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CASE REPORT

S. J. Hamilton · J. P. Sunter · P. N. Cooper

Commotio cordis–A report of three cases

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Abstract Commotio cordis is a recognised cause of sudden death in which an apparently minor blow to the chest causes ventricular fibrillation and cardiac arrest. It is best known for causing death during games of youth baseball in the United States, but individual cases have been recorded as a result of a wide range of activities, principally sporting. The underlying biochemical and mechano-electric causes have been well documented. However, there are few reported cases where commotio cordis is implicated as the cause of death in homicide cases. We present three cases from the north-east of England where an assault caused death by this mechanism.

Keywords Commotio cordis · Homicide · Chest trauma · Mechano-electric feedback

Introduction

Commotio cordis (concussion of the heart) is a condition in which a seemingly minor blow to the chest causes ventricular fibrillation (VF), cardiac arrest and sudden death. Typically, autopsy reveals no cardiac lesion and little, if any, external signs of the blow. Nesbitt et al. have proposed the following as a definition: "Commotio cordis: a mechanical stimulation of the heart by non-penetrating, impulse-like impact to the precordium that, through intrinsic cardiac mechanisms, gives rise to disturbances of cardiac rhythm of various type, duration, and severity, including sudden cardiac death, in the absence of structural damage which would explain the observed effects." [1].

The condition has been recognised for well over a century, with case reports in the literature from 1876 [2] and 1879 [3]. Indeed, some fanciful speculation suggests that the legendary dim mak or "death touch" of oriental martial arts is actually a homicidal use of this phenomenon! (see http://martialarts.jameshom.com/library/weekly/aa033102a.htm).

In recent years, this hitherto little known condition has gained in prominence after it was recognised that it is a far from insignificant cause of death in young people involved in sporting activities. A consumer product safety commission report in 1996 identified 38 deaths from baseball strikes to the chest in the period 1973–1995 [4]. Abrunzo suggested that commotio cordis was the single most common cause of death in youth baseball [5]. Maron et al. at the commotio cordis registry in Minneapolis have identified 128 cases as of their last report, 107 of which were sports-related. Baseball was the most common in the series, but hockey, American football, soccer, lacrosse, karate, cricket and rugby were also implicated [6]. The majority of the others were tragic accidents, but eight cases involved either parent-child chastisement or the rather enigmatic "gang rituals" and seven of these led to homicide convictions.

The underlying mechanisms of commotio cordis have been extensively researched, and as long ago as the 1930s, Schlomka identified that the type, force and location of impact were of cardinal import [7]. More recently it has been recognised that timing of impact is also very important [8]. Blows occurring during the upstroke of the T wave have been shown to induce VF, and those coinciding with the QRS complex can lead to ST elevation (one has to imagine that martial arts experts would have to be spectacularly talented to time a blow to coincide exactly with the upstroke of the T wave!).

Link et al. have implicated sudden activation of the K^{+}_{ATP} channel in the pathogenesis of commotio cordis in an experimental swine model [9, 10], and from this a model of channel activation and subsequent arrhythmia by mechano-electric feedback has emerged [8].

Despite the extensive literature on sports-related events, there is limited published material on commotio cordis in forensic practice. Aside from the Minnesota work, Frazer

S. J. Hamilton (✉) · J. P. Sunter · P. N. Cooper
Forensic Medicine Unit University Department of Pathology,
Royal Victoria Infirmary,
Newcastle upon Tyne, NE1 4LP, United Kingdom
e-mail: Margaret.brown@nuth.northy.nhs.uk
Tel.: +44-191-2227169
Fax: +44-191-2223416

and Mirchandani have presented four case reports [11], and Denton and Kalelkar reported two cases in children [12]. Baker et al. also detail a case of fatal child abuse involving the condition [13]. Here, we detail three cases of commotio cordis fatalities as a result of assaults in the north-east of England between 1990 and 2003.

Case reports

Case 1

The victim, A, was a 31-year-old male who had been drinking in a public house in a notorious suburb of a large industrial city. He was intoxicated and belligerent and became involved in an altercation with a group of men at a nearby table. This spilled out into the car park. During the melee, A was struck in the chest with a pool cue. He was seen to stagger a few yards then to collapse unconscious. An ambulance arrived quickly, but no pulse could be detected and he had fixed, dilated pupils. Despite resuscitation attempts both at the scene and in hospital, A was declared dead at 21.30 p.m., approximately half an hour after the incident.

