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Sudden Cardiac Death in Athletes: Magnitude, Causes, and Prevention Strategies - A Literature Review

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ABSTRACT: Sudden cardiac death (SCD) in athletes, though rare, is the leading cause of exercise-related mortality and poses significant concern due to the symbolic health and vitality of athletes. This review examines the epidemiology, causes, and prevention strategies of SCD in athletes. SCD, defined as sudden death presumed to be of cardiac origin within one hour of symptom onset, affects athletes variably depending on age, race, and type of sports. Key causes include hypertrophic cardiomyopathy (HCM) and arrhythmogenic right ventricular cardiomyopathy (ARVC) in younger athletes, and coronary artery disease (CAD) in older athletes. Ion channelopathies and structural heart diseases also contribute significantly. Preparticipation screening (PPE)

involving ECGs, lifestyle adjustments, and medical interventions are critical in prevention. Continuous research and adaptive guidelines are essential for safeguarding athletes against these fatal events.
Keywords: sudden cardiac death, athletes, sports, exercise, cardiovascular diseases, hypertrophic cardiomyopathy, arrhythmogenic right ventricular cardiomyopathy, coronary artery disease, ion channelopathies, long QT syndrome, Brugada syndrome, preparticipation screening, electrocardiogram (ECG), risk factors, prevention strategies.

INTRODUCTION

Sudden cardiac death (SCD) is always a dramatic event despite circumstances, especially if it happens to athletes who epitomise healthy lifestyle and strength. Moreover, it reaches the journalistic spotlight at once regarding celebrities in sports or representatives of the most popular competitive sports. Despite the rareness of SCD in the general population, it is the leading cause of sports and exercise-related mortality in athletes. [1, 14, 16]. It is also an issue that attracts the academic community's attention. It therefore demands further research to fill the gaps and to find the approach to decrease the incidence of SCD in future.

DEFINITIONS AND EPIDEMIOLOGY

SCD due to European Society of Cardiology (ESC) Guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death (2022) is defined as sudden natural death presumed to be of cardiac cause that occurs within 1 hour of onset of symptoms in witnessed cases, and within 24 hours of last being seen alive when it is unwitnessed; SCD in autopsied cases is defined as the natural unexpected death of unknown or cardiac cause. The problem we face while reviewing the literature is that sometimes there is no distinguishing between SCD and **sudden cardiac arrest (SCA)**, which in turn due to the mentioned ESC Guidelines is defined as sudden cessation of normal cardiac activity with haemodynamic collapse. So, we may have some difficulties with delivering statistics, as we could not verify if SCD survivors were included. Nevertheless, plenty of research has brought up the topic and should not be omitted from our attention. On the other hand, there is no certainty with the definition of **athlete**. Following ESC, the athletes are “defined as individuals of young and adult age, either amateur or professional, who are engaged in exercise training regularly and take part in official sports competition. Official sports competition (local, regional, national, or international) is defined as an organised team or individual sports event that, placing a high premium on athletic excellence and achievement, is organised and scheduled in the agenda of a recognized Athletic Association”.

Despite this definition being unequivocal, we cannot ignore the growing popularity of "recreational" sports with significant intensity. Although exercise is health-promoting for most individuals [2, 3] and reduces the long-term risk of cardiovascular disease [4-6], for a small number of individuals who harbour cardiac conditions or for those who practice strenuous exercises, acutely increases the risk of SCA [7-9]. This is the so-called "exercise paradox", which illustrates how exercise can provide long-term protection against cardiovascular events while also acutely predisposing a vulnerable individual to a relatively increased risk of SCA [10]. The episodes of vigorous exercise may activate the sympathetic nervous system and trigger ventricular arrhythmias in the presence of susceptible myocardial substrate [11]. Nowadays, the number of people who do regular physical exercise is continuously growing. Thus, we deal with so-called exercisers, who probably may be evaluated as "athletes".

