

# Emergency Medicine and Trauma Care Journal

## Case Report

Taylor L, et al. Emerg Med Truama. EMTCTJ-100023

## Death Punch: A Case of Commotio Cordis Resulting from Sibling Rivalry

Taylor L<sup>1</sup>, Surrey A<sup>1\*</sup> and Humble J<sup>1</sup>

<sup>1</sup>Department of Emergency Medicine, Virginia Commonwealth University Health System, Richmond, Virginia, USA

\*Corresponding author: Aaron Surrey, Department of Emergency Medicine, Virginia Commonwealth University Health System, 1200 E Marshall St, Richmond, Virginia, USA.

**Citation:** Taylor L, Surrey A, Humble J (2020) Death Punch: A Case of Commotio Cordis Resulting from Sibling Rivalry. Emerg Med Truama. EMTCTJ-100023

**Received date:** 11 January, 2020; **Accepted date:** 20 January, 2020; **Published date:** 27 January, 2020

### Abstract

Commotio cordis is a rare cause of sudden cardiac death resulting from blunt chest trauma. Of the 10-20 cases reported per year, the majority are in male children in the setting of a blow from a projectile during a sporting event. To the best of our knowledge, this is the first reported case in the medical literature of commotio cordis caused by a blow from a human fist. The objective of this clinical case report is to highlight this unusual and rare injury to increase awareness, particularly in the setting of blunt trauma in a child.

**Keywords:** Blunt Cardiac Injury; Commotio Cordis; Cardiac death

### Introduction

Commotio cordis (Latin, agitation of the heart) is sudden cardiac death resulting from blunt chest trauma. Victims of commotio cordis are frequently young and most often found in ventricular fibrillation without significant cardiac or thoracic damage [1] Commotio cordis has been described in contemporary medical literature since the late 1970's however, it has been documented in textbooks as early as 1857 [1,2] Some experts believe the condition may have been described in early 17th century China in the form of Dim mak (death touch). Dim mak is a series of martial arts combat techniques concentrated on impact to acupuncture points. One of these is described as a blow to the left sternum that could cause sudden death in opponents [3,4]. In this case report, we present a young female with sudden cardiac arrest as a result of being punched in the chest by her brother.

### Case Report

A 17-year-old female with no significant medical, surgical or family history presented to the emergency department following an episode of sudden cardiac death. Earlier in the evening she and her brother were engaged in an argument when they began to scuffle. He struck her in the chest during their altercation.

According to the patient's mother, the patient stiffened immediately upon being struck, grabbed her brother's shirt and fell to the floor. Her brother tried to awaken her, then immediately ran to his mother who called 911 and began cardiopulmonary resuscitation (CPR). Fortunately, emergency medical services (EMS) was just 2 blocks away and arrived within 3-5 minutes.

The initial rhythm detected on the EMS monitor was ventricular fibrillation and they provided one defibrillation of 200J with immediate resolution of the ventricular fibrillation and a change of rhythm to sinus tachycardia. The patient was transported to a local community hospital where she was subsequently intubated. Arrangements were then made to transfer her to VCU for a complete trauma evaluation. The patient had transient hypotension during transport, however, it resolved after reduction of her sedation medications of propofol and fentanyl.

On arrival, the patient's initial vital signs were as follows: blood pressure 112/72, pulse rate of 136 beats/min, respiratory rate 23 breaths/min, temperature of 37.6°C (99.7°F), and oxygen saturation of 97% on minimal ventilator settings. The only other significant injuries noted on secondary survey were contusions over the patient's chest wall.

The patient's initial laboratory testing was remarkable for a troponin of 0.14 ng/mL (<0.03ng/mL-0.19ng/mL), hemoglobin 9.2g/dL, WBC count 21.7 10<sup>9</sup>/L, platelet level 144 10<sup>9</sup>/L, PT 21.7 sec, INR 1.8. Initial EKG showed sinus tachycardia with a rate of

102 beats/minute, normal intervals, no signs of ischemia and no waveforms that would suggest an underlying dysrhythmia. Chest imaging revealed significant bilateral posterior consolidations consistent with possible hemorrhage or contusions, and the remainder of her imaging including: bedside echocardiogram, chest radiograph, CT: Head, and CT: Cervical spine, were unremarkable.

