Prevention and Management of Sudden Cardiac Death in Athletes

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Abstract

Sudden cardiac death (SCD) is the biggest challenge of all sports emergencies, as it is the leading cause of preventable deaths in both professional and recreational athletes. There is also an ongoing concern about COVID-19-associated cardiac pathology among athletes because myocarditis is an important cause of SCD during exercise. Hypertrophic cardiomyopathy represents 24% of SCD and Sudden Unexplained Death (normal heart at autopsy) represents 34% of SCD. To make sports participation safer, it is important to synergistically combine primary prevention of SCD by pre-participation identification of athletes affected by at-risk cardiomyopathies and secondary prevention with backup defibrillation of unpredictable sudden cardiac arrest on the field. The prompt application of an automated external defibrillator itself is associated with a greater likelihood of survival. With the advancement in the field of sports cardiology, the implantation of implantable cardioverter defibrillator has been promising in getting the athlete back on the field including in contact sports. Hence, knowledge of primary and secondary prevention is of great importance in reducing the incidence of SCD as well as improvising existing strategies.

Keywords: Athlete's heart, preparticipation cardiac screening, on-field death, cardiac arrest, ECG changes.

Introduction

All over the world sports-related medical fraternity and sports governing bodies dedicate a lot of time and resources to injury prevention and risk mitigation strategies. Among the list of injurypreventable conditions, sudden cardiac death (SCD) tops the list. Even among the cardiac pathologies, SCD or sudden unexplained death occupies the highest percentage in the United Kingdom registry, US military registry, and NCAA registry [1, 2, 3]. To make sports safer for both recreational and professional athletes and to push their bodies to the maximum limit, much progress is still yet to be made.

SCD is defined as natural death due to

cardiac causes, heralded by abrupt loss of consciousness within 1 h after the onset of symptoms. The mechanisms are the following: (1) Ventricular fibrillation, (2) Ventricular tachycardia and flutter with subsequent ventricular fibrillation, (3) torsade de pointe, and (4) Bradyarrhythmias and asystolic Arrest [4].

SCD is the leading cause of death in athletes during exercise and usually results from intrinsic cardiac conditions that are triggered by the physiologic demands of vigorous exercise. Current rates of SCD appear to be at least 4 to 5 times higher than previously estimated, in the range of 1 in 50,000 athlete-years overall [5]. <35 years individuals, fall into

three categories-electrical, acquired, and structural cardiac abnormality. Most of them are inherited disorders and are always quiescent and predispose the athletes to inherited SCD. Examples of these abnormalities: Structural-Hypertrophic Cardiomyopathy, Right Ventricular Cardiomyopathy, Electrical-Wolff-Parkinson-White syndrome, Brugada Syndrome, and Acquired -Myocarditis, Toxicity [1].

SCD can be a devastating condition and can present without any warning signs in seemingly normal athletes. Years of accumulated training bring about significant physiological changes in the athlete's heart. This remodeling of the heart is due to multiple factors. These changes can be demonstrated in electrocardiography. The incidence rate drastically differs ranging from 1:917000 athlete years (AY) to 1:3000 AY based on the methodological design. Among

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Table 1: Primary and secondary preventive strategies for SCD	
Primary prevention	Secondary prevention
Symptomatic history during play	Prompt on-field identification of Cardiac arrest
Family history for genetic predisposition	Early defibrillation
Routine clinical examination	Immediate hospital-based cardiac intensive care
Screening with 12 lead ECG	Implantation of ICD
Annual cardiac profiling	Tailored Cardiac rehabilitation
Awareness, and education of athletes, coaches, and	Careful decision on return to play following
parents on common cardiac pathologies	cardiac procedures

college athletes, the incidence is shown to be 1:50000 AY, and among high school athletes, it is 1:80000 AY [5]. Several studies have noted the morphology of a normal heart and these cases are referred to as sudden death syndrome, autopsy negative SCD, or sudden unexplained death [6].

Evaluating the nature of SCD in athletes needs a deeper understanding of the risk factors involved, nature of the sport, the extent of cardiac remodeling, preparticipation screening methods, and on the field/first aid measures. This narrative review gives a comprehensive knowledge of SCD in athletes and evidence-based recommendations which can be applied in clinical practice.

