CASE REPORT

A patient with commotio cordis successfully resuscitated by bystander cardiopulmonary resuscitation and automated external defibrillator

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Sudden deaths of children and adolescents during competitive sports are usually due to congenital heart diseases. Ventricular fibrillation, however, may also occur in individuals with no underlying cardiac disease who have sustained a low-impact chest wall blow. This phenomenon is described as commotio cordis, and the overall survival rate is poor. Successful resuscitation can be achieved by prompt cardiopulmonary resuscitation and early defibrillation. We report a teenager who sustained a chest wall blow that resulted in a cardiac arrest during a rugby competition. Cardiopulmonary resuscitation was given by bystanders. The ambulance crew arrived with an automated external defibrillator. Ventricular fibrillation was detected and responded to defibrillation. Subsequent investigations including imaging and electrophysiological studies did not reveal any cardiac or brain abnormality, and the patient recovered well neurologically. Accessible cardiopulmonary resuscitation–trained personnel and automated external defibrillators should be present at all organised sporting events.

Introduction

Sudden cardiac death as a result of a blunt, and often apparently innocent, chest wall blow, is known as commotio cordis (CC). The condition is rare, but is reported with increasing frequency, with an overall survival rate of only 16%.[1] The survival rate can be improved with prompt resuscitation. This report is of a teenager who sustained CC after a minor chest wall blow, and illustrates the advantages of having readily available access to cardiopulmonary resuscitation (CPR) and an automated external defibrillator (AED).

Case report

A healthy 14-year-old male sustained a chest wall blow by the elbow of an opponent in a college rugby competition in July 2007. He had walked for a few steps before he suddenly collapsed. He had no pulse or respirations. Bystander CPR of chest compressions and bag-valve mask ventilation were immediately started by the St John First Aid providers and a CPR-trained coach. The emergency medical system was activated and the paramedics arrived 4 minutes after the incident. Cardiopulmonary resuscitation was continued by the paramedics and an AED detected ventricular fibrillation (VF) [Fig 1a]. Defibrillation at biphasic 200 joules was given once, and idioventricular rhythm was noted (Fig 1b). Spontaneous circulation with sinus rhythm was returned subsequently (Fig 2) about 11 minutes after the cardiac arrest. Due to persistent unresponsiveness, bag-valve mask ventilation with an oropharyngeal airway was continued and the patient was transferred to the Department of Accident and Emergency Medicine, Caritas Medical Centre, Hong Kong.

On arrival at the emergency department, the patient was in a coma with a blood pressure of 133/66 mm Hg and pulse of 130 beats/min. He was breathing spontaneously and oxygen saturation was 99% with a non-rebreathing mask. In view of his continuing

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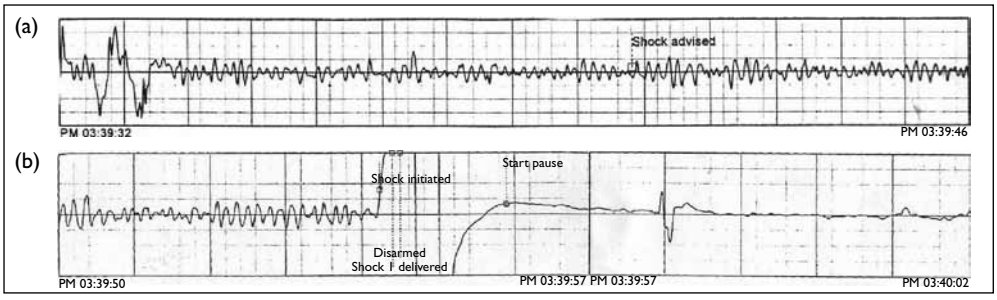

FIG 1. (a) Initial rhythm strip demonstrating ventricular fibrillation. (b) Rhythm strip showing defibrillation with 200 joules and idioventricular rhythm after defibrillation

