Emergency Bulletin
Hong Kong College of Emergency Medicine

An innocent blow to the heart

Dr. Law Ping Keung, Joe
FHKCEM, FHKAM (Emergency Medicine)

A healthy 30-year-old man collapsed after being hit by a football on chest wall in a soccer game. He was unresponsive with no pulse and breathing. Bystander cardiopulmonary resuscitation (CPR) was started immediately by his teammates. Emergency medical system was activated and an on-site automated external defibrillator (AED) arrived after 2 minutes.

The AED detected a shockable rhythm and recommended a shock, which was then administered. The paramedics arrived 8 minutes afterwards and detected a palpable pulse. The timeline of the events was shown (Figure 1).

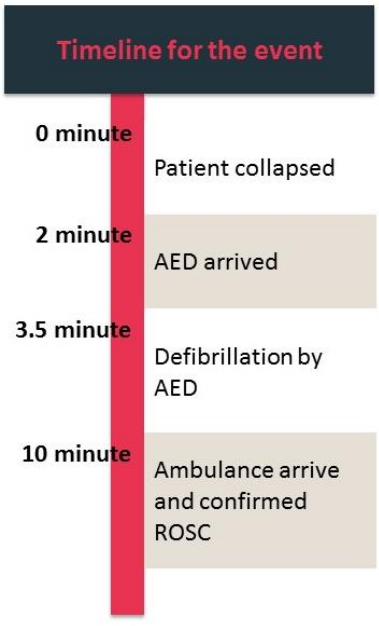

Figure 1: Timeline of the events

He was brought in to the emergency department. On arrival, his vitals were

- GCS E1V1M1
- Blood pressure: 114/54 mmHg, pulse 101 beats per minute
- SpO2: 98%, bagging with 100% oxygen
- Small bruise on left forehead
- No external wound on chest wall.

ECG reviewed sinus rhythm with QTc of 429 ms (Figure 2) and chest x-ray was unremarkable.

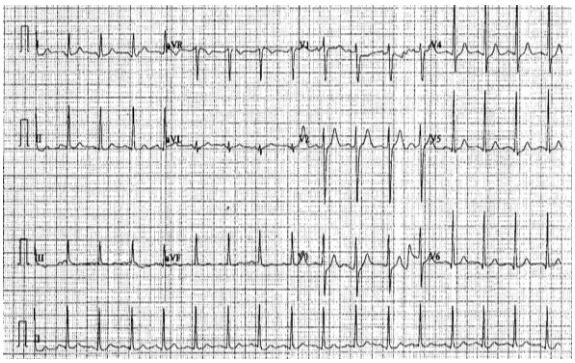

Figure 2: ECG of the patient

What is the role of basic life support?

Basic life support is the foundation for saving lives. It includes immediate recognition of sudden cardiac arrest and activation of the emergency response system, early CPR and rapid defibrillation with an AED.[1]

High quality CPR improves survival from cardiac arrest and consists of[1]

- Chest compression of adequate rate
- Chest compression of adequate depth
- Allow full chest recoil between compressions
- Minimize interruptions in chest compressions
- Avoidance of excessive ventilation

Progress of patient

The record of the AED was retrieved (Figure 3). Rhythm analysis identified a shockable rhythm which is ventricular fibrillation. Shock advice was recommended and subsequently delivered (Biphasic 168 joules).

Following the defibrillation, CPR was continued. There was a brief period of asystole for three minutes before restoration of spontaneous circulation. The first organized rhythm was ventricular escape rhythm with wide QRS complex.

Why early defibrillation is important for our patient?

Our patient is in cardiac arrest with a shockable rhythm. Early defibrillation is crucial. Time to defibrillation is probably the single most important determinant of survival for our patient.[2] The likelihood of successful defibrillation decreases rapidly over time as ventricular fibrillation generally evolves to asystole within a few minutes.[3]

Survival rates after VF cardiac arrest decrease approximately 7% to 10% with every minute of delay in defibrillation. The approximate survival rate with time from defibrillation was showed in table 1.[2]

Time from defibrillation	Survival
Within first minute	As high as 90%
5minutes	50%
7minutes	30%
9 to 11 minutes	10%
Beyond 12 minutes	2% to 5%

Table 1: Survival rate with time from defibrillation

Progress of patient

Rapid sequence intubation was performed and the patient was transferred to the intensive care unit for further management.

