunt chest trauma. Ota and Bratincsak [40] reported blunt chest trauma during a football game, causing a new-onset AF in a healthy teenager. The mechanically induced AF was self-terminated in 3 days. No structural or electrical cardiac abnormality or arrhythmia history was identified. It is hypothesized that SACs mediated AF generation similarly to VF in CC after blunt chest trauma [40].

Blunt chest trauma was also reported to induce transient right bundle branch block or even complete heart block (CHB). The CHB can, in turn, precipitate VF. 38 Ali et al. [41] analyzed the clinical data of 50 patients who have been reported with CHB after blunt chest trauma. They reported death in 20% of cases mainly caused by malignant arrhythmia in the initial 72 hours, with no AV system structural damage in 50% of their autopsies. In the surviving CHB cases, half of the patients returned to normal AV conduction within 7-10 days. However, a permanent pacemaker was needed for the other half [41].

Management Strategies

Early Post event management

Pre-participation screening tools such as 12 lead electrocardiogram (EKG), stress EKG, or echocardiogram can catch high-risk personnel such as those with structural heart diseases, arrhythmogenic right ventricular dysplasia, cardiomyopathies, and coronary artery anomalies. The EKG and stress EKG can also uncover arrhythmogenic syndromes such as prolonged QT and Brugada syndrome; however, all screening tools failed to show any degree of sensitivity to predict CC cases. In other words, we lack the tool for the primary prevention of CC [4].

The history of chest wall blunt trauma is the cornerstone of diagnosing CC as the cause of sudden cardiac arrest [16]. Immediate supportive care, external defibrillation, using automated external defibrillators according to basic life support (BLS) and advanced cardiac life support (ACLS) protocols. These protocols may be followed by therapeutic hypothermia as clinically indicated [16]. In the last decades, extra efforts have been exerted to implement programs of early external defibrillation of unpredictable arrhythmic cardiac arrest in sports fields [4].

Late post-event outcome

CC survivors should undergo a thorough workup to rule out any other cause of sudden cardiac death. The workup should include EKG, echocardiography, stress testing, and ambulatory EKG monitoring. Suspicion of some clinical syndrome, such as arrhythmogenic right ventricular dysplasia or hypertrophic or restrictive cardiomyopathies, may require cardiac magnetic resonance imaging (CMR). All structural or arrhythmogenic heart disease has to be excluded before returning to practice competitive or recreational sports. Once ruled out, there are no restrictions for returning to athletic activities [16].

Few blunt chest trauma victims were reported to have sustained or recurrent ventricular tachycardia (VT) of monomorphic morphology in the chronic stage. In most cases, arrhythmogenic tissue related to preexisting heart disease was thought to provide the substrate for VT reentrant circuit. Fortunately, in the absence of overt preexisting heart disease, VTs are focal and easily ablated [42]. Also, some CC victims showed echocardiographic evidence of regional wall motion abnormalities. The mechanism is not fully uncovered but may be associated with inflammation and complement activation [1].

Mechanical Prophylaxis

CC mainly occurs in the younger population. With age progress,

the chest wall is expected to be more mature and, therefore, rigid. Therefore, theoretically, rigid chest protectors will provide primary prevention against CC. However, current, commercially available chest wall protectors have not been shown to protect against CC either in animal models or in humans on the sports fields [16].

The softer baseballs were shown to have a lower incidence of CC [20]. The smaller diameter objects, like baseballs, cause a higher sharper increase in LV pressure and subsequently more risk of VF. On the contrary, Spreading the impact force over a larger area is associated with a lower risk of CC [43]. Our recent football player case 12 could be an exception, as a helmet was the object that precipitated CC. However, it is unclear whether his relative older age and more mature and rigid chest wall were related to being "out of" the common rule.

In 2022, Dickey, Bian, Liu, et al. [44] performed a study to identify the most dangerous impact locations to help provide better designs of effective chest design protectors in a 10-year-old children model using the CHARM-10 chest model. They found the highest LV strain with direct impacts over the LV and slightly lateral and superior to the cardiac silhouette. The most elevated LV pressure was also found with direct impacts over the LV. They encouraged the protective material manufacturers to focus on these points to minimize the incidence of CC in young athletes [44].

Community Awareness

Although all the efforts against CC occurrence in competitive sports, educational awareness among individuals, closer team surveillance, adequate rescue devices, and medical assistance are the cornerstone of preventing CC's poor outcomes [9]. The current recommendations set a 3 minutes time frame to deliver the first shock in sports fields. All the athletic trainers and participants have to be aware of the initial symptoms as exertional syncope or presyncope, agonal gasping, or chest pain. Seizure-like activity or myoclonic jerking from CC can be mistaken for a seizure. The National Athletic Trainers' Association also suggests also to release a family history of sudden cardiac arrest or a family history of sudden death and exercise intolerance to the athletic trainers [45]. Similar to BLS and ACLS certificates for healthcare workers, CPR training is becoming mandatory for professionals in workplaces where they may be the first witness to emergencies as coaches, personal trainers, teachers, childcare providers, and safety officers in various workplaces [46].

