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REVIEW PAPER

Sudden cardiac arrest and sudden cardiac death in athletes: an updated perspective

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Abstract

Sudden cardiac arrest (SCA) and sudden cardiac death (SCD) in athletes are rare but catastrophic events that generate substantial medical, ethical, and public health concern. Although regular physical activity is associated with significant cardiovascular benefits, intense training and competition may unmask underlying structural or electrical heart diseases in susceptible individuals. The most common causes of SCA and SCD in young athletes include hypertrophic cardiomyopathy, arrhythmogenic cardiomyopathy, left ventricular hypertrophy, myocarditis, congenital coronary artery anomalies, and primary electrical disorders such as long QT syndrome. In athletes over 35 years of age, asymptomatic atherosclerotic cardiovascular disease predominates. Pre-participation cardiovascular screening, including medical history, physical examination, and, in some regions, electrocardiography (ECG) aims to identify individuals at risk. However, controversy persists regarding the sensitivity, specificity, cost-effectiveness, and potential for false-positive findings associated with screening strategies. Rapid recognition of SCA, immediate cardiopulmonary resuscitation, and early defibrillation with automated external defibrillators (AEDs) remain critical determinants of survival. The aim of this review is to summarize current evidence on the epidemiology, risk stratification, screening strategies, and emergency management of SCA and SCD in athletes and to highlight ongoing challenges and areas for future research.

Keywords: pre-participation examination, commotio cordis, heart arrest, sudden cardiac death, athletes

Introduction

Physical activity and sport participation represent an important component of modern lifestyles and are widely promoted for their health benefits, reinforcing the role of sport as a socially embedded aspect of everyday life. Regular exercise is widely promoted as a cornerstone of cardiovascular prevention [1] and overall well-being, and athletes – both professional and amateur – are often perceived as exemplars of health, vitality, and resilience. This perception is reinforced by public health initiatives that emphasize the protective effects of habitual physical activity across all age groups [2]. Sudden cardiac death (SCD) and sudden cardiac arrest (SCA) represent significant clinical and public health concerns, affecting not only professional athletes but also a large population of recreational sports participants, including adolescents and young adults. Among people who practice sports, the risk of sports-related SCA or sports-related SCD is small, but it is a dramatic event that may be avoided [3]. The issue has significant social and public health relevance and therefore attracts widespread attention. Long-term exercise has a protective effect and reduces the risk of many diseases, including stroke and heart attack, but during intensive exercise and in the short term after physical exercise, the risk of SCA increases [4]. Although survival rates following sports-related SCA have improved in recent years [5,6], it remains a significant public health concern and a continuing challenge for prevention and emergency response systems.

SCD is defined as an unexpected death resulting from cardiac arrest, typically occurring within minutes and generally within 1 hour of symptom onset. Sports-related SCD refers to events occurring during or shortly after physical activity and affects both professional and recreational athletes. SCD represents the leading cause of sports-related mortality in athletes [1]. The most important risk factors for sports-related SCD include: primary cardiovascular disease, intense physical exercise or competitive sports, as well as other factors related to sports competition, such as dehydration and electrolyte imbalance [7].

SCA refers to a sudden loss of cardiac function resulting in collapse due to a presumed cardiac cause, requiring cardiopulmonary resuscitation (CPR) or defibrillation. It represents a life-threatening condition with a time-dependent prognosis. In young athletes, SCA constitutes the most common cause of mortality [1,8].

This review aims to summarize current evidence regarding epidemiology, risk factors, screening strategies, and prevention of SCA and SCD in athletes, highlighting current challenges in screening and emergency response.

Aim of the work

The aim of this review is to summarize current evidence on SCA and SCD in athletes, focusing on epidemiology, underlying causes, risk stratification, pre-participation screening, and prevention strategies. The review seeks to clarify existing controversies regarding screening and emergency preparedness and to identify gaps in knowledge that may guide future research and improve cardiovascular safety in athletes.

Methods

A narrative review with a structured literature search was conducted to summarize current evidence on sudden SCA and SCD in athletes. The methodology was designed to enhance transparency and reproducibility, taking into account key principles of the PRISMA guidelines, although a formal systematic review protocol was not registered.