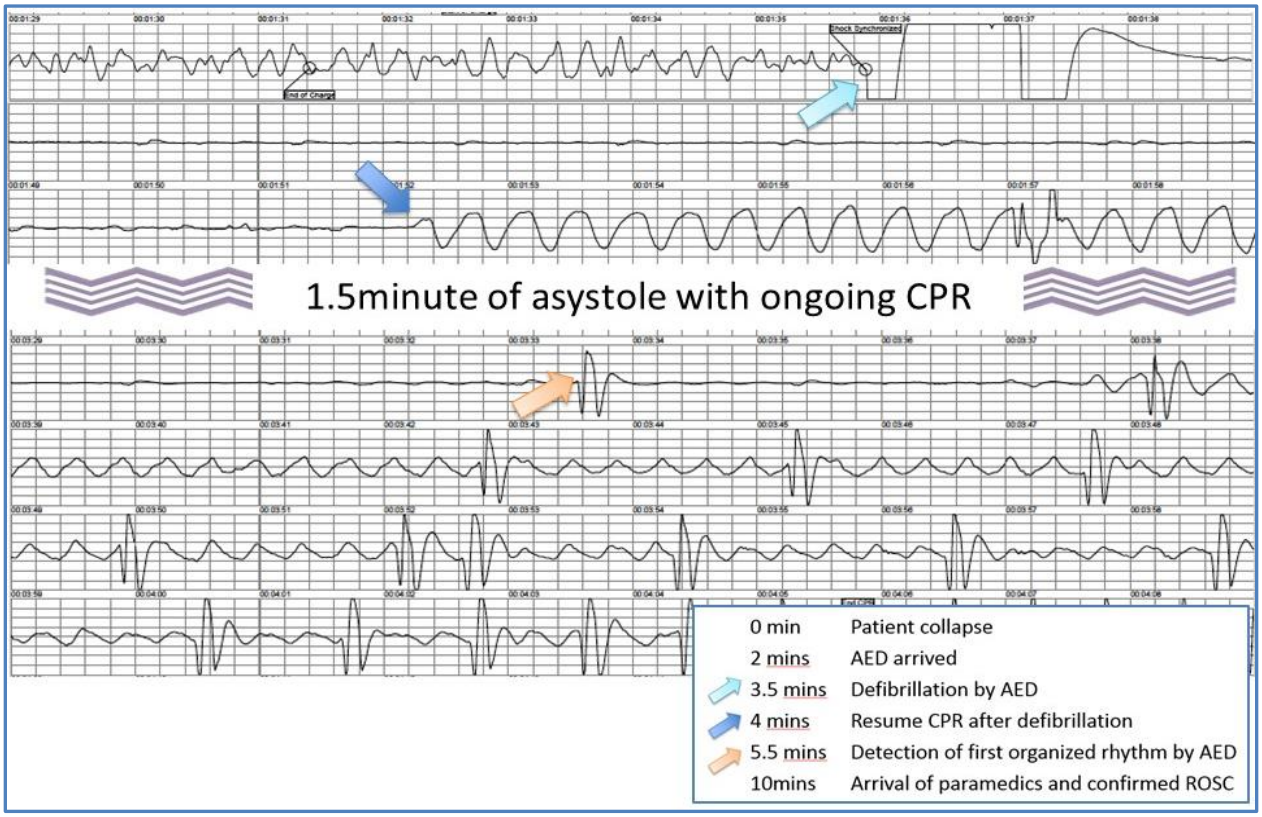

Figure 3: Record of the automated defibrillator

What are the common etiologies of sudden cardiac arrest in athletes?

