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【Review】

Commotio Cordis: importance of awareness

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Abstract

Commotio Cordis, primarily reported by B. J. Maron since 1990, is one of the causes of sudden cardiac death. In large autopsy-based surveys performed in the United States, hypertrophic cardiomyopathy has consistently been the single most common cardiovascular cause of sudden death, accounting for high incident rates in prior reports. However, it has been suggested that the prevalence of these events is not as high in younger individuals. Commotio cordis is the second most frequent cause of sudden cardiac death, with approximately 10 to 20 cases added to the Commotio Cordis Registry yearly since the late 1990s.

In patients with commotio cordis, ventricular fibrillation or complete A-V block is recognized before cardiac arrest. In recent years, survival from these incidents has increased, likely because of more rapid response times and access to defibrillation devices, as well as greater public awareness of this condition. In addition, automatic external defibrillator availability has contributed to this increase.

I Introduction

Athletes are often considered to be some of the healthiest people in society, and sudden cardiac death (SCD) in a young athlete is shocking and profoundly impacts the school or community where it occurs. However, it has been suggested that the prevalence of these events is not as high as it may seem as compared to the considerable media attention that often accompanies SCD in a young individual.[1-3] On the other hand, SCD is the leading medical cause of death in college athletes in the United States, as well as the most common cause of death during sport or exercise participation, and occurs at a much higher rate than previously thought. The American College of Cardiology defines SCD as "nontraumatic and unexpected sudden death that may occur from a cardiac arrest, within 6 hours of a previously normal state of health".[2, 4]

A variety of cardiovascular abnormalities represent the most common causes of sudden death in competitive athletes, such as dilated cardiomyopathy, aortic rupture in the context of Marfan syndrome, myocarditis, valvular disease (aortic stenosis, mitral valve prolapse), and electrical disorders (Wolff-Parkinson-White syndrome, long QT syndrome, Brugada syndrome), as well as commotio cordis (malignant arrhythmia due to blunt chest trauma).[5]

Commotio cordis, a cause of SCD, has primarily been reported by B. J. Maron.[1, 2, 4, 6, 7] In those studies, sudden death from cardiac arrest that occurred during sports play after a blunt blow to the chest in the absence of structural cardiovascular disease or traumatic injury (cardiac concussion or commotio cordis) was studied in young cases, and the clinical features of this apparently uncommon but important phenomenon

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were reported. The global occurrence of commotio cordis is very similar in regard to demographics is very similar in the United States and other countries.[7] However, the frequency of chest blows from soccer balls causing commotio cordis events, particularly during sports events played internationally, seems to contradict the prevailing notion that air-filled projectiles convey less risk for ventricular fibrillation than those with solid cores (e.g., baseballs or lacrosse balls). In Japan, some cases of Commotio cordis have also been reported to be caused by a fall[8, 9] or traffic accident,[10, 11] as well as sports activities.[12] As the reported papers of commotion cordis in Japan on sports activities is only one paper[12], we investigated reports of commotio cordis including those presented in Japan and provide here an overview.

Ⅱ Epidemiology

In large autopsy-based surveys of a total of 387 athletes performed in the United States, hypertrophic cardiomyopathy was consistently the single most common cardiovascular cause of sudden death, accounting for the high incidence noted in prior reports[13-15] (Table 1). However, it has been suggested that the prevalence of these events is not as high as it may seem, because of the considerable media attention that often accompanies SCD in a young individual.[1, 2] In those studies, commotio cordis was the second most common, occurring in 20% of those cases (Table 1).

Commotio cordis is generally seen in younger individuals, though not exclusively.[16-18] In another study, the condition showed a predilection for children and adolescents (mean age, 15 ± 9 years; range, 6 weeks to 50 years).[20] Among 224 cases over the past 15 years,[6, 21, 22] 26% of the affected individuals were younger than 10 years of age, whereas only 9% were 25 years or older. Commotio cordis has rarely been reported in blacks or females, as most victims are male (95%) and white (78%).

Table.1 Cause of sudden death in 387 young athletes[13)-15)]

Cause	No. (%)
Hypertrophic cardiomyopathy (HCM)	102 (26.4)
Commotio cordis	77 (19.9)
Coronary artery anomalies	53 (13.7)
Left ventricular hypertrophy of indeterminate causation (autopsy: suggestive of HCM (nsufficient for diagnosis)	29 (7.5)
Myocarditis	20 (5.2)
Ruptured aortic aneurysm	12 (3.1)
Arrhythmogenic right ventricular cardiomyopathy	11 (2.8)
Tunneled (bridged) coronary artery	11 (2.8)
Aortic-valve stenosis	10 (2.6)
Atherosclerotic coronary artery disease	10 (2.6)
Dilated cardiomyopathy	9 (2.3)
Myxomatous mitral-valve degeneration	9 (2.3)
Asthma (or other pulmonary condition)	8 (2.1)
Heat stroke	6 (1.6)
Drug abuse	4 (1.0)
Other cardiovascular cause	4 (1.0)
Long QT syndrome (documented on clinical evaluation)	3 (0.8)
Cardiac sarcoidosis	3 (0.8)
Trauma involving structural cardiac injury	3 (0.8)
Ruptured cerebral artery	3 (0.8)

