Commotio cordis

Introduction

Commotio cordis (cardiac concussion) was first described in the 19th century by Schlomka and Schmitz for cases of instantaneous cardiac arrest following chest wall trauma (Kohl et al, 2001). In terms of location, chest blows striking the left chest, most directly over the cardiac silhouette, are frequently associated with commotio cordis. The condition affects young males particularly as a result of their more pliable chest wall frame (Link, 2003). In terms of demographics, the commotio cordis registry currently records a mean age of 14 years with 80% 18 years or younger (Maron et al, 2002).

Sports such as baseball, softball, ice hockey, football and lacrosse are notorious for such a phenomenon. This article highlights a case and discusses the pathogenesis, clinical presentation and prevention.

Physiological mechanism

Studies by Georg Schlomka uncovered the underlying pathophysiological mechanisms responsible. He successfully identified three main factors strongly associated with cardiac concussion: type of impact, location and force (McCrory, 2002). Force of chest blows are as such difficult to quantify. Analysis of the velocity of the impact object has shown it to usually correspond to what is considered normal for that particular sport. For example, in youth baseball a pitched baseball can travel at 50 miles/hour, which is sufficient to induce the condition (Seefeldt et al, 1993).

Researchers noted that when the precordial impact was delivered between 30 and 15 ms before the peak of the T wave, a ventricular fibrillation was induced often resulting in subsequent death (McCrory, 2002). Such an arrhythmia was not produced by impacts at any other time during the cycle and with impacts delivered just outside the 15 ms period, a polymorphic ventricular tachycardia was noted (Link et al, 1998).

The initiation of ventricular fibrillation is multifactorial requiring a premature ventricular depolarization as the trigger factor in addition to an altered myocardial substrate, both being produced by the chest wall blow itself (Link, 2003). The activation of specific ion channels in the myocardial substrate allows for initiation of such an arrhythmia, the most likely one being the K+ ATP channel. This was proven through experimental use of glibenclamide, a K+ ATP channel blocker, which was shown to reduce the incidence of ventricular fibrillation following chest wall blows (Link, 2003). Other channels may also be activated including the stretch-activated channel which may induce ventricular fibrillation following chest wall injury. However, further study is needed to confirm the importance of such a channel in the underlying mechanism. Finally, left ventricular pressure rises following chest trauma have been documented to mediate a ventricular fibrillation. The peak probability of such an arrhythmia being seen is with left ventricular pressures ranging between 250 and 450 mmHg (Link, 2003).

Alternative explanations of cardiac concussion exist and include trauma-induced vasospasm leading to ventricular fibrillation, cardiac contusion, electromechanical dissociation, asystole, hypervagatonia and the long QT syndrome (Estes, 1995).

Cardiac concussion or contusion?

Cardiac concussion and contusion are both rare events with significant differences (*Table 1*). The differences associated with the two conditions were defined in the early 1970s.

The use of isoenzyme studies may have some bearing with regards to differentiation. For example, creatine kinase isoenzyme (CK MB) screening in addition to two-dimensional echocardiography has been used to differentiate between cardiac concussion and contusion. In the former, two-dimensional echocardiography is usually within normal limits but is abnormal in cases of contusion (Frazee et al, 1986). However, this assessment is not concrete in terms of distinguishing between the two types of cardiac injury: autopsy studies have demonstrated a normal CK MB level with significant blunt cardiac injury (Tellez et al, 1987).

Clinical presentation

Clinically most patients with commotio cordis lose consciousness and collapse immediately after such an injury. Some reports have described a lucid phase whereby the individual falls to the ground after being struck and stands up briefly only to collapse again (Valani et al, 2004).

Case Report

A 22-year-old man, Mr X, and his friend, were playing a game of softball. His friend (as batsman) accidentally struck the ball into Mr X's chest. Mr X subsequently collapsed and lost consciousness. Cardiopulmonary resuscitation began following the arrival of paramedics. The patient was found in ventricular fibrillation and defibrillated to sinus rhythm. Upon arrival in the emergency department, the patient remained unresponsive but haemodynamically stable. An echocardiogram was performed which showed no structural abnormalities, pericardial effusions or valvular damage. The patient soon reverted back to ventricular fibrillation and despite the best efforts of the medical team could not be revived.