Electronic databases, including PubMed, Scopus, and Web of Science, were systematically searched for articles published between January 2000 and February 2026. In addition, reference lists of relevant articles and position statements were manually screened to identify further eligible studies.

The search strategy included combinations of the following keywords: “sudden cardiac arrest”, “sudden cardiac death”, “athletes”, “sports”, “pre-participation screening”, “electrocardiogram”, and “commotio cordis”. Only articles published in English were included.

All the records identified through database searching (n=3305) were imported, and duplicates were removed (n=1128), leaving (n=2177) studies for screening. Titles and abstracts were screened for relevance, and studies not related to athletic populations or SCA/SCD were excluded (n=1724). The full texts of the remaining articles (n=453) were assessed for eligibility.

Studies were included if they addressed epidemiology, etiology, screening, risk stratification, prevention, or emergency management of SCA/SCD in athletes. Eligible study types included original research articles, systematic reviews, meta-analyses, case reports, and official guidelines. Conference abstracts without full text and studies not involving athletic populations were excluded. A total of (n=50) studies were ultimately included in the review.

Given the narrative nature of the review, no formal risk of bias assessment was performed. However, priority was given to high-quality evidence, including cohort studies, registry data, and international guidelines.

Although the search covered publications from 2000 onwards, particular emphasis was placed on studies published within the last three years to reflect the most recent evidence and contemporary clinical recommendations.

The main limitations of this review include its narrative design, the lack of formal quality assessment, and restriction to English-language publications, which may introduce selection bias.

Literature review results

Epidemiology of sports-related SCA and SCD

There is a significant range in the prevalence estimates of sports-related SCA and sports-related SCD, making it difficult to clearly define the epidemiology. These discrepancies stem primarily from the lack of a unified system for reporting sports-related SCA and SCD cases and the lack of mandatory reporting of SCD cases [9,10]. Estimates [1] indicate that the incidence of SCD in competitive athletes ranges from nearly 1 in 1 million to 1 in 5,000 athletes per year. FIFA records 2014-2018 [9] indicate an incidence of SCD of 6.8/100,000 adolescent high-level football players per year. Other results [11] estimate the prevalence of SCA and SCD among young athletes to be between 1 in 2,400 person-years and 1 in 417,000 person-years. This marked heterogeneity is unlikely to reflect true differences in disease occurrence alone but rather results from significant methodological variability between studies. Key contributing factors include inconsistent definitions of SCA/SCD, differences in case ascertainment methods (e.g. registry-based vs. media-based data), underreporting due to the lack of mandatory reporting systems, and variability in the populations studied (competitive vs. noncompetitive athletes) [9,10]. Overall, while there is general agreement that sports-related SCA/SCD is a rare event, the true incidence remains uncertain [12,13]. Notably, data on noncompetitive athletes are particularly limited, further contributing to the lack of reliable epidemiological estimates in this population.

Contrary to widespread media narratives about an increase in the number of SCA and SCD cases during the COVID-19 pandemic (related to SARS CoV-2 infection or vaccines against this virus), studies [10,12,14] have not shown such an association.

Survival outcomes following sports-related SCA are generally more favorable compared to non-sports-related cases [15], although the magnitude of this difference varies across studies. This is likely explained by contextual factors rather than intrinsic differences in the underlying pathology. In particular, SCAs occurring during sports are more likely to be witnessed, to occur in public settings, and to involve individuals with a higher baseline level of physical fitness, all of which contribute to improved survival [9].

The higher survival rates observed in sports facilities (e.g. 22.8% vs. 8% outside such facilities) are primarily attributed to earlier recognition, more rapid initiation of CPR, and greater availability of automated external defibrillators (AEDs) [16,17].

Despite these favorable outcomes, the risk of SCD is reported to be higher in athletes compared to the general population, particularly among young competitive athletes [18]. However, in absolute terms, the majority of SCA/SCD cases occur in middle-aged individuals engaged in recreational sports, especially after the age of 35 [17,19].

There is also consistent evidence of significant demographic disparities [20]. Sports-related SCA/SCD occurs markedly more often in men than in women [4,6,10,21] and is disproportionately more common in Black athletes, who also exhibit lower survival rates

[4,5,10,22]. These differences likely reflect a combination of biological, environmental, and healthcare access factors, although the exact mechanisms remain incompletely understood.