Although cardiovascular collapse is virtually instantaneous, 20% of affected individuals remain physically active for a few seconds after the blow (e.g., continuing to walk, run, skate, throw a ball, or even speak), which may reflect individual tolerance for sustained ventricular tachycardia accompanied by arrhythmia. For example, a baseball pitcher struck in the chest by a batted ball was able to retrieve the ball at his feet, successfully complete the play (throwing out the base runner), and then prepare for the next pitch before collapsing. In another instance, a batter playing baseball was struck by a pitch while attempting to bunt and collapsed only after running to first base.[20] In such cases, ventricular tachycardia accompanied by arrhythmia appears

first, after which cardiac arrest occurs, with cerebral and body blood flow continuing for a few or several seconds.

Fifty percent of reported commotio cordis events have occurred in young competitive athletes participating in a variety of amateur sports after receiving a blow to the chest wall that is usually (but not always) delivered by a projectile used to play the game. In baseball, for example, commotio cordis is often triggered when players are struck in the chest by balls that have been pitched, batted, or thrown in a variety of scenarios[20] (Table 2).

Approximately 10 to 20 cases are added to the Commotio Cordis Registry each year,[20, 23] though it was only rarely reported until the late 1990s. It is thought that this increase in number of cases is not due to an increase in incidence, but rather greater awareness following a *New England Journal of Medicine* report on commotio cordis presented in 1995,[6] with many more cases subsequently recognized. Furthermore, what was once thought to be a uniquely North American phenomenon is increasingly being reported in countries outside the United States.[24] In recent years, survival from commotio cordis has increased, likely owing to more rapid response times and access to defibrillation devices, especially during high-risk sports events, as well as greater public awareness of this condition.[25, 26]

Table.2 Example circumstances in which chest blows have triggered commotio cordis (some parts omitted or reorganized)[20]

Sports
Baseball Softball Cricket Football Soccer Hockey Lacrose
Fights and scuffles, with blow from hand or elbow
Psychiatric aide struck by patient Teacher struck while restraining student during fight Youth struck during play-shadowboxing or roughhousing Youth struck by boxing glove during sparing Child struck by parent or babysitter (with disciplinary intent) Young adult struck during slam dancing Student involved in fist fight at fraternity party Youth hit by snowball Adult struck in prison gang initiation ritual Infant struck with open hand while having diaper changed
Other circumstances
Child kicked by horse Youth hit with recoil of gun butt while deer hunting Child hit with rebound of playground swing Adult thrown against steering wheel during automobile accident Youth hit by tennis ball filled with coins Young adult kicked during cheerleading routine Adult received chest blow by falling into body of water Child received blow from head of 23-kg (50-lb) pet dog Child received blow from falling on playground apparatus Child hit by tossed hollow plastic bat Child hit by plastic sledding saucer Youth received blow intended to terminate hiccups Child hit handlebars while falling off of bicycle

Ⅲ Mechanism

Ventricular fibrillation[11, 12, 24] and complete A-V block[8, 9, 26] have been recognized prior to cardiac arrest in patients with commotio cordis. When a projectile strikes an area near the heart, the initial depolarization trigger is likely a focal phenomenon from the direct impact, similar to a premature ventricular contraction induced by a catheter, as seen in electrophysiology and cardiac catheterization laboratories. Alternatively, it could be occur after depolarization induced by changes in current flow. This 2-step process is similar to the R-on-T phenomenon, in which a premature ventricular contraction that occurs on the upslope of the T wave will cause ventricular fibrillation in acute ischemic conditions, but not in nonischemic situations.[24] In addition, that study noted that impacts occurring throughout the cardiac cycle may cause ST-segment elevation and left bundle-branch block in impact events in which ventricular fibrillation is not induced.[24]

Moderate precordial impact is particularly effective in triggering instant ventricular fibrillation if

delivered during the early T-wave of the ECG. This timing is identical to the 'vulnerable period' for electrical induction of ventricular fibrillation, initially identified in 1936,[27, 28] and raises questions as to what mechanism would: (i) allow near-instantaneous translation of a mechanical stimulus into an electro-physiologically relevant signal, which is (ii) powerful enough to cause serious rhythm disturbances and that is (iii) particularly potent in doing so during the T-wave. Obviously, that set of questions might be inappropriate, as the link between the timing of a mechanical impact and its electrophysiological effect may be primarily related to the mechanical rather than the electrical cycle of the heart.