The last, but not the least point that should be emphasized is that some authors consider that there is a principal difference between the level of motivation between an exerciser, who is focused on health improvement, and an athlete who directs all the efforts to win a competition. In such a way, we may deal with patients who have different cardiovascular risk profiles [12, 13]. These inconsistencies help account for the wide range of estimated incidence of SCD in athletes in prior reports, from 1 in 3,000 up to 1 in 1 million [1].

Another critical issue is the age of the athletes. The incidence of SCD in athletes increases with age [14, 15]. In apparently healthy athletes (35 years), the estimated incidence of SCD ranges from 2 to 6,3 per 100 000 participant-years. In comparison in young competitive athletes (≤35 years), the incidence of fatal events is significantly lower, 0,4–3 per 100 000 participant years [15-17].

It also has been recognized that some populations of athletes may be at substantially higher risk than others for SCD. Among National Collegiate Athletic Association (NCAA) athletes, increased risk has been found with male gender [18-20], Black race [18], basketball [18, 21] and football [22] participation. One of the most recent events which paid a lot of public attention was SCA, which happened to a Danish football player in the middle of a Euro 2020 match [23]. Team staff and physicians immediately administered cardiopulmonary resuscitation (CPR) and used an automated external defibrillator (AED) to obtain the return of spontaneous circulation (ROSC). Such cases remind us that we have still much to discover and even more to improve.

CAUSES NAD RISK FACTORS OF SCD

To answer the question of how to prevent SCD in athletes soon we are obliged to deal with its diverse aetiology. Before analysing specific pathological conditions and diseases which may increase the risk of SCD, we should consider such risk factors as male sex, African American ethnicity and vigorous exercise, especially if it comes to starters. [24, 25].

The causes of SCD can be classified in diverse ways. The first approach that we offer is analysing the causes by age. First, the roots of pathological conditions in athletes of different age categories (younger and older than 35 years old) may differ significantly. We must also take into consideration that as the population ages and the popularity of endurance sports increases, the burden of SCD risk in an elderly group is likely to grow. There is also a way to subdivide the cause of SCD into congenital and acquired ones, thus we would be able to distinguish what methods of prevention are the most appropriate. The last but not the least approach of classification is to categorise by the heart's morphology, meaning causes with structural abnormal heart (cardiomyopathies (CMP), aortopathy, valvular heart disease, coronary artery disease (CAD), myocarditis) and structural normal heart (Wolf-Parkinson-White (WPW) syndrome or other accessory pathways, long QT syndrome (LQTS), Brugada syndrome or other ion channelopathies, commotio cordis) [27]. SCD in young athletes is usually caused by a genetic or congenital structural cardiac disorder [17, 18, 20, 26, 27, 28]. However, more recent analyses have figured out that a predominant cause of sudden death (SD) in athletes is **autopsy-negative sudden unexplained death (AN-SUD)**, which is defined as a SD with non-diagnostic autopsy findings [17, 29-31, 32-34]. Some studies have estimated the incidence of ANSUD may be as high as 31%. [18, 34]. More recent data on NCAA athletes suggests that the most common rank list of causes of SCD may merit re-evaluation [18].

Historically, **hypertrophic cardiomyopathy (HCM)** was thought to be the most common underlying condition that led to SCA in athletes [26, 35, 36]. HCM is the most common cause of SCD in young adults, although the risk of sudden death persists through midlife and beyond [37]. According to the 2023 ESC Guidelines for the management of CMP, HCM is defined as the presence of increased left ventricle (LV) wall thickness (with or without right ventricle (RV) hypertrophy) or mass that is not solely explained by abnormal loading conditions [38]. A foundational study from 1996, with data revision from 2009, found that nearly 36% of SCD in athletes was a result of HCM [26]. We should pay attention, that the frequency of HCM may be vastly different, depending on countries and regions. HCM