The patient was admitted to the Surgical-Trauma ICU where she was monitored, weaned from sedation and ultimately extubated within 48 hours. The patient was then transferred to the general pediatrics floor. The patient's troponin peaked at 0.58ng/mL, and presumed to be related to the chest compressions. A pediatric transthoracic echocardiogram did not demonstrate any structural or functional abnormalities. A follow-up cardiac MRI was unremarkable except for noting mildly reduced right and left ventricular function. The patient continued to improve, and she was discharged 10 days after arrival at her neurologic baseline.

## Discussion

### Epidemiology

The National Commotio Cordis Registry was established in the mid-1990's to collect data on commotio cordis.<sup>1,3</sup> Over 200 cases have been reported, of which at least 60 cases have been reported from outside the U.S.<sup>1,4</sup> While the exact incidence is unknown given the lack of mandatory reporting, approximately 10-20 cases are added to the registry every year [5]. These reported cases make it the second most common cause of sudden death in young athletes after hypertrophic cardiomyopathy [1].

According to the registry, commotio cordis more commonly afflicts the young with a mean age of 15 years reported [5] This may be due to the fact that 75% of the cases occur during athletic activities, which children are more likely to participate in. The fact that children have thinner and more compliant chest walls relative to those of adults may also play a role. In addition, 95% of patients are male. The presented case is particularly interesting in that the patient is female and it did not involve athletic activity.

While survivability was reported to be as low as 10% from 1970-1993, reported rates have increased to as high as 33-58% in recent years [5] Delays in resuscitation longer than 3 minutes cause an 8-fold drop in survival rate from 40% to 5%. In addition, significantly lower rates of survival were found in African Americans (4%) than in Caucasians (33%) ( $p=0.004$ ), a concerning finding likely due to reduced access to care and facilities with AEDs.<sup>5</sup>

### Pathophysiology

There are several factors involved in the actions that put a patient at risk of commotio cordis. Susceptible events include impact directly over the cardiac silhouette from more spherically shaped objects with smaller diameters such as a baseball [6-8]. Harder objects are more likely to cause commotio cordis than softer ones.<sup>8</sup> There is an increasing incidence of commotio cordis as projectile speed increases up to 40 mph; however, impacts greater than 40 mph are more likely to cause structural damage and other blunt cardiac injury [9].

The most important variable in developing ventricular

fibrillation in commotio cordis is the timing of the chest wall impact. Impacts during a 20-40 ms window of early ventricular repolarization, or the upslope of the T-wave on the cardiac cycle, are susceptible to commotio cordis [8]. It is thought that ATP-sensitive  $K^+$  channels are activated by the stretch of the chest blow, which results in increased inhomogeneity of ventricular repolarization and subsequently ventricular fibrillation [1,3,10].

## Treatment and Management

Management of patients who present in ventricular fibrillation due to commotio cordis does not differ from other causes of ventricular fibrillation [4] Initial managements should focus on early resuscitation including closed chest compressions, early defibrillation, rescue ventilation, and appropriate medications as described by ACLS [11] It is prudent to exclude other forms of traumatic arrest early such as tension pneumothorax, cardiac tamponade, and hemorrhagic shock if clinically indicated.

The American Heart Association and American College of Cardiology recommend that, after return of spontaneous circulation, patients should undergo a comprehensive evaluation for underlying cardiac pathology and susceptibility to dysrhythmias.<sup>12</sup> An initial electrocardiogram (ECG) and troponin may reveal evidence of cardiac dysfunction, though it may be difficult to distinguish if any abnormalities observed were the cause of or were caused by the arrest.<sup>4,12</sup> ECG may also identify electrical abnormalities such as Brugada syndrome, long-QT syndrome, and Wolf-Parkinson-White syndrome. Recall that intervals and wave morphology differ in the young, and thus age-based ECG criteria should be implemented. Other diagnostics that should be considered include echocardiogram and magnetic resonance imaging to rule out underlying structural abnormalities.<sup>12</sup> Ambulatory ECG monitoring and stress testing should be considered. No recommendations exist to perform cardiac catheterization in all patients, though may be considered if appropriate.