Sudden cardiac arrest associated with athletic activity is uncommon. The majority of events are due to malignant arrhythmias. Many of these deaths occur in the presence of previously undiagnosed cardiovascular disease.[5]

The etiology can be broadly classified into structural heart disease, primary electrical disease and others: (Table 2)

Structural heart disease
- Hypertrophic cardiomyopathy (HCM)
- Anomalous origin of coronary artery
- Arrhythmogenic right/left ventricular cardiomyopathy (ARVC)
- Myocarditis
- Coronary atherosclerosis
Primary electrical disease
- Long QT syndrome
- Brugada syndrome
- Catecholaminergic polymorphic ventricular tachycardia
- Short QT syndrome
- Early repolarization syndrome
Others
- Commotio cordis
- Medication toxicity including doping
- Electrolyte abnormalities
- Idiopathic

Table 2: Etiology of sudden cardiac arrest in athletes

Progress of patient

The patient underwent targeted temperature management in the intensive care unit. Blood test showed elevated creatine kinase which peaked at 5867 U/L (48 hours after attendance), and elevated high sensitive troponin I which peaked at 139 ng/ml (24 hours after attendance). Serum electrolytes, thyroid function and lipid profile were normal. Echocardiography and private MR coronary angiogram and MRI brain were all unremarkable.

What is the most likely diagnosis?

Based on the history and extensive investigation results, the most likely diagnosis is commotio cordis.

Commotio cordis, derived from Latin, means "agitation of the heart".[6] It is defined as sudden cardiac arrest secondary to relatively low-impact blunt trauma to the anterior chest wall.[7]

It most often occurs during participation in certain competitive sports (50%) e.g. baseball, soft ball, ice hockey, football or lacrosse, or recreational sports (25%), with rare occurrences during normal, routine daily activities e.g. being kicked in the chest or struck by a playground swing as it rebounds.[8]

What is the difference between commotio cordis and contrusio cordis (cardiac contusion)?

Although preceded by a traumatic event, commotio cordis is a primary electrical event with ventricular fibrillation (VF) occurring immediately upon chest wall impact that is usually not of sufficient force to causes any significant structural injury to the heart.[6,9]

This is different from contrusio cordis, in which high impact blows result in direct myocardial tissue damage e.g. contusion of myocardial muscle, rupture of a cardiac chamber or disruption of a heart valve, and often injury to the overlying structures of the chest and thorax.[8]

What is the pathophysiology of commotio cordis?

The mechanism of impact-induced VF is currently under investigation. The dual-abnormality hypothesis suggested that the initiation of ventricular fibrillation in commotio cordis requires a trigger and a susceptible myocardium.[7,8,10,11]

A trigger

- Chest wall impact activates mechano-sensitive K+ ATP channel which creates focal ventricular depolarization and produce a premature ventricular complex (PVC).

A susceptible myocardium

- Chest wall impact results in rapid increase in LV intra-cavity pressure, which causes cell membranes to stretch, activate ion channels and increase trans-membrane current flow. This increases the heterogeneity of repolarization (dispersion of repolarization) and creates an electrically vulnerable substrate that is susceptible to VF.

If a trigger (PVC) falls on the vulnerable portion of the T wave (upstroke of T wave), induction of VF may occur.

Based on animal models using rabbits, cats, dogs and juvenile swine mimicking the clinical profile of commotio cordis, several critical variables are necessary for the development of it.[6,12,13,14]

1. Timing of impact relative to the cardiac cycle[14]

   The most important variable for the development of commotio cordis is the timing of impact relative to the cardiac cycle.

   Only impacts occurring 40 ms before the peak of the T-wave to the T-wave peak (early repolarization) will cause ventricular fibrillation. The likelihood markedly increases if the impacts occur between 10 to 30 ms before the peak of the T wave. (Figure 4)

   Chest impacts occurring in other period of the cardiac cycle produce various other effect e.g. ST–segment elevation, PVC, transient heart block and left bundle branch block, but never resulted in VF.