An autopsy was warranted which indicated no evidence of injury or natural disease to account for his death. A section from the autopsy report is as follows: 'The heart weighs 316 g. The pericardial sac is free of significant fluid or adhesions. The coronary arteries arise normally and follow the distribution of a right dominant pattern with no significant atherosclerosis. The chambers and valves are proportionate. The valves are normally formed, thin and pliable and free of vegetations and degenerative changes. The myocardium is dark red brown, firm and free of focal or regional fibrosis, erythema, pallor or softening. The atrial and ventricular septa are intact and the septum and free walls are free of muscular bulges.'

Microscopic assessment of the heart as well as toxicological analysis of the blood proved unremarkable. The medical examiner assigned commotio cordis as the immediate cause of death.

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Diagnostic methods

Death from cardiac concussion is unlikely to yield any significant pathological findings. There is often no gross or microscopic damage to the heart and no cellular damage. Chemical investigation is also unremarkable with no release of creatine phosphokinase from the myocardium (Koehler et al, 2004). Some have suggested a role for conductive system assessment in such cases, but normal gross and histological examination have been deemed sufficient in terms of diagnosis, time and expense (Thiene et al, 1983).

Survival rates

Studies have documented the difficulty in restoring normal cardiac rhythm in such individuals and hence potential survival. One study assessed survival rates of 25 victims who had collapsed with cardiac arrest immediately after an unexpected blow to the chest with a sporting projectile (such as a baseball or hockey puck). Of the 25, 19 underwent cardiopulmonary resuscitation (CPR) within about 3 minutes with normal cardiac rhythm being restored in only two of those individuals. What proved even more remarkable was the fact that 25% of these victims were wearing some form of protective chest padding (Maron et al, 1995). Studies have also shown the importance of timing with regard to resuscitation delivery. A 25% survival rate was observed if CPR was performed within 3 minutes compared to only 3% if delayed by more than 3 minutes (Link, 2003). A major determinant of survival is therefore early defibrillation.

Prevention

Prevention of commotio cordis can be sought through various means. Using animal models, Link et al (1998) found that the use of safer equipment such as softer baseballs could decrease the risk of arrhythmias. They observed a directly proportional relationship between risk and ball hardness. Janda et al (1998) found that in 78% of cases, safety test balls significantly lowered the impact force compared to the standard baseball.

In addition, focus on more advanced chest wall protection which specifically covers the left chest wall and precordium may help to reduce the number of deaths from this phenomenon. At velocities of about 50–70 mph, 80% of chest protectors decreased the impact force of the projectile (Viano et al, 2000). However, only 20% of commercially available protectors statistically lowered the impact force of baseballs at 40 mph (Link, 2003).

Legal implications

There are unfortunately significant legal implications with regard to commotio cordis even if the blow was not intended to cause harm or occurred during the sporting event itself. One hockey player was charged with manslaughter when his opponent died after being hit accidentally in the chest by the player's stick. Owing to such medicolegal implications, forensic pathologists must work closely with police officials and scene investigators in order to be fully confident of such a diagnosis.

Table 1. Clinical differences between cardiac concussion and cardiac contusion

Feature	Cardiac concussion	Cardiac contusion
Site	Precordial only	Chest anywhere
Direction of blow	Sternum to vertebrae	Insignificant
Force	Sharp, but not necessarily violent	Generally violent
Thoracic cage intact	Essential	Not essential
Onset	Immediate	Gradual
Course	Transitory	Lasting
Loss of consciousness	As a rule	No
Blood pressure	Frequently drops	Normal
Disturbances of rhythm and conduction	Characteristic: immediate	Absent or delayed
Changes in ST segment and T wave	Generally absent	Always present

From Koehler et al (2004)

Conclusions

Cardiac concussion is a rare unfortunate consequence of chest trauma seen commonly in sports such as baseball and ice hockey. Death is common from ventricular fibrillation. Early defibrillation is therefore essential in helping restore individuals to normal sinus rhythm. However, this is not always possible. Athletes and sport manufacturers must therefore appreciate the severity of this cardiac injury. By taking preventative measures, such as more appropriate use of protective chest padding as well as safer sporting equipment, the risk of death from commotio cordis may be significantly reduced. **BJHM**

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