一宗由旁人進行心肺復甦及用自動體外去顫器成功救活的心臟震盪個案

劇烈運動中無論是兒童或成人突然死亡通常都是因先天性心臟病引致。不過，在胸壁受到撞擊後，即使沒有患心臟病的人也可能出現心室顫動，這情況被稱為心臟震盪。心臟震盪的總生存率低，但只要及時進行心肺復甦和及早使用去顫器都可成功挽救病者。本文報告在一場欖球賽事中一名青年因胸壁受到撞擊引致心博停止。病人即時由旁人進行心肺復甦。救護人員抵達時發現病者有心室顫動，即時用自動體外去顫器進行去顫術，病人對去顫術有反應。後為病人進行影像學及電生理檢查，未有發現任何心臟或腦部異常，而病人神經學功能方面亦恢復良好。所有運動賽事都應安排受過心肺復甦訓練的人員在場，並隨時準備自動體外去顫器。

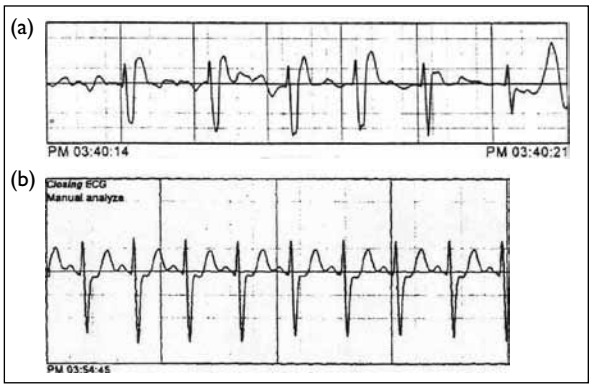

FIG 2. Rhythm strip demonstrating subsequent conversion to sinus rhythm after defibrillation (a) 1 minute later (b) 14 minutes later*
* ECG denotes electrocardiography

unresponsiveness, the patient was intubated. Physical examination of the cardiovascular system was unremarkable. Initial electrocardiography (ECG) only revealed sinus tachycardia, with no significant ischaemic change, and the chest radiograph was normal. A paediatrician was consulted. The patient was stabilised and transferred to the paediatric intensive care unit (PICU) for further treatment. Electrocardiography after admission showed sinus tachycardia with QTc of 448 ms and ST elevation in leads V1 to V3. Initial laboratory investigations revealed increased creatine kinase level of 2850 (reference range, 50-200) U/L. Initial echocardiography on the day of admission showed fair right ventricular contraction and left ventricular apical hypokinesia, with an ejection fraction of 50%. Computed tomography (CT) of the brain was normal. The patient was extubated soon after admission with good respiratory effect. He was fully awake on day 3, and neurological examination did not reveal any significant abnormality except mild intention tremor. Repeated blood tests showed that all cardiac markers were decreasing. The patient was transferred from the PICU to a general ward on day 3. The patient recovered well, and except for mild deterioration in hand-eye coordination, his functional status returned to normal. The patient was discharged on day 7, and was followed up by a cardiologist and a neurologist.

All subsequent cardiac investigations of echocardiography, 24-hour Holter monitoring, exercise treadmill test, coronary CT angiogram, and cardiac magnetic resonance imaging were normal. Exercise myocardial perfusion test showed no focal perfusion defect in the left ventricle at peak exercise with an ejection fraction of 65%. The patient did not have any arrhythmias in subsequent ECGs, and had normal QTc interval and ST segment. A clinical diagnosis of CC was made by a cardiologist. The patient returned to school 2 months after the event, at the start of a new term with satisfactory performance. He had satisfactory exercise tolerance and continued to play rugby with no major cardiac or neurological sequelae.