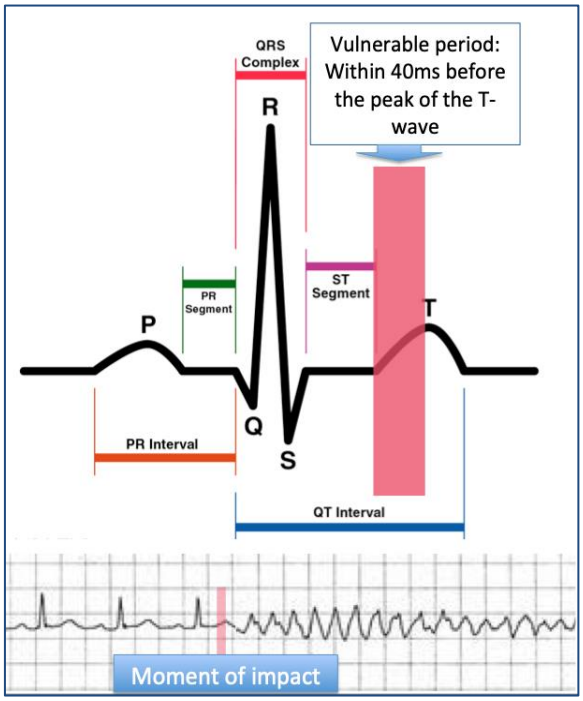

Figure 4: Vulnerable period of the cardiac cycle

2. Impact location[15]

   Impacts must occur directly over the cardiac silhouette for the development of VF. VF occurs most commonly with impacts over the center of the cardiac silhouette (30% of the impacts) versus those over the left ventricular base (12% of impacts) or apex (5% of impacts). Strikes not over the cardiac silhouette never result in ventricular fibrillation.[16]

   This finding is consistent with clinical observations that precordial bruises of a blow are frequently evident in victims.[17]

3. Velocity of impact[15]

   The threshold velocity to cause VF was 40-48 km/h (25 to 30 mph). VF never occurs at velocities below 20 mph. As impact velocity increased, the incidence of VF rose to a peak of nearly 70% of impacts at 64 km/h (40 mph).

At velocities >80 km/h (50 mph), the likelihood of VF decreased while the incidence of cardiac rupture and major trauma increase.

4. Hardness of the object[13,18]

   Softer, more pliable objects are less likely to cause ventricular fibrillation because of their propensity to collapse on contact and to absorb some of the energy of the impact.

   This is compatible with the observation that air-filled balls used in soccer, tennis and basketball rarely implicated in commotio cordis (only account for 4% of commotio cordis)

5. Shape of object[13,21]

   Smaller spheres were more likely to induce ventricular fibrillation, whereas ventricular fibrillation never occurred with flat objects.

Which population is more susceptible to commotio cordis?

Commotio cordis shows a predilection for children and adolescents (mean age 15+/-9 years). 26% of victims were younger than 10 years of age and only 9% were 25 years of age or older. Most victims are males (95%).[8,19,20]

The predisposition to commotio cordis in young people may be related to physical characteristics of the thorax in the young and also the reduced participation of adult in ball-related sports.

The relatively thin, underdeveloped compliant chest cage and immature intercostal musculature is less capable of blunting the arrhythmogenic consequences of precordial blow. This is supported by animal study that ventricular fibrillation is more easily induced in a smaller swine than in a larger animal. There is a significant drop-off in ventricular fibrillation inducibility with animal weights >40 kg.[15]

In addition, since children probably incur chest blows more frequently than adults in a variety of circumstances, they may generally be at greater risk for commotio cordis.

What is the prognosis of commotion cordis?

Most of the data related to the commotio cordis are derived from the US commotio cordis registry.

Survival during the initial years of the registry (1970-1993) was only 10%. There has been a progressive improvement in survival in recent years with survival up to 58% in 2006-2012.

This can be attributed to increased public awareness, earlier recognition of the event and commencement of CPR and increased availability of public-access AED for defibrillation.[8,22]

The treatment of commotio cordis is not different from any other cardiopulmonary emergency associated with a non-perfusing cardiac rhythm. Early CPR and rapid defibrillation can significantly increase the chances of survival.

However, CPR is often delayed because observers tend to underestimate the severity of the trauma. Survival is most likely to occur if institution of CPR and defibrillation are available within 3 minutes of the incident.[23]

The survival rate is only 5% or less in cases in which resuscitative efforts were delayed longer than 3 minutes.[23] Although numerous individuals have been resuscitated with the restoration of a perfusing heart rhythm, many of these individuals have experienced irreversible ischaemic encephalopathy and ultimately died as a result of the injury.

How to prevent commotio cordis?

Deaths of young athletes from commotio cordis are tragic. Several approaches for prevention have been proposed.