was responsible for 36% of SCD cases in the U.S. National Registry of Sudden Death in Athletes [26], but a prospective study of SCD among children and young adults conducted in Australia and New Zealand from 2010 through 2012 attributed only 6 of 54 cases (11%) of HCM during or post-exercise, whereas 20 were unexplained (37%) [39]. Alpert et al. [40] condensed several published studies to generate an estimated risk of SCD for athletes due to HCM at 0,03% to 0,1%, which is comparable to contemporary published mortality rates in the overall HCM population [41]. In the UK HCM was diagnosed in only 6% of SCD cases; however, idiopathic left ventricular hypertrophy was present in another 16%, raising the question of whether it represents an initial form of HCM [42]. In an autopsy study of 118 athletes from the UK who had SCD, the second identified cause of SCD (14%) was arrhythmogenic right ventricular cardiomyopathy (ARVC) [43]. The reports from other countries such as Italy [44] and Denmark [45] have found that the most common cause of SCD in young athletes is arrhythmogenic right ventricular cardiomyopathy (ARVC), responsible for around 25% of SCD cases in athletes from those countries.

According to the 2023 ESC Guidelines for the Management of CMP, **arrhythmogenic right ventricular cardiomyopathy (ARVC)** is defined as the presence of predominantly RV dilatation and/or dysfunction in the presence of histological involvement and/or electrocardiographic abnormalities following published criteria [46]. ARVC is an inherited disease characterized by progressive myocyte loss with fibrofatty tissue replacement, which happens most often in the right ventricle (RV) but can affect the LV or both. ARVC is characterized by variable disease expression that can increase the risk of SCD, notably during exertion [46]. Exercise appears to be an independent risk factor for expediting the expression of the disease phenotype and triggering fatal tachyarrhythmias. Consequently, according to the 2022 ESC Guidelines for the Management of Patients with Ventricular Arrhythmias (VA) and the prevention of SCD avoidance of high-intensity exercise is recommended in patients with a definite diagnosis of ARVC (class I, level B) [17, 20, 47] and may be considered in carriers of ARVC-related pathogenic mutations and no phenotype (class IIb, level C) [47, 48]. At the same time, participation in high-intensity exercise may be considered for asymptomatic adult HCM patients without risk markers [49].

Dilated cardiomyopathy (DCM) is another type of CMP which can be a potential cause of SCD in athletes. Simultaneously, we deal with the issue of the “athlete’s heart”. A small but considerable proportion of endurance athletes will have dilated LV cavities with LV function in the interior limit, which overlaps with the findings of a DCM [50]. These

physiologic changes in the LV may be also accompanied by RV dilatation and reduced systolic function, which could raise concern for ARVC in the appropriate context. In such cases, the expert team evaluation is crucial. Turning back to the 2022 ESC Guidelines for the management of patients with ventricular arrhythmias (VA) and the prevention of SCD, participation in high-intensity exercise including competitive sports is not recommended for individuals with DCM/hypokinetic non-dilated cardiomyopathy (HNDCM) and a lamin A/C (LMNA) mutation [51].

Considering the importance of the issue of CMP as one of the leading causes of SCD in athletes, however, we cannot omit other specific conditions. Continuing the topic of AN-SUD, we must emphasize that it is suspected that a large portion of the cases are attributable to cardiac **ion channelopathies** including long QT syndrome (LQTS), Brugada syndrome (BrS) and catecholaminergic polymorphic ventricular tachycardia (VT) [52].

Following the 2022 ESC Guidelines for the management of patients with ventricular arrhythmias (VA) and the prevention of SCD, **long QT syndrome (LQTS)** is characterized by a prolonged QT interval and VA mainly triggered by adrenergic activation. The mean age at presentation is 14 years. The annual rate of SCD in asymptomatic patients with untreated LQTS has been estimated to be less than 0,5% [53] while it increases to around 5% in those with a history of syncope [54]. The diagnosis is clinical, and one of the most widespread ways to conduct it is based on a validation score. LQTS is estimated to affect one in every 2000 individuals, and if left untreated, the SCD rate would be between 0,33–0,9% per year. By now, more than 600 types of gene mutations are known, giving names to 17 different autosomal dominant LQTSs. Despite being numerous and clinically heterogeneous, LQTS-1, LQTS-2, and LQTS-3 are responsible for more than 90% of cases, with LQTS-1 being the most frequent [12]. The triggering events are different according to the type of LQTS. Exercise, especially swimming, was the trigger in 62% of cardiac events in LQTS-1, 13% in LQTS-2, and 13% in LQTS-3. Moreover, in LQTS-1, exercise was the trigger in 68% of arrhythmic events. On the other hand, for LQTS-2, loud and sudden auditory stimuli were the main trigger; and in LQTS-3, vagal situations such as sleep. Therefore, referring to ESC Guidelines, avoiding genotype-specific triggers for arrhythmias is recommended (class I, level C) [55].