Discussion

Commotio cordis, sudden death as a result of a blunt innocent-appearing chest wall blow, has been reported with increasing frequency, and ranks as the second leading cause of death among young athletes who do not have heart disease.[2] Among 128 cases in the US Commotio Cordis Registry, 62% occurred during organised or recreational sporting activities such as baseball, softball, and hockey.[1] In most of the incidences, the victims were struck by projectiles that are regarded as standard implements of the game. Other cases occurring in normal daily life were brought about by unintentional and innocent chest wall blows, although some were due to criminal behaviour.[1]

Affected patients typically have neither underlying heart disease nor structural damage to the heart, chest wall, or thoracic cavity. An absence of morphologic cardiac or intrathoracic injury distinguishes CC (cardiac concussion) from contusio cordis (cardiac contusion with evidence of structural damage).[3] The likely cause of death in CC is VF, although complete heart block and other arrhythmias have also been described.[4] The mechanism of impact-induced VF is currently under investigation, and the available evidence[2] suggests that the underlying mechanism is multifactorial. Three main determinants are timing, location, and force of the blunt chest impact. Some investigators believe that this explanation is too straightforward and the mechanism of sudden death due to CC is more complex, and includes components of coronary artery vasospasm, myocardial contusion, or both.[5] This complex theory attempts to provide a rationale for those patients who have been successfully resuscitated. However, the development of an experimental swine model in recent years has provided better insight into the pathophysiology

of CC.[4] Angiography performed immediately after impact did not reveal any evidence of stenosis or spasm in the epicardial coronary arteries.[6] Myocardial perfusion imaging with technetium-99m sestamibi performed after impact revealed only small, mild apical defects in a few of the animals tested. Left ventriculograms and echocardiograms performed immediately after impact revealed mild apical or distal septal hypokinesis in regions distant from the area of precordial impact. Pathological examination has not revealed structural heart damage for impact velocities of less than 50 miles per hour (mph).

Multiple animal experiments have shown that development of VF requires precise timing of chest wall impact.[4] When the chest wall impact occurred in a narrow window of the cardiac cycle at 10 to 30 ms before the peak of the T wave, VF was consistently produced.[4] The site of impact was also studied using echocardiographic guidance. Commotio cordis occurred commonly with blows directly over the centre of the cardiac silhouette,[7] as the compliant chest walls in young people enabled greater transmission of impact energy to the myocardium. Impacts at sites that did not overlay the heart would not result in VF or other electrophysiological effects.

The risk of cardiac arrest after a chest blow is related to the force of the impact, with force being a function of both the mass and acceleration of the object. The likelihood of VF is proportional to the hardness of the projectile.[7] The impact velocity is also important. In an experimental swine model, Link et al[8] showed that the impact velocity relative to the incidence of VF exhibited a Gaussian relationship. The incidence of VF increased from no episodes at 20 mph through 7% at 25 mph to a maximum of 68% at 40 mph.

The current survival rate for resuscitation is only about 16%, which is likely to be due to lack of early recognition and failure to initiate timely aggressive resuscitation. Survival is most likely to occur if institution of CPR and defibrillation are available within 3 minutes of the incident.[2] Animal studies have shown that defibrillation with an AED within 1 and 2 minutes of ventricular resuscitation resulted in 100% and 92% successful resuscitation rates, respectively.[9] After 6 minutes, the survival rate dropped to 25%. Therefore, more widespread access to an AED at organised youth sporting events and training of AED users are strongly encouraged. After the incident reported here, the organiser of the local college rugby competition and St John Ambulance Association purchased an AED, and trained AED users are required for all forthcoming college rugby competitions.

Deaths from sports-related CC are tragic. Several approaches for the prevention of CC have been proposed. The softness of balls used in sports has been studied and balls with a softer core could reduce triggering VF from 69% to 11%.[10] Chest wall protectors have also been studied, but the value was not well defined. None of the currently available chest wall protectors was shown to decrease the rate of VF.[11] However, it is reasonable to suspect that maintaining coverage of the entire heart during athletic activities is protective.

Commotio cordis is a rare, but devastating event that can result from blunt chest trauma in any teenager or child, even those who are previously healthy. Prompt bystander CPR and rapid detection of VF with early defibrillation was the key to the successful outcome for this patient. These authors suggest that CPR-trained instructors and AEDs should be present at all organised sporting events in this area.

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