For primary prevention, education on commotio cordis and the importance of avoiding precordial blows should be stressed.[24] Even an unintentional modest-seeming blow to the chest can trigger life-threatening ventricular tachyarrhythmias.[19]

In organized sports, better coaching techniques such as teaching inexperienced young player to turn away from the ball or avoid using their chest to block the ball may help to prevent commotio cordis. [24,25]

Improved design of sports equipment may also help to prevent commotio cordis. In animal study, safety baseball consisting entirely of rubber without the dense, hard core significantly reduced the risk of lethal arrhythmias. However, their effectiveness in reducing the risk of commotio cordis has not been proved in the field and safety baseballs have been responsible for several fatal events. [18]

Chest wall protectors have also been proposed. However, none of the currently available chest protectors was shown to significantly decrease the incidence of VF when compared to controls.[6,19,20,26]

Indeed, chest protectors may create a false sense of security, given that almost 20% of victims of commotio cordis were wearing chest protectors. In several fatal cases, the ball struck the chest protector directly indicating that the material of the chest protectors was inadequate.[20] In other cases, the chest protector migrate upwards while a player's arms are fully raised, exposing the precordium to the direct blow. [27]

An ideal chest protector should be made from hard, rigid and resistant material to prevent penetration of projectiles, and from foams capable of absorbing and dispersing great amount of energy. Besides, such protector should be adaptable for use by players playing any position in any sport. Further research on the development of an ideal chest protector is needed.

For secondary prevention, AED plays an important role. It has substantial life-saving capability. A public program to make AED widely available in sporting events and recreational settings is likely to improve the survival of people in the event of CC.[28,29]

Progress of the patient

The patient was transferred back to general ward after completion of therapeutic hypothermia. He fully regained his consciousness but developed memory loss and retrieval deficit in verbal memory. His mini-mental state exam (MMSE) was 26/30 and Montreal Cognitive Assessment Hong Kong version score was 23/30. He was discharged home on Day 39 after a course of rehabilitation.

Should our patient avoid competitive sport in future?

There is no increased risk for subsequent arrhythmic events nor is there evidence that athletes who have had a commotio cordis events should be disqualified from competition solely for that reason.[8]

Similarly, prophylactic implantable defibrillators are not indicated for persons who have survival commotio cordis in the absence of cardiac disease.[8]