Risk Factors

The age group of <35 years, males > females, genetics, training years, type of training, nutrition, comorbidities, black> white, high intensity> Moderate- lowintensity sports have a higher incidence of SCD [7]. Although athlete heart includes both structural and functional adaptations, the most common autopsy findings were autopsy-negative sudden unexplained death. Hypertrophic cardiomyopathy is the most common cause of SCD 24% and sudden unexplained death (normal heart at autopsy) represents 34% according to Maron et al. [8]. The leading cause of death for players >35 years is coronary artery disease, and <35 years is sudden unexplained death. Cardio pulmonary resuscitation (CPR) resulted in a survival rate of 85% with the use of automated

external defibrillator (AED), and 35% without AED [9].

Globally, about 90 genes associated with hereditary cardiomyopathies have been identified.

The advent of the next-generation sequencing has provided an important tool in the molecular genetics of inherited cardiac diseases, providing a relatively inexpensive instrument with which to investigate a broad spectrum of genes involved in ion channel disorders and cardiomyopathy. Following conditions are proven to have positivity of genetic biomarkers: Brugada syndrome (SCN3B, ABCC9), long QT syndrome (KCNH2, KCNQ1), arrhythmogenic cardiomyopathy (PKP2), dilated cardiomyopathy (TTN, LMNA), hypertrophic cardiomyopathy (MYBPC3, MYH7), left ventricular noncompaction (MYH7), and restrictive cardiomyopathy and catecholaminergic polymorphic ventricular tachycardia $\lceil 10 \rceil$.

Athletes Heart

Cardiac adaptation is defined as a continuous process and is said to vary within the same sport. Cardiac adaptation to exercise is dimensional rather than functional. The slight decrease in EF in some athletes could be explained by cardiac dyssynchrony. Dynamic exercise leads to eccentric left ventricular remodeling and right ventricular dilatation. Cardiac MRI is very helpful in distinguishing athletes' heart from cardiomyopathy conditions such as HCM and ARVC, particularly in

extreme cases of overlap which are seen in elite athletes. Enlargement of cardiac chambers, a modest increase in wall thickness with preserved systolic function, normal or enhanced diastolic function, and normal strain measurements support adaptive changes [11].

The endurance athlete will develop morphological, functional, and electrical characteristics of the athletic heart and for a small minority this will place them in a diagnostic "grey zone."

In the vast majority of endurance athletes, the chronic accumulation of acute exercise stress will produce a healthy, physiological adaptation [12]. Regular sports participation and training causes a decrease in resting heart rate due to increase in the vagal tone [13].

According to a modern definition of athlete's heart, the athlete's heart should no longer be defined by outdated concepts of volume and pressure load. The effect of strength and power training on cardiovascular adaptation has repeatedly been demonstrated to be manifold less important than exercise intensity and exercise duration. Thus clinicians must consider a much simpler appreciation of the athlete's heart, defined by the two simple variables of exercise intensity and duration. The result can be measured by testing fitness and monitoring the training [14].

Pre-Participation Screening

A systematic review/meta-analysis from Harmon et al. [7] reported a sensitivity and specificity for the detection of

serious cardiac abnormalities to be 20% and 94%, respectively, for the history and 9% and 97%, respectively, for the physical examination. This seemingly reflects a high variety of initial positive responses (24–68%) reportable by high school and school athletes on questionnaires.

Despite uncertainty on the risk factors, there is universal agreement from the American Heart Association (AHA), international olympic committee, European Society of Cardiology, and Federation Internationale de Futbol Association that cardiovascular screening in athletes should be undertaken [15]. The goal of cardiovascular screening is to identify athletes with intrinsic cardiac disorders at risk for SCD and to maximize their health and safety on the playing field. ECG improves the ability to detect many of the diseases associated with SCD. The ECG has enhanced sensitivity to detect ion channelopathies as well as primary electrical diseases.

The ECG exhibits superior sensitivity compared to history and physical examination to detect conditions predisposing to SCA/D. However, the identification of disease does not necessarily lead to a significant reduction in SCA/D. The "Canadian Heart Rhythm Society/Canadian Heart Rhythm Society Joint Position Statement on the Cardiovascular Screening of Competitive Athletes" recommended against the routine performance of an ECG for the initial cardiovascular screening of competitive athletes [16].

As strongly recommended in the "Canadian Cardiovascular Society/Canadian Heart Rhythm Society Joint Position Statement on the Cardiovascular Screening of Competitive Athletes," AED access and emergency action plans are the foundation of ensuring the enhanced safety of our athletes [17].