Trials for Pharmacological Prophylaxis

1- Gadolinium is a widely used contrast agent in MRI studies for its ability to differentiate between healthy and unhealthy tissues. Gadolinium is a calcium channel blocker that reduces membrane activation and synaptic neurotransmitter release [47]. The gadolinium is also a SACs blocker in the myocardial cell. It showed a dose-dependent ability to decrease the incidence of stretch-induced AF vulnerability in rabbit hearts. It was hypothesized to be a prophylactic agent against atrial fibrillation evolvement in those with atrial pressure or volume overload [39]. Zhang, Walcott, and Rogers 22 performed a study using six pig hearts and mechanical stimulations to investigate the concentration-dependent properties of gadolinium to suppress electrical activation from mechanical stimuli. They ensured that the gadolinium perfusate was not recirculating back to prevent gadolinium precipitation with waste products. Unfortunately, their experiment failed to show any potential role of gadolinium in inhibiting mechanical stimulus-induced ventricular arrhythmias [22]. In addition, the cumulative gadolinium effects prohibited its role in arrhythmia prophylaxis. The concentration-dependent deposition of gadolinium in the brain, particularly in the globus pallidus and dentate nucleus, raises concerns about its long-term safety [48].

2- Class III antiarrhythmic medications are K channel blockers that delay potential action repolarization (phase 3), which in turn causes increased duration of action potential and the effective refractory period (ERP). Class III antiarrhythmics are widely used for treating and preventing both atrial and ventricular arrhythmias (Table 1) [49]. On the opposite side, Class III antiarrhythmics have a pro-arrhythmic effect through the QT prolongation and subsequent increased incidence of Torsade de Pointes or VF [50]. The transmural dispersion of repolarization (TDR) is considered essential during sustained scroll wave re-entry responsible for VF in CC [31]. Using computer simulations, Haraguchi et al. (2011) studied the TDR as a VF inducer with various pharmacological blockers, such as Class III antiarrhythmics. They used the scroll wave-organizing center, known as a filament, to quantify the activity during scroll wave re-entry. K channel blockers, such as dofetilide, quinidine, vesnarinone, and nifekalant, did not change the relationship between the TDR and the average filament total length. Still, they did have different effects on TDR duration. In other words, the studied K channel blockers did not show any potential benefit in preventing VF in the context of CC but only delayed the "supernormal or vulnerable window" when mechanical triggers induce CC [31].

Ion Channel	The phase of Action Potential	Medications
Na+	Rapid depolarization (Phase 0)	Lidocaine (Class 1B), Procainamide (Class 1A), Flecainide (Class 1C), Propafenone (Class 1C), Mexiletine (Class 1B)
K+	Early repolarization (Phase 1)	Amiodarone (Class 3), Dofetilide (Class 3), Sotalol (Class 3), Ibutilide (Class 3)
Ca2+	Plateau (Phase 2)	Verapamil (Class 4), Diltiazem (Class 4)

SAC (Stretch-activated)		Gadolinium
Funny (If)	Sinoatrial node spontaneous depolarization	Ivabradine

Table 1: Cardiac Ion Channels, Associated Medications, and Phases of Action Potentials.

Conclusion

Commotio cordis is a fatal condition that appears in asymptomatic healthy young individuals. Usually, it is caused by a mechanical impact resulting in arrhythmia such as VF, which could lead to sudden cardiac death. This event mainly happens in competitive and recreational sports. Improving sports safety equipment is expected to decrease the incidence of CC. For example, possible magnetic equipment that would direct balls away from the vulnerable points could be an option. However, non-all the possibilities could be covered; in the recent American football player incident, it was the helmet causing the event. Possibly, a higher impact momentum would be needed for a more mature chest wall to cause a sudden increase in LV strain and, consequently, CC. The cornerstone in CC management is the availability of automated defibrillators, increased awareness about the disease, and the importance of immediate application of BLS protocol.

Ethics Approval and Consent to Participate

Not applicable (N/A) as this is a review article and does not involve participants or require ethics approval.

Consent for Publication

Not applicable (N/A) as this is a review article and does not involve participants or require individual consent for publication.

Availability of Data and Materials

The data and materials used in this review article are obtained from publicly available sources, which are appropriately cited in the reference section. No specific datasets were generated for this study.

Competing Interests

The authors declare that they have no competing interests.

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Authors' Contributions

All Authors have contributed equally to the manuscript by gathering information, rigorously reviewing available literature, drafting the manuscript, and providing critical reading and feedback to one another.

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