Finally, the incidence of sports-related SCA/SCD varies across sporting disciplines, with the majority of cases in young athletes occurring in high-intensity sports such as basketball, football, and soccer [4]. This suggests that both the type and intensity of physical activity may modulate risk in susceptible individuals.

Clinical characteristics and triggering mechanisms

Most athletes with SCD do not show prior symptoms indicating heart disease [23], highlighting the key role of healthcare providers in screening young athletes. Intense physical activity, especially in unprepared individuals, may significantly increase the risk of SCD and acute myocardial infarction in susceptible people [4,20]. If someone engaging in physical activity has a hidden or diagnosed cardiovascular disease, intense exercise may trigger a sudden cardiac event, though sport itself is not the direct cause. At the same time, there is no evidence that limiting physical activity reduces SCD risk, and thus eliminating sports is not an appropriate preventive strategy [24]. Sudden, high-intensity exercise (e.g. sprints, long marathons) is more often linked to cardiac events than regular, moderate exercise.

Caffeine and stimulant medications increase sympathetic activity and have been discussed as potential contributors to arrhythmic risk in individuals with an underlying predisposition [25]. However, evidence linking their use to SCD in athletes is limited and inconsistent. The combined effects of stimulant use, caffeine intake, and intense physical exertion remain insufficiently characterized. In athletes with ADHD treated with stimulants, consideration of cardiovascular evaluation may be appropriate based on individual risk profile.

Etiology of SCA and SCD in athletes

Researchers have reported substantial variability in the most common causes of SCA and SCD in athletes, reflecting differences in age, population characteristics, and study methodology. Overall, a clear distinction emerges between younger and older athletes.

In athletes under 35 years of age, SCD is most commonly attributed to inherited or congenital cardiac conditions [1]. Cardiomyopathies – particularly hypertrophic cardiomyopathy (HCM) and arrhythmogenic right ventricular cardiomyopathy (ARVC) [26] – as well as myocarditis and coronary artery anomalies (CAA), are consistently identified as the main etiologies. Supporting this, large-scale screening data from adolescent football players indicate that cardiomyopathies may account for the 88% of SCD cases [27], although many of these conditions remain undetected during pre-participation screening.

However, postmortem studies introduce some heterogeneity into this picture. Importantly, these findings are derived from mixed-age populations rather than exclusively young athletes. In a substantial proportion of cases, no structural abnormalities are identified, suggesting sudden arrhythmic death syndrome (SADS) as a frequent underlying mechanism [23]. Among cases with identifiable myocardial disease, left ventricular hypertrophy (LVH), fibrosis, ARVC, and HCM are among the most commonly reported findings. These discrepancies highlight the limitations of both screening and postmortem diagnostics and suggest that the true distribution of causes may be underestimated or misclassified.

In contrast, in athletes over 35 years of age, there is strong consensus that coronary artery disease (CAD), largely driven by asymptomatic atherosclerotic cardiovascular disease (ASCVD) [28], is the predominant cause of SCD [3,4,19]. This age-dependent shift in etiology is one of the most consistent findings across studies.

Importantly, the risk associated with specific conditions is not uniform. ARVC, in particular, has been identified as a strong independent predictor of SCD during physical activity [23], with affected individuals demonstrating a markedly increased risk compared to those with other cardiac diseases. This underscores the interaction between underlying pathology and exercise as a trigger for malignant arrhythmias.

Finally, emerging evidence suggests that the causes of SCD may also vary by ethnicity [29]. For example, arrhythmogenic cardiomyopathy (ACM) appears to be more prevalent among Black athletes, whereas CAD is more frequently reported in Asian populations. These observations, although not yet fully understood, indicate that genetic and environmental factors may influence disease distribution and highlight the need for more personalized approaches to prevention [29].