Another cause of SCD, which can be attributed to AN-SUD cases is another genetically conditioned one such as **WPW syndrome**, while autopsy examination cannot reliably identify the existence of accessory pathways. High-intensity physical activity

increases the risk of WPW-related SCD, as observed in a study where two-thirds of the population suffered from an aborted ventricular fibrillation (VF) cardiac arrest during exercise or under emotional stress [56]. In patients with WPW syndrome, the most common arrhythmia is atrioventricular re-entry tachycardia (AVRT; 80%), followed by AF (20–30%). SCD secondary to pre-excited AF resulting in VF is the most feared manifestation of WPW syndrome. The risk of CA/VF in untreated WPW patients has been estimated at 0.9–2.4 per 1000 person-year [57, 58]. The management of WPW patients has recently been updated with a particular focus on athletes in 2020 [59]. Due to the 2022 ESC Guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death, in patients with ventricular pre-excitation and symptomatic AVRT, catheter ablation is recommended (class I). In asymptomatic patients with ventricular pre-excitation, both invasive (class IIa) and non-invasive (class IIb) assessments are options for risk stratification for SCD [60]. Following the 2020 ESC Guidelines on sports cardiology and exercise in patients with cardiovascular disease, for athletes who refuse ablation, or if the procedure is associated with high risk, such as an anteroseptal accessory pathway, participation in competitive sports activities can be discussed on an individual case by case basis including the use of pharmacological therapy, although there are currently no data about its efficacy; sports in which the potential loss of consciousness could be fatal should be discouraged.

The next issue that also cannot be ignored is **myocarditis**. Myocarditis is a non-ischaemic inflammatory disease of the myocardium, which may cause cardiac dysfunction and arrhythmias [61, 62]. The aetiology, as well as clinical presentation of myocarditis, are heterogeneous, but viral infection is the most common cause in the developed world [63, 64]. In the context of young individuals, toxins such as cocaine and amphetamine-based supplements should also be evaluated in the clinical history [61]. Case series have proved myocarditis as a risk factor for SCD, which accounts for up to 2-20% of sudden death in athletes [26, 42, 60, 61, 65, 66]. Athletic individuals with a probable or definitive diagnosis of recent myocarditis should be recommended to abstain from competitive sports or leisure sports while active inflammation is present, regardless of age, sex, or extent of LV systolic dysfunction [67, 68].

Referring again to the age of the athletes, mentioned above, in athletes >35 years of age, more than 80% of all SCD is due to **atherosclerotic CAD**, and vigorous physical exertion is associated with an increased risk of AMI and SCD [69, 70-74]. Nevertheless, the benefits of regular exercise greatly outweigh the exercise-related (Ex-R) risk, even in

individuals with chronic coronary syndrome (CCS) [75, 76]. Individuals at risk of CAD and asymptomatic individuals in whom CAD is detected at screening should have aggressive management of risk factors for atherosclerosis [77-80]. Considering the benefits of exercise on primary and secondary prevention of CCS [77, 81] individuals with risk factors should be restricted from competitive sport only when there is a substantial risk of an adverse event, as indicated by functional tests, or when there is evidence of disease progression during serial evaluations [82].