Reference

1. Berg RA, Hemphill R, Abella BS, Aufderheide TP, Cave DM, Hazinski MF, Lerner EB, Rea TD, Sayre MR, Swor RA. Part 5: Adult basic life support: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. *Circulation*. 2010;122(suppl 3):S685–S705.
2. "Part 4: The Automated External Defibrillator: Key Link in the Chain of Survival." Resuscitation, vol. 46, no. 1–3, Aug. 2000, pp. 73–91
3. Martin DR, Gavin T, Bianco J, Brown CG, Stueven H, Pepe PE, et al. Initial countershock in the treatment of asystole. Resuscitation 1993;26:63-8.
4. Weaver WD, Copass MK, Bufi D, et al. Improved neurologic recovery and survival after early defibrillation. Circulation. 1984;69:943–948.
5. Tseng, Zian H., et al. "Prospective Countywide Surveillance and Autopsy Characterization of Sudden Cardiac Death: POST SCD Study." Circulation, vol. 137, no. 25, June 2018, pp. 2689–700.
6. Maron BJ, Poliac L, Kaplan JA, Mueller FO. Blunt impact to the chest leading to sudden death from cardiac arrest during sports activities. N ENGl J Med 1995;333:337-42
7. Link MS, Wang PJ, Pandian NG, et al. AN experimental model of sudden death due to low-energy chest-wall impact (commotio cordis). N Engl J Med 1998;338:1805-11.
8. Maron BJ, 3rd Estes NA. Commotio cordis. N Engl J Med. 2010;362(10):917-27.
9. Nesbitt AD, Cooper PJ, Kohl P. Rediscovering commotio cordis. Lancet. 2001
10. Bode F, Franz MR, Wilke I, Bonnemeier H, Schunkert H, Wiegand UK. Ventricular fibrillation induced by stretch pulse: implications for sudden death due to commotion cordis. J Cardiovasc Electrophysiol 2006;17:1011-7.
11. Viano DC, Andrzejak DV, Polley TZ, King AI. Mechanism of fatal chest injury by baseball impact: development of an experimental model. Clin J Sport Med 1992;2:166-71.
12. Link MS, Commotio cordis ventricular fibrillation triggered by chest impact-induced abnormalities in repolarization. Circulation: arrhythmia Electrophysiol. 2012;5(2):425-32
13. Link MS, Wang PJ, Pandian NG, Bharati S, Udelson JE, Lee MY, et al. An experimental model of sudden death due to low-energy chest wall impact (commotio cordis). N Eng J Med. 1998;338(25):1805-11.
14. Madias C, Maron B, Weinstock J, Estes NA, 3rd, Link M. Commotio cordis – sudden cardiac death with chest wall impact. J Cardiovasc Electrophysiol. 2007;18:115-22.
15. Link MS, Maron BJ, Wang PJ, Vander Brink BA, Zhu W, Estes NA. Upper and lower limits of vulnerability to sudden arrhythmic death with chest-wall impact (commotio cordis). J Am Coll Cardiol. 2003;41(1):99-104.
16. Link MS, Maron BJ, Vanderbrink BA, et al. Impact directly over the cardiac silhouette is necessary to produce ventricular fibrillation in an experimental model of commotion cordis. J Am Coll Cardiol 2001;37649-654.
17. Sheppard, Mary N. "Aetiology of Sudden Cardiac Death in Sport: A Histopathologist's Perspective." British Journal of Sports Medicine, vol. 46, no. Suppl 1, Nov. 2012, pp. i15–21.
18. Link MS, Maron BJ, Wang PJ, Pandian NG, VanderBrink BA, Estes NA. Reduced risk of sudden death from chest wall blows (commotion cordis) with safety baseballs. Pediatrics. 2002;109:873-877.
19. Maron BJ, Gohman TE, Kyle SB, Estes NAM III, Link MS. Clinical profile and spectrum of commotion cordis, JAMA 2002;287:1142-6
20. Maron BJ, Doerer JJ, Haas TS, Estes NAM III, Hodges JS, Link MS. Commotio cordis and the epidemiology of sudden death in competitive lacrosse. Pediatrics 2009;124:966-71.
21. Kalin J, Madias C, Alsheikh-Ali AA, Link MS. Reduced diameter spheres increases the risk of chest blow-induced ventricular fibrillation (commotio cordis). Heart rhythm 2011;8(10):1578-81.
22. Maron BJ, Haas TS, Ahluwalia A, Garberich RF, Estes NA 3rd, Link MS. Increasing survival rate from commotion cordis. Heart Rhythm. 2013;10(2):219. Epub 2012 Oct 26.
23. Link MS, Maron BJ, Stickney RE, et al. Automated external defibrillator arrhythmia detection in a model of cardiac arrest due to commotion cordis. J Cariovasc ELectrophysiol 2003;14:83-87.
24. Link MS, Bir C, Dau N, Madias C, Estes NAM III, Maron BJ. Protecting our children from the consequences of chest blows on the playing field: a time for science over marketing. Pediatrics 2008;122:437-9
25. Strasburger JF, Maron BJ. Commotio cordis N Engl J Med 2002;347:1248.
26. Doerer JJ, Haas TS, Estes NAM III, Link MS, Maron BJ. Evaluation of chest barriers for protection against sudden death due to commotio cordis. AM J Cardiol 2007;99:857-9.
27. Kaplan JA, Karofsky PS, Volturo GA. Commotio cordis in two amateur ice hockey players despite the use of commercial chest protectors: case reports. J Trauma. 1993;34:151-3
28. Myerburg RJ, Estes NAM III, Fontaine JM, Link MS. Zipes DP. Task force 10: automated external defibrillators. J Am Coll Cardiol 2005;45:1369-71.
29. Salib EA, Cyran SE, Cilley RE, Maron BJ, Thomas NJ. Efficiacy of bystander cardiopulmonary resuscitation and out-of-hospital automated external defibrillation as life-saving therapy in commotion cordis. J Pediatr 2005;147:863-6.