Genetic background and molecular diagnostics

It is noteworthy that the findings reported in study by Finocchiaro et al. [30] suggest that, in young healthy athletes, SCD may be more frequently attributable to concealed genetic cardiomyopathies than to classical channelopathies, even in cases where standard autopsy examination reveals no structural cardiac abnormalities. This shifts the diagnostic perspective and highlights the need for broader inclusion of cardiomyopathy genes in molecular autopsy

(MA) panels. Although MA is a promising tool in SCD diagnostics, pathogenic or likely pathogenic variants were identified in 21% of cases, of which 17% were considered clinically actionable or likely causative [30]. Research into the effectiveness of this method and its potential development in the future should continue.

Primary prevention

Classic pre-participation screening of athletes includes medical history and physical examination, while in Europe, it is commonly supplemented with a resting 12-lead ECG [20]. In contrast, American strategies traditionally rely more on history and physical examination alone, reflecting ongoing controversy regarding the effectiveness, cost-effectiveness, and potential harms of ECG-based screening.

Overall, studies provide conflicting evidence on the impact of screening on SCD reduction. Some studies [31,32] suggest that the addition of ECG improves detection of cardiomyopathies, particularly HCM, which may account for a substantial proportion of SCD cases in young athletes. ECG is generally more sensitive than history and physical examination [31] and may identify the majority of HCM cases, supporting its role in enhanced screening protocols.

However, other data have not demonstrated a significant reduction in SCD incidence despite the use of combined screening strategies (history, physical examination, and ECG) [33]. These discrepancies likely reflect differences in study design, population characteristics, duration of follow-up, and definitions of SCD, as well as the relatively low absolute incidence of events. Importantly, ECG is less effective in detecting certain conditions, such as asymptomatic ASCVD, and screening overall remains limited by imperfect sensitivity and specificity [28]. Taken together, ECG screening offers higher sensitivity for detecting certain structural heart diseases, particularly cardiomyopathies, but its overall effectiveness is limited by false-positive results, reduced ability to detect non-structural conditions, and uncertain impact on SCD mortality [4,22].

False-positive results and cost considerations represent additional limitations of ECG-based screening [20]. Although modern interpretation criteria have improved specificity, screening programs may still generate unnecessary follow-up testing and psychological burden, raising concerns about cost-effectiveness, particularly in large populations of low-risk athletes.

Importantly, screening has inherent limitations regardless of the method used. SCD is often the first manifestation of underlying disease [20], and a normal screening result does not exclude future risk [27]. Data show that a substantial proportion of athletes who experienced SCD had previously normal screening results [27], and the time interval between screening and the event may span several years. These findings suggest that single-time screening is insufficient and that longitudinal monitoring may be more relevant in identifying athletes at risk.

Furthermore, the effectiveness of screening is challenged by the fact that a significant proportion of sports-related SCA cases are not caused by structural heart disease [3], limiting the utility of strategies primarily focused on structural abnormalities. This raises important questions about the overall capacity of current screening models to prevent SCD.

Given these limitations, secondary prevention strategies may have a greater impact on survival. Widespread access to AEDs and training in CPR are consistently associated with improved outcomes after SCA and may be more effective at the population level than attempts to identify all at-risk individuals in advance [4,12].

Postmortem examination also plays a critical role in prevention by identifying hereditary causes of SCD and enabling cascade screening in affected families [20,34]. This is crucial for initiating genetic testing and further evaluation of family members, potentially preventing future cases and identifying individuals genetically predisposed to similar events [35,36]. Furthermore, postmortem studies contribute to improving current prevention strategies [37].

Finally, emerging approaches, including advanced data analysis techniques, such as hierarchical clustering (HC) and principal component analysis (PCA), may improve risk stratification by integrating large-scale clinical and physiological data [7]. These methods have the potential to complement traditional screening and support more individualized prevention strategies.

Notably, artificial intelligence (AI) and wearable technologies may increasingly contribute to pre-participation screening by enabling earlier detection of asymptomatic cardiac abnormalities [32]. In addition, these tools have the potential to facilitate continuous remote monitoring through the collection of real-time cardiopulmonary data. Furthermore, the application of machine learning to ECG and cardiac imaging may improve diagnostic accuracy, supporting a more personalized and predictive approach to athlete evaluation.