Continuing the topic of coronary arteries, there is another potential cause of SCD which is the **anomalous origin of coronary arteries (AOCA).** It is a common cause of SCD in young athletes [26, 60, 83, 84], but is rarely implicated in individuals >40 years of age [85, 86]. The proposed mechanism of SCD is ischemia-triggered ventricular arrhythmia once exercise tends to induce expansion of the aortic root and pulmonary trunk, and both may compress the passing coronary artery [87]. Due to the mentioned 2020 ESC Guidelines on sports cardiology and exercise in patients with cardiovascular disease, in asymptomatic individuals with an AOCA that does not course between the large vessels, does not have a slitlike orifice with reduced lumen and/or intramural course, competition may be considered, after adequate counselling on the risks, provided there is absence of inducible ischaemia (class IIb, level C). In the case of patients after surgical repair of an AOCA, participation in all sports may be considered, at the earliest 3 months after surgery, if they are asymptomatic and there is no evidence of inducible myocardial ischaemia or complex cardiac arrhythmias during maximal exercise stress test (class IIb, level C). On the contrary, participation in most competitive sports with a moderate and high cardiovascular demand among individuals with AOCA with an acutely angled take-off or an anomalous course between the large vessels is not recommended (class III, level C).

The last but not the least issue we would like to deliver is commotio cordis (CC) which is an arrhythmic cause of SD of traumatic origin caused by a blunt chest trauma leading to VF. It needs to be differentiated from cardiac contusion (contusio cordis), in which the trauma causes structural cardiac damage, such as observed in motor vehicle accidents. Young males are affected the most because of the overwhelming predominance of males in the sports in which CC occurs. Among the sports, baseball along with fistfights are the ones with the highest risk for CC [88]. For instance, nearly all CC events are caused by direct baseball strikes to the anterior chest. In other sports, it can also occur due to impacts with elbows, fists, and helmets. In a recently published registry that gathered SCD data from football matches

(2014-2018) 7 of 617 SCDs reported, were confirmed to be due to commotio cordis, and another 7 were very likely to be as well [89].

Fortunately, most of the mentioned specific conditions nowadays are treatable if diagnosed. If there is still much that must be done with the management of CMP, contemporary invasive cardiology can deal with CAD, and recent achievements of electrophysiology allow the successful treatment of ion channelopathies or ventricular pre-excitation. A much more vulnerable issue is the prompt detection of the diseases which can be a potential cause of SCD. Even more difficult is to make the screening and diagnostics process harmless for the athletes and the whole sports community.

SCREENING AND EVALUATION

Since many of the cardiac conditions that cause SCD in athletes may not present warning symptoms, there has been considerable discussion about the role of preparticipation screening tests to evaluate occult cardiovascular disease. The value of any screening test is determined by the characteristics of the population to which it is applied. Accordingly, there are major differences in contemporary guidelines for the preparation screening of athletes. The **pre-participation evaluation (PPE)** before competing in athletics has been recommended by multiple societies [90–95] to identify or raise the suspicion of cardiovascular abnormalities that could potentially result in SCD on the athletic field [90]. Moreover, the proper pre-participation cardiovascular evaluation offers the potential to identify athletes at risk for cardiovascular disease before the onset of symptoms [75, 96, 97-99] and, following the 2022 ESC Guidelines for the management of SCD, should be considered in competitive athletes (class IIa, level C) [16, 75, 96, 100].

The American Heart Association (AHA) and American College of Cardiology (ACC) recommend screening that is limited to a targeted **medical history and physical exam** - a 14-point questionnaire aimed at identifying individuals at the highest risk. It includes questions, about a personal history of cardiovascular symptoms (i.e., chest pain, syncope, dyspnoea) and a family history of premature sudden death or disability from heart disease in addition to a focused physical exam [91]. However, recent studies have pointed to the limited sensitivity and specificity of this questionnaire [93].