At autopsy, there was a rectangular area of bruising measuring 150×30 mm which had a "tramline" appearance and lay approximately over the course of the fifth rib on the left side of the chest. There was also some ill-defined bruising in the centre of the chest. Deep to the lateral end of the injury there were fractures of the 4th, 5th and 6th ribs. The left lung showed minor interstitial bleeding but the heart itself showed no evidence of injury or natural disease. No head injury was present, nor was there any other natural disease which would account for sudden death. There were no other internal injuries and the abdominal organs were unremarkable. Histology revealed no abnormality of the heart, spleen, kidneys, pancreas or adrenal gland. The liver showed mild steatosis and the presence of mild interstitial bleeding in the lung was confirmed. Pulmonary oedema was present. The cause of death was given as acute cardiac inhibition due to a heavy blow to the left side of the chest.

Case 2

The deceased, B, was a 26-year-old man who had been drinking alcohol with a friend in the friend's house. B left the house alone and was on his way to a nearby shop when he was assaulted by three individuals. The friend stated that one of the assailants struck B with a wooden stick. An ambulance was called and arrived 3 min later, but B already appeared to be dead. Resuscitation was attempted, including pericardiocentesis, but to no avail. Death was confirmed just over half an hour after the incident.

In this case, autopsy revealed numerous bruises and abrasions all over the body. These were all superficial and not apparently life-threatening. Of most significance, there was an area of bruising 70×30 mm below the left nipple. Marks of medical intervention were present, including a needle puncture wound just to the left of the midline of the chest. No significant head injuries were present and the skull was not fractured. The brain was not swollen or injured. Beneath the chest injury noted above there was extensive bruising. There was a needle puncture wound in the pericardium consistent with the external puncture, and the needle track penetrated the apex of the heart into the right ventricle with surrounding bruising. Fluid and clotted blood weighing 390 g was present in the pericardial sac. No cardiac lacerations were present and other than a 1 cm area of extremely superficial bridging of the left anterior descending artery, no pathology was present. No other significant internal injuries were present. Histology of the adrenal gland, kidneys and liver was unremarkable. The lungs were oedematous. The myocardium showed no intrinsic abnormality, but the presence of myocardial damage and haemorrhage where the pericardiocentesis needle had entered the heart was noted. Toxicology revealed a blood ethanol of 152 mg/dl and urine ethanol of 198 mg/dl. Blood and urine both tested positive for cannabis. No other drugs were detected. A weapon was recovered which matched the wooden implement described by the witness. The cause of death was given as a blunt impact to the front of the chest.

Case 3

The victim in this case, C, was a 22-year-old male who was found dead in the early hours of an autumn morning in the grass square of a housing estate. An ambulance crew were called but he was clearly dead. Not long prior to the discovery of the body, a group of youths were seen carrying stick-like objects, and another report was received of a group (presumably the same group) hitting each other with sticks. At autopsy there were abrasions and bruises, predominantly on the face and torso. Of particular note, there were two rectangular bruises on the right side of the chest and a square ended mark to the left of the midline, 100 mm below the sternal notch. There was also a rectangular mark, 30×23 mm positioned 95 mm below the sternal notch and 55 mm to the left of the midline. Close to this there was irregular bruising measuring 40×25 mm positioned 135 mm to the left of the midline and 85 mm below the sternal notch. Internal examination revealed a fracture of the left 5th rib underlying these injuries but no pathological changes within the heart and no evidence of trauma. Scattered petechiae were present on the epicardium. There was some vomit in the airways. Histology of the heart, liver, kidneys, adrenal, spleen and pancreas was normal. Ethanol was detected at a level of 195 mg/dl in blood. Toxicology also revealed disulfiram at therapeutic levels and a trace amount of cannabis in the blood. The cause of death was given as reflex cardiac inhibition due to a blow or blows to the chest.

Discussion

These cases all concern incidents in which the victims died almost immediately following a blow to the chest. Autopsy findings demonstrated neither injuries which would immediately explain the collapses suffered by these individuals nor was there natural disease that would explain their sudden demise. There was, however, in each case either a clear history of a blow to the chest or good evidence at autopsy that this had occurred. The injuries can be seen to conform to Nesbitt et al.'s non-penetrating, impulse-like blow to the precordium. It cannot, of course, be confirmed whether the impact occurred during the upstroke of the T wave but it is reasonable to infer that the mechanism of death in these individuals was commotio cordis. The actual term "commotio cordis" was not used in the cause of death statement in order to make the cause of death more understandable to non-medical investigators and to the court.

In case 1, there were fractured ribs suggesting a greater degree of force than is often seen in death by this mechanism, but the heart itself did not show bruising. In case 2, cardiac bruising and haemopericardium were present. However, there was a single needle puncture wound to the chest and the only episode of penetration of the chest was from pericardiocentesis, when B was already in a state of cardiac arrest following the blow to the chest. Case 3 is a little less clear as the fatal episode was not witnessed, but the presence of three clear marks on or around the cardiac shadow along with the absence of any other findings to explain the death make commotio cordis by far the most likely mechanism of death. The presence of vomit in the airways is a common finding at autopsy and is rarely of significance. In this case, it was felt that this finding was due to a combination of agonal regurgitation and resuscitation attempts.