Resuscitation and early defibrillation

Rapid recognition of SCA, immediate response, high-quality CPR, and early use of an AED are consistently identified as the most effective strategies to reduce SCA mortality [9,20,24,38], often exceeding the impact of pre-participation screening alone. Importantly, early recognition remains a critical limiting step, as symptoms such as seizures or agonal breathing may delay correct diagnosis; therefore, any sudden loss of consciousness with unresponsiveness should be treated as SCA until proven otherwise [38].

Across studies, survival outcomes strongly depend on the timeliness and type of intervention. Prompt AED use is associated with markedly higher survival rates (up to ~85%), whereas CPR alone, although still beneficial, achieves lower survival (~50%) [9]. This difference reflects the high prevalence of shockable rhythms in sports-related SCA, highlighting defibrillation as the key determinant of survival rather than CPR alone.

However, reported survival rates vary substantially between settings, which can be explained by differences in emergency preparedness, AED availability, and bystander training. Environments with structured emergency response systems, such as competitive sports, consistently report better outcomes than amateur settings, likely due to greater access to trained personnel and defibrillation equipment [16]. This supports a broader consensus that system-level factors, rather than individual interventions alone, drive survival.

Accordingly, effective prevention strategies extend beyond individual response and require integrated infrastructure, including widespread public training in basic life support (BLS) and improved AED accessibility, particularly in schools and sports facilities [9,16]. High-risk sports settings should implement comprehensive emergency response plans [39], incorporating rapid recognition, clear response protocols, and immediate AED access.

Commotio cordis

Commotio cordis (CC) represents a distinct mechanism of SCA, defined as a blunt, nonpenetrating impact to the precordium that triggers malignant arrhythmia – most commonly ventricular fibrillation (VF), though a broader spectrum of arrhythmias has also been reported –without structural cardiac injury [40]. Although blunt chest trauma is relatively common in both sports and everyday activities, progression to CC is rare [41]. This reflects the requirement for a highly specific combination of factors, including impact location directly over the cardiac silhouette, a velocity typically in the range of 30-50 miles per hour, and precise timing within a narrow window of cardiac repolarization, estimated at approximately 20 ms [42]. As a result, CC is largely unpredictable and cannot be identified through conventional pre-participation screening, as it does not depend on underlying structural heart disease [43].

Despite this classical definition, the concept of CC is evolving. Increasingly, authors argue that restricting the diagnosis to structurally normal hearts may be overly narrow. A broader framework has been proposed in which a chest impact acts as the immediate trigger of arrhythmia even in individuals with pre-existing cardiac conditions [40]. This interpretation presents CC as a spectrum of mechanically induced electrical instability rather than a strictly defined condition. While there is general agreement that a mechanical trigger is central to the condition, disagreement persists regarding classification, which may partly explain differences in reported epidemiological estimates.

CC is the third most common cause of SCD in young athletes [44]. It is most frequently associated with sports involving projectiles or direct physical contact, such as baseball, lacrosse, American football, ice hockey, and, more recently, soccer and rugby [45]. Across studies, the highest risk is observed in adolescent males, likely due to increased chest wall compliance. The mean age of affected individuals is approximately 13.8 years, with only about 22% of cases occurring in those aged ≥18 years [46]. Although most events occur during organized sports (62%), a substantial proportion (38%) arises in non-sport settings, including recreational play, indicating that even relatively low-energy impacts may act as triggers under specific conditions.

Survival outcomes following CC vary markedly depending on the context of the event. Higher survival rates are consistently reported in organized sports compared with non-competitive settings, largely due to faster recognition, higher rates of bystander CPR, and earlier access to AEDs [47]. Conversely, delayed recognition and limited access to resuscitation in non-sport environments contribute to worse outcomes. These observations reinforce the viewpoint that rapid recognition, immediate CPR, and early defibrillation are the key determinants of survival [48].

Preventive strategies remain limited. Protective equipment, such as chest barriers, has not been shown to reliably prevent arrhythmia and may provide a false sense of security [46,49], as impacts transmitted through the barrier can still induce VF. Therefore, reducing mortality depends less on preventing the triggering impact and more on optimizing emergency response systems, including CPR training, AED accessibility, and structured emergency action plans [44]. Accordingly, individuals involved in sports should be familiar with CC and appropriate emergency procedures.