It is not difficult to imagine that the extent of background tissue strain and/or dimensions of the cardiac chambers determine susceptibility to mechanical induction of ventricular fibrillation. Although maximum filling of the ventricles coincides with the PQ segment, that does not explain the observed peak sensitivity to mechanical induction of ventricular fibrillation during the T-wave.[29]

Causative theories regarding a predisposition to ventricular fibrillation include mechanical electrical coupling. A sudden myocardial stretch can be elicited when an external blow occurs during a vulnerable window that is based on repolarization inhomogeneity and stretch pulses applied during this vulnerable window can lead to nonuniform activation. Repolarization dispersion might play a crucial role in the occurrence of fatal tachyarrhythmia during commotio cordis.[30] In an experimental model of commotio cordis that utilized anesthetized juvenile swine, ventricular fibrillation was produced by a baseball travelling at 30 mph when the strike occurred during the vulnerable period of repolarization, on the upslope of the T-wave (Fig. 1, 2). [29] Energy from the impact object was also found to be a critical variable with baseballs travelling at 40 mph, while ventricular fibrillation was more likely with velocities less or greater than 40 mph. In addition, more rigid impact objects and blows directly over the center of the chest more often caused ventricular fibrillation. Peak left ventricular pressure generated by a chest wall blow was found to be correlated with the risk of ventricular fibrillation, with activation of the K (+) (ATP) channel a likely cause of that fibrillation.[24, 31]

Successful resuscitation is attainable with early defibrillation,[32] and initiation of ventricular fibrillation may be mediated by an abrupt and substantial increase in intracardiac pressure.[33] The energy of the chest-wall impact is an important variable in the generation of ventricular fibrillation and impacts at 40 mph were more likely to produce ventricular fibrillation than those with greater or lesser velocities, suggesting that the predilection for commotio cordis is related in a complex manner to the precise velocity of the chest-wall impact.[34] These swine studies also showed that a rapid rise in left ventricular pressure to between 250 and 450 mm Hg may mediate electrophysiological consequences, as stretch channels may be activated by a specific degree of myocardial stretch.[30-35] On the other hand, in another swine experimental model of commotio cordis, blockade of the K (+) (ATP) channel reduced the incidence of ventricular fibrillation and magnitude of ST-segment elevation, indicating that selective K (+) (ATP) channel activation may be a pivotal mechanism in sudden death resulting from a low-energy chest-wall trauma in young individuals occurring during sporting activities.[24,31]

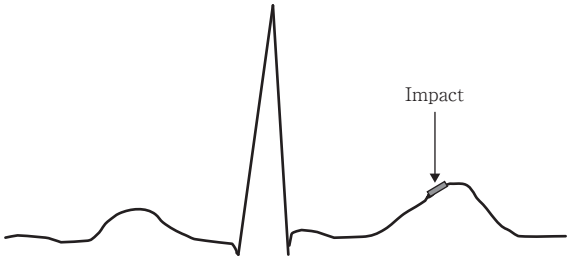

Fig. 1 Schematic representation of effects of pre-cordial mechanical stimulation on cardiac rhythm.
Kohl P et al. Cardiovasc Res 2001;50:280-289

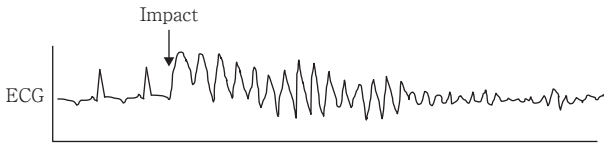

Fig. 2 ECG obtained from an anaesthetised pig subjected to a pre-cordial impact that coincided with the upstroke of the T-wave.
(Kohl P et al. Cardiovasc Res 2001;50:280-289.)

Ⅳ Protection and Prevention

Commotio cordis is a devastating cause of sudden death in young and healthy individuals, in which ventricular fibrillation is the most common arrhythmia.[23] Subsequent survival rates are dismal. Increased awareness of this phenomenon is imperative, especially among those who may be first responders, such as parents, coaches, game officials, and medical personnel. Current commercially available chest barriers are not sufficiently effective in preventing chest-blow-induced sudden cardiac death and, in fact, probably offer only a false sense of security to athletes, families, and the general public.[36] When soft safety baseballs is used, the rate of induction of VF was at its lowest with warring chest protection.[37] However, preventive chest wall official using protectors produced for baseball catchers and lacrosse players are important, though current designs are not impeccable. Safer and more appropriate protectors to be worn while playing sports are needed. We believe that Kevlar body armor with ceramic plate inserts[38] for the partial left chest wall might be able to protect commotio cordis.

Prevention may be enhanced through the use of soft "safety" baseballs and improved chest protector design. Ready availability of automated external defibrillators (AEDs) at youth sport venues may also improve survival rates, as early defibrillation improves outcomes. In 7 cases of commotio cordis reported in Japan, 6 were recovered by utilization of CPR and/or an AED. In addition, cardiopulmonary resuscitation education and first-responder awareness are important factors related to survival.[31]

Ⅴ Conclusive view

In the future perspectives of this research and clinical fields based on the contents of this article, further efforts are needed to prevent avoidable deaths by providing more education concerning commotio cordis, ideal Kevlar body armor with ceramic plate inserts for the partial left chest wall for such as baseball catchers or lacrosse goalies, and wider equipment of AEDs at every organized athletic events.

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