In contrast to the AHA, ESC and the International Olympic Committee (IOC) advocate for screening that also includes a resting 12-lead electrocardiogram (**ECG**) [101]. This approach was mandated in Italy and Israel. While a landmark study in Italy succeeded, in which the rate of SCD declined by nearly 90% after the addition of the ECG to the

preparticipation examination [96, 100], a similar study conducted in Israel found no significant difference in the rate of SCD following the implementation of mandatory ECG screening [102]. The role of the ECG in preparticipation screening has garnered considerable debate [103, 104]. Although ECG, as one of the essential parts of PPE, appears effective in identifying cardiovascular disease, especially in young athletes (≤35 years of age) by identification of relevant symptoms (e.g. exertional syncope) or abnormalities consistent with inheritable CMP or channelopathies [100, 105-107], it is criticised significantly because of the high number of false positive tests. While true positive cases are important to identify, false positive cases result in individuals undergoing additional testing, which may lead to undue cost, unnecessary sports restriction, and anxiety. In the Italian study, mentioned above, 9% of all athletes were referred for additional evaluation despite only 2% being determined to have an underlying condition that could predispose them to SCD [100]. These false positives are largely due to the physiologic adaptations of an athlete's heart that manifest as otherwise abnormal ECG tracings in the general population. Observational studies have been performed to figure out the unique ECG findings specific to athletes [91, 108-114] and improved characterisation of the athlete ECG has led to the development of standardised ECG interpretation criteria, including now widely accepted international criteria published in 2017 [91,115,116]. This consensus statement added the borderline ECG concept and provided a more thorough evaluation [117].

In cases of positive findings at any of the first-line examinations, the athlete should be evaluated with **other invasive and non-invasive tests** such as Holter, echocardiogram, exercise stress test, CMR, coronary computed tomography (CCT), invasive angiography, electrophysiological study with electroanatomic mapping, and endomyocardial biopsy (EMB) [14,77,118-121].

Exercise testing should be adapted to the specific type of sport responsible for the arrhythmic events [122]. Exercise testing is reasonable, even in the absence of symptoms, for the evaluation of persons at high risk of CAD, especially elder athletes who plan to start a vigorous exercise program [123, 124].

Prolonged ECG monitoring with a **Holter device** can be useful in patients with frequent or reproducible symptoms, such as palpitation or syncope. Athletes with intermittent, infrequent symptoms are best evaluated with an implantable loop recorder [122].

The first choice as initial morphological evaluation in those athletes who are suspected of having an underlying cardiogenic substrate is usually **transthoracic echocardiography**

(TTE) [125]. Although TTE can increase the sensitivity of screening for structural heart disease (SHD), it is unfeasible as a routine test in mass screening [106, 108]. TTE is typically normal in catecholaminergic VT, AOCA, BrS, and LQTS, but TTE findings vary according to the CMP [52].

Cardiac magnetic resonance (CMR) has emerged as an important non-invasive, non-radiating imaging technique, particularly suited to provide detailed cardiac tissue characterisation in athletes in the last decade [126]. CMR overcomes the difficulty of the TTE to obtain images in patients with a poor acoustic window. It has also proved to be important in identifying the LV hypertrophy not seen on TTE. Myocarditis as well can be suggested by CMR. A recent study from Andreini et al. sought to figure out whether CMR could identify structural heart disease (SHD) in patients with ventricular arrhythmia and normal TTE. CMR detected SHD in 241 patients (25.5%) and abnormal findings not specific for a definite SHD diagnosis in 187 patients (19.7%) [127].

The new imaging technologies including **electrophysiological study (EPS) and electroanatomical mapping** are promising tools to better characterize the arrhythmic substrate. They could be useful in those athletes who are suspected of having an underlying myocardial substrate for arrhythmias due to abnormal second-level examinations [128]. Such diagnostic opportunities provide the following proper treatment. For instance, in patients with ventricular pre-excitation and symptomatic AVRT or even with asymptomatic accessory pathways with high-risk features, **catheter ablation** is recommended [59]. In patients who will undergo such treatment, continuation of sports activity could be allowed [129]. Moreover, catheter ablation is not the only intervention, after which competitive activity is possible. We should not forget about athletes with **implanted cardioverter-defibrillator (ICD)**. Even participation in sports with higher peak static and dynamic components in such athletes may be considered if the athlete is free from ventricular arrhythmias requiring device therapy for more than 3 months [13].