Management of CC is identical to standard SCA protocols and requires immediate CPR and rapid defibrillation. Over recent decades, survival has improved substantially [42], increasing from 14.8% in earlier periods to 66.6% in more recent cohorts, most likely reflecting increased awareness, better training, and wider AED availability rather than changes in underlying mechanisms. Survivors do not appear to have an increased long-term risk of recurrent SCD or persistent arrhythmogenic heart disease. Recurrence of CC is exceedingly rare, and implantable cardioverter-defibrillators (ICDs) are not routinely indicated, as they do not prevent subsequent mechanically triggered events [44,50].

Discussion of the review results

SCD and SCA in athletes remain rare but highly impactful events with important clinical, social, and public health implications. Their occurrence in young, apparently healthy individuals requires a comprehensive and system-oriented preventive approach. The evidence reviewed in this paper highlights the heterogeneous etiology of these events, including structural heart diseases, primary electrical disorders, acquired conditions, and cases that remain unexplained despite detailed evaluation. This variability highlights the challenges of accurate risk stratification and suggests that uniform prevention strategies may not be sufficient across diverse athlete populations.

A consistent finding across studies is the substantial variability in reported incidence and underlying causes of SCD/SCA. This reflects both true population differences and methodological inconsistencies, including varying definitions and study populations. As a result, comparisons between studies are limited, and the true burden of disease may be underestimated. These observations highlight the need for standardized reporting systems and national registries to improve epidemiological accuracy and support targeted prevention strategies [13].

Pre-participation screening remains a cornerstone of primary prevention; however, its effectiveness continues to be debated. While the addition of ECG improves detection of certain cardiomyopathies, its overall impact on reducing SCD mortality is uncertain. Screening is further limited by false-positive results, reduced sensitivity for non-structural conditions, and the fact that many SCD cases occur in individuals with previously normal results. Taken together, current screening strategies appear insufficient to prevent the majority of cases, highlighting the need for more individualized and longitudinal approaches to risk stratification, potentially incorporating emerging technologies such as genetic testing and AI. Importantly, balancing sensitivity and specificity remains challenging, as excessive screening may lead to overdiagnosis and unnecessary sports restriction.

In contrast, secondary prevention strategies demonstrate a clear and consistent impact on survival. Early recognition of SCA, prompt CPR, and rapid defibrillation using AEDs remain the most effective interventions. Importantly, survival outcomes are largely determined by system-level preparedness, including AED availability and public training in BLS [4,38]. This emphasizes that improving outcomes depends not only on medical advances but also on public health policies and education.

Post-mortem evaluation, including comprehensive and molecular autopsy, plays an important role in identifying underlying causes of SCD and enabling cascade screening in affected families, although its diagnostic yield remains incomplete.

The main limitation of this review is its narrative design and restriction to English-language publications, which may introduce selection bias. However, the inclusion of cohort studies, registry data, and international guidelines strengthens the comprehensiveness and clinical relevance of the analysis.

This review contributes to current knowledge by providing an updated synthesis of epidemiology, etiology, and prevention strategies for SCD and SCA in athletes. It emphasizes the limited effectiveness of screening, highlights the central role of emergency preparedness, and supports a shift toward more personalized and system-based prevention models. Additionally, it underscores the emerging role of MA and AI-based approaches as complementary tools that may enhance diagnostic precision and future risk stratification.

Conclusions

SCD and SCA in athletes represent rare but devastating events with complex etiologies. While pre-participation screening plays a relevant role, it cannot fully prevent such outcomes. Strengthening mandatory reporting systems, establishing national registries, and standardizing comprehensive autopsy and molecular diagnostic protocols are crucial to improving etiological understanding and preventive strategies. Ultimately, reducing cardiovascular mortality in sport requires an integrated approach that combines reliable epidemiological surveillance, accurate etiological diagnosis, and robust emergency preparedness, including widespread CPR education and universal access to defibrillation.

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Artificial intelligence (ChatGPT) was used solely for the initial language editing and grammar correction of the English text to improve readability and linguistic accuracy. All the scientific content, ideas, data interpretation, conclusions and intellectual contributions were conceived and written by the authors. No AI tool was used to generate new scientific content, replace author judgment, create figures, or fabricate results.

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