It is interesting to note that all three of the decedents had been drinking alcohol prior to their death, as it is known that alcohol sensitises the heart to catecholamines and arrhythmia [14]. It is also interesting to note that although in two of the cases (A and B), prompt resuscitation attempts were performed, they were unsuccessful. This reflects the poor prognosis commonly observed in such cases [15].

Homicide cases where death is believed to come about as a result of commotio cordis are relatively rare in the literature, certainly in comparison to reported cases in, say, youth baseball. This may partly reflect the fact that the circumstances surrounding a homicide are often less clear than a sporting accident, without clear witness evidence. In a case where a body is found with no particular external injuries and no history of a blow being struck, the presence of concurrent disease, particularly diseases which predispose to cardiac arrhythmiae, such as severe coronary artery atheroma, valvular disease or cardiomyopathies (amongst others) [16], may both predispose the victim to a sudden cardiac death and suggest a cause of death which does not necessarily involve violence. In addition, the presence of drugs (particularly stimulants such as cocaine or amphetamine) may cause catecholamine release which increases the likelihood of arrhythmia [17]. Similarly, the sympathetic activation which usually accompanies an altercation releases catecholamines and may predispose to arrhythmia. Such factors make the true cause of death much more difficult to elucidate than that of a healthy young person with negative toxicology collapsing after being struck by a ball in front of a crowd of witnesses. However, careful examination of the precordium may reveal injuries which could at least suggest the possibility of an impact. Needless to say, in the absence of witness evidence, the diagnosis of commotio cordis should be made with care.

In a witnessed assault, the temporal association between the blow to the chest and the subsequent collapse, are strongly suggestive of commotio cordis as the fatal event but even in these cases, pre-existing cardiac disease, drugs and heightened sympathetic activity may predispose to cardiac arrhythmia and may be contributory factors in death.

Commotio cordis is an uncommon cause of sudden death, but one which has the potential to occur in almost any episode involving a blow to the chest and should therefore be borne in mind by the forensic practitioner.

References

1. Nesbitt AD, Cooper PJ, Kohl P (2001) Rediscovering commotio cordis. Lancet 357:1195–1197
2. Nelaton A (1876) Elements de pathologie chirurgicale, 2nd edn. Librarie Germer Bateliere, Paris
3. Meola F (1879) La commozione toracica. G Int Sci Med 1:923–937
4. Adler P, Monticone RC (1996) Injuries and deaths related to baseball. In: Kyle SB (ed) Youth baseball protective equipment project final report. Consumer Product Safety Commission, Washington DC, pp 1–43
5. Abrunzo TJ (1991) Commotio cordis: the single most common cause of traumatic death in youth baseball. Am J Dis Child 145:1279–1282
6. Maron BJ, Gohman TE, Kyle SB, Estes NA 3rd, Link MS (2002) Clinical profile and spectrum of commotio cordis. JAMA 287:1142–1146
7. Schlomka G (1934) Commotio cordis und ihre Folgen. Die Einwirkung stumpfer Brustwandtraumen auf das Herz. Ergebn Inn Med Kinderheilkd 47:1–91
8. Kohl P, Nesbitt AD, Cooper PJ, Lei M (2001) Sudden cardiac death by commotio cordis: role of mechano-electric feedback. Cardiovasc Res 50:280–289
9. Link MS, Wang PJ, VanderBrink BA, Avelar E, Pandian NG, Maron BJ, Estes NA 3rd (1999) Selective activation of the K^{+}_{ATP} channel is a mechanism by which sudden death is produced by low-energy chest-wall impact (commotio cordis). Circulation 100:413–418
10. Link MS, Wang PJ, Pandian NG 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
11. Frazer M, Mirchandani H (1984) Commotio cordis, revisited. Am J Forensic Med Pathol 5:249–251
12. Denton JS, Kalelkar MB (2000) Homicidal commotio cordis in two children. J Forensic Sci 45:734–735
13. Baker AM, Craig BR, Lonergan GJ (2003) Homicidal commotio cordis: the final blow in a battered infant. Child Abuse Negl 27:125–130
14. Zakhari S (1991) Vulnerability to cardiac disease. Recent Dev Alcohol 9:225–260
15. Link MS, Ginsburg SH, Wang PJ, Kirchhoffer JB, Berul CI, Estes NA 3rd, Paris YM (1998) Commotio cordis: cardiovascular manifestations of a rare survivor. Chest 114:326–328
16. Antezano ES, Hong M (2003) Sudden cardiac death. J Intensive Care Med 18:313–329
17. Karch SB (2002) Pathology of drug abuse, 3rd edn. CRC Press, Boca Raton, FL