We should also remember **endomyocardial biopsy (EMB),** which may be needed in cases of specific myocardial disorders that are seldom diagnosed by non-invasive testing and have unique prognoses and treatment [130].

Referring to mentioned above AN-SUD, we still have enough gaps that should be settled. As noted above, lots of specific disorders are likely to have genetic backgrounds [52] and could require a genetic autopsy for detection. A prospective study of nearly 500 sudden cardiac death cases in children and young adults found that 27% of AN-SUD cases had a

clinically relevant cardiac gene mutation when the genetic autopsy was performed. Moreover, an inherited cardiac disorder was identified in 13% of the families in which AN-SUD occurred. These results highlight the need for the consideration of the addition of **genetic testing** to autopsy investigation to gain a full understanding of the aetiologies resulting in SCD [39].

Besides diagnostic tools, which can be an essential part of the screening and evaluation of athletes, the prominent issue is proper **statistics**. For instance, there is a need for databases in different populations to report catastrophic events. A good example is the Federation International de Football Association (FIFA), which has attempted to fill this gap through the creation of the FIFA Sudden Death Registry (FIFA-SDR). Starting in 2014, the FIFA-SDR allows for any involved individuals (athletes, coaches, relatives) to report a soccer-related SCA or SCD event and provide additional information regarding the circumstances. FIFA also employs a systematic media monitoring process to capture any events that their self-reporting system may have missed. Between 2014 and 2018, a total of 617 players from 67 countries suffered from SCA, with only 142 (23%) surviving. Registries such as the FIFA-SDR are important tools for understanding the incidence, risk factors, and potential interventions for SCA in athletes. Unfortunately, this only includes cases from "association" football [89].

The last but not the least tool, which could be used for the proper evaluation is better **epidemiology,** which will help to identify those at the highest risk. The crucial thing is the responsibility for tough decisions, which is taken by healthcare specialists. It is also should be noticed that there is a lack of studies, particularly if it comes to asymptomatic athletes, which complicates to find the solution to eligibility for sports activity. Given these levels of uncertainty, participation in competitive athletics with a cardiovascular disorder should be a **shared decision-making process** [131] between the physicians, the athlete, the family, and the governing body.

If it comes to SCD prevention, the essential part is the emergency action plan (EAP), which the Interassociation Task Force, AHA, American College of Cardiology (ACC), and FIFA strongly encourage sports organisations to set up for the effective management of SCA events. EAP should include training for personnel on how to respond to a collapsed athlete, easily access an automated external defibrillator (AED), call for emergency medical services, and transport to the nearest advanced care facility [132-135]. CPR and AED use has shown a significant reduce of mortality from SCA when executed promptly. As a result, ensuring

athletes and respective staff at all levels have an effective EAP implemented and consistently rehearsed is imperative to improving outcomes from SCA. Educating athletes on what qualifies for initiating CPR could also eliminate delays during initiation.

CONCLUSION

SCD in athletes is a relatively rare event, but as sports practice has become more popular in recent years, the issue is more than relevant. Considering all the above, we have plenty of tools, which can be convenient for SCD prevention in athletes, but we still have a lot to research how to use them. We should begin with the proper analysis of statistics by creating flexible databases and continue with adding ECG to routine PPE and second-line examinations mentioned above if they are considered necessary after the shared decision of sports cardiologists. Any screening strategies should be adapted to the local demand. The effective implementation of EAP could also play an essential role in SCD prevention. Randomised trials need to be performed in the near future to prove different management strategies following clinical implementation.

Author's contribution

Conceptualization, BM and RJ; methodology, BM and AR; software, BM and ML; check, BM, ML and EL; formal analysis, BM and MM; investigation, BM and AR; resources, BM and RJ, data curation, BM and ML; writing - rough preparation, BM; writing - review and editing, BM and EL; supervision, BM; project administration, BM. All authors have read and agreed with the published version of the manuscript.

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