On-Field Management

A study from Weisfeldt et al. [18] demonstrated that prompt application of an AED is associated with a greater likelihood of survival (odds ratio: 1.75; 95% confidence interval: 1.23 to 2.50; p<0.002). Highest survival to hospital discharge noted in places of recreation. For instance, very recently 30-year-old Denmark Footballer Christian Eriksen collapsed on-field during Euro 2020 Group B fixture due to sudden cardiac arrest, he was administered CPR and application of AED on-field and shifted to hospital care. He is the first top-flight player fitted with an implantable cardioverter defibrillator (ICD), which can reset the heart following an electrical abnormality. Such lifesaving incidents paves way for more extra vigilant measures in on field care.

Post-COVID and SCD

In this era of COVID-19, where the virus has proven to be an important cause of SCD during exercise due to myocarditis has increased the concern for athlete's cardiac health. Emerging observational data coupled with widely publicized reports of athletes in competitive sports with reported COVID-19-associated cardiac pathology suggest that myocardial injury may occur in cases of COVID-19 that are asymptomatic and of mild severity. In the absence of definitive data, there is ongoing uncertainty about the optimal approach to cardiovascular risk stratification of athletes in competitive sports following COVID-19 infection [19]. Following Sports eligibility myocarditis recommendations have been made by a scientific statement from the AHA and the American College of Cardiology [20]: 1. Before returning to sports, athletes diagnosed with a clinical syndrome consistent with myocarditis should undergo a resting echocardiogram, 24-h Holter monitoring, and an exercise 12-lead electrocardiogram no <3 to 6 months after the illness (Class I; level of evidence C), 2. It is reasonable that athletes can

resume training and/or competition if all of the following criteria are met (Class IIa; level of evidence C): A. Ventricular systolic function has normalized. B. Serum markers of myocardial injury, heart failure, and inflammation have returned to normal levels. C. Clinically relevant arrhythmias on Holter monitor and graded exercise 12-lead ECG are absent.

Preventive Strategies-Recommendations

The best strategy is to combine synergistically primary prevention of SCD by pre-participation identification of athletes affected by at-risk cardiomyopathies and secondary prevention with backup defibrillation of unpredictable sudden cardiac arrest on the athletic field [21].

As with all things in American medicine, there are legal ramifications in the cardiac screening debate. Lawsuits might arise if an athlete is prohibited from participating in sports. Lawsuits might also arise if an athlete is cleared for sports participation and then dies of SCD. Once an athlete is restricted from play and follows the advice of their physician, it does not necessarily eliminate the risk of sudden death. SCD is more common during physical exertion but can occur at any time. Furthermore, restriction from physical activity may itself be harmful. Exercise is good for cardiovascular health and emotional well-being [22].

ICD

The ICD Sports Safety Registry is a multinational, prospective, observational registry. It was established to identify and quantify the risks associated with sports participation for ICD patients. The ICD sports registry prospectively follows athletes who continue sports participation after receiving an ICD. Competitive sports participation for patients with an ICD no longer carries a blanket restriction, but rather, recommendations need to be

considered. Prevention of inappropriate shock is also critical for the athlete, in whom sinus rates may be above standard rate cutoffs. Types of ICDs are transvenous and subcutaneous. On the other hand, in contact sports, the position of the generator may drive the decision. For the transvenous device, the generator is placed prepectoral, protected by standard padding for contact sports such as American Football or ice hockey, while the S-ICD generator is placed lower and laterally, over the lower ribs, and maybe less protected. What device is best-suited to the athlete remains an important avenue of future research [23].

Young athletes with ICDs may experience shocks during sports and at other times, but no serious adverse outcomes occurred. The decision on return-to-sport participation after an ICD should be individualized and discussed between physician, athlete, and parents [24].

Recommendations focusing on primary and secondary prevention strategies to reduce the incidence of SCD in athletes can be made based on the existing evidence available. Table 1 lists these preventive strategies which aid in cardiac risk mitigation.

Conclusion

Sudden cardiac arrest is the leading cause of preventable deaths in both professional and recreational athletes. Prompt use of AED and implantation of ICD has shown promising benefits to the athletes involved in competitive sports. Hence, knowledge of primary and secondary prevention is of great importance in reducing the incidence of SCD, improvising existing strategies, and warrants future research in the field of sports cardiology.

Declaration of patient consent: The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the Journal. The patient understands that his name and initials will not be published, and due efforts will be made to conceal his identity, but anonymity cannot be guaranteed.

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