## Prevention

In the absence of underlying cardiac disease, it is unlikely for a second randomly occurring event to occur. However, some individual susceptibility has been reported in animal models for unclear reasons [12] Therefore, while individuals may safely resume training and competition after a commotio cordis event if workup is otherwise benign, avoidance of sports at risk for chest wall impact may be considered [12,13] Techniques to reduce chest blows should be taught to athletes. While not absolutely protective, safety balls should be used when applicable [8,14] Commercial chest protectors have not been shown to consistently prevent commotio cordis events, as chest protectors were worn in 37% of commotio cordis events occurring in competitive sports [15] Prompt recognition and defibrillation increase survivability; as such, automatic external defibrillators (AEDs) should be available anywhere athletic activities occur [5].

## Conclusion

Commotio cordis is a rare event but a common cause of sudden death in young athletes. Specific timing of impact during the cardiac cycle as well as other risk factors cause susceptibility to ventricular fibrillation. While more commonly occurring in males and in the course of athletic events, care should be taken to

consider commotio cordis in all causes of arrhythmogenic cardiac arrest in young patients. Early recognition, readily available AEDs and prompt resuscitation including chest compressions and defibrillation have increased the survivability of commotio cordis.

## References

1. Maron BJ and Estes NA (2010) Commotio Cordis. *N Eng J Med* 362: 917-27.
2. Casper JL (1857) *Pactisches Handbuch der gerichtlichen Medicin*, Hirschwald, Berling, Verlag Von A.
3. Kelly M (2001) *Death touch: the science behind the legend of Dim-Mak*. Boulder, CO: Paladin Press. Pp: 176.
4. Tainter CR, Hughes PG. *Commotio Cordis* (2019) Prime PubMed. Treasure Island, Fl: StatPearls Publishing.
5. Maron BJ, Haas TS, Ahluwalia A, Barberich RF, Estes NA, et al. (2013) Increasing survival rate from commotio cordis. *Heart Rhythm* 10: 219-23.
6. Link MS, Maron BJ, VanderBrink BA, Takeuchi M, Pandian NG, et al. (2001) Impact directly over the cardiac silhouette is necessary to produce ventricular fibrillation in an experimental model of commotio cordis. *J. Am. Coll. Cardiol* 37: 649-654.
7. Kalin J, Madias C, Alsheikh-Ali AA, Link MS (2011) Reduced diameter spheres increases the risk of chest blow-induced ventricular fibrillation (commotio cordis). *Heart Rhythm* 8: 1578-1581.
8. Link MS, Wang PJ, Pandian NG, Bharati S, Udelson JE, et al. (1998) An experimental model of sudden death due to low-energy chest-wall impact (commotio cordis). *N. Engl. J. Med* 338: 1805-1811.
9. Link MS, Maron BJ, Wang PJ, VanderBrink BA, Zhu W, Estes N, et al. (2003) Upper and lower limits of vulnerability to sudden arrhythmic death with chest-wall impact (commotio cordis). *J. Am. Coll. Cardiol* 41: 99-104.
10. Bode F, Franz MR, Wilke I, Bonnemeier H, Schunkert H, et al. (2006) Ventricular fibrillation induced by stretch pulse: implications for sudden death due to commotio cordis. *J Cardiovasc Electrophysiol* 17: 1011-1017.
11. Link MS, Berkow LC, Kudenchuk PJ, Halperin HR, Hess EP et al. (2015) Part 7: Adult Advanced Cardiovascular Life Support: 2015 American Heart Association Guidelines Update for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. *Circulation* 132: S444-S464.
12. Braverman AC, Harris KM, Kovacs RJ, Maron BJ (2015) Eligibility and Disqualification Recommendations for Competitive Athletes with Cardiovascular Abnormalities: Task Force 13: Commotio Cordis: A Scientific Statement from the American Heart Association and American College of Cardiology. *Circulation* 132: e303-e309.
13. Alsheikh-Ali AA, Madias C, Supran S, Link MS (2010) Marked variability in susceptibility to ventricular fibrillation in an experimental commotio cordis model. *Circulation* 122: 2499-2504.
14. Link MS, Maron BJ, Wang PJ, Pandian NG, VanderBrink BA, et al. (2002) Reduced risk of sudden death from chest wall blows (commotio cordis) with safety baseballs. *Pediatrics* 109: 873-877.
15. Doerer JJ, Haas TS, Estes NA, Link MS, Maron BJ (2007) Evaluation of chest barriers for protection against sudden death due to commotio cordis. *Am J Cardiol* 99: 857-859.