Causes and Risk Factors of Sudden Cardiac Death in Athletes: Demographic and Environmental Impacts and the Importance of Screening – A Literature Review

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Abstract

Aim

The review focuses on understanding the etiological, demographic, and external contributors to sudden cardiac death in athletes, while also introducing a screening model and specifying the most important diagnostic methods.

Materials and Methods

A systematic review was conducted, including meta-analyses, randomized controlled trials, and systematic reviews published between 1998 and 2024, involving professional athletes of different ages and sporting disciplines.

Results

Sudden cardiac death (SCD) among athletes arises from a complex interaction of individual predispositions, the nature of the sport, and training environments. The estimated incidence is approximately 1 per 40,000–80,000 person-years. Men exhibit a higher risk than women, attributable to both biological factors and differing patterns of sports engagement. In athletes under 35, hypertrophic cardiomyopathy is the predominant cause, whereas coronary artery disease is most frequent in those over 35. The highest SCD rates occur in basketball and football, likely due to the considerable cardiovascular demands of high-intensity activity. Current meta-analyses indicate that COVID-19 does not significantly elevate SCD risk, whereas doping substantially increases mortality. Preventive strategies include pre-participation screening, timely access to automated external defibrillators (AEDs), and immediate resuscitation.

Conclusion

Sudden cardiac death (SCD) in athletes is an uncommon event, with risk determined by age, sport type, and individual characteristics. Despite variations in SCD incidence among different athletic populations, systematic screening remains essential for preventing fatal cases. Optimal prevention strategies should include structured screening programs, targeted education, personalized risk evaluation, and immediate access to automated external defibrillators (AEDs) along with adequate first aid training.

Keywords and search strategies: sudden cardiac death, SCD, SCA, athletes, sport, hypertrophic cardiomyopathy, AAS, anabolic-androgenic steroids
Additional filters: myocarditis, coronary artery disease, prevention, screening

1. Introduction

Sudden cardiac death (SCD) is defined as an unexpected death due to cardiac causes, typically characterized by a sudden loss of consciousness. It usually occurs in individuals without a previously diagnosed heart condition and most often presents without warning symptoms. The diagnostic criterion is death occurring within one hour of the onset of the first clinical manifestations [21]. SCD is relatively rare, with an estimated annual prevalence of approximately 0.1% in the general population. Nevertheless, it represents a significant challenge for healthcare systems, underscoring the importance of identifying risk factors and developing effective preventive strategies [22].

The aim of this paper is to systematically review recent literature on the impact of various etiological factors, the incidence of SCD in young athletes, and the role of screening in this population.

2. Results of the impact of various factors divided into individual sections

2.1.Epidemiology

Epidemiological data from the past two decades indicate that the true incidence of sudden cardiac death (SCD) in athletes is difficult to establish with precision. The most frequently cited large cohort study reports an incidence ranging from 1 case per 40,000 person-years to 1 case per 80,000 person-years [1]. By contrast, the most recent cohort study of NCAA (National Collegiate Athletic Association) athletes, spanning 20 years (2000–2019), reported an overall incidence of 1 case per 63,682 person-years [2]. The variability in these estimates is largely attributable to differences in study populations, including age distribution and type of sport practiced. Consequently, the reported figures should be interpreted as approximate indicators of risk. Although statistically rare, SCD remains a major focus of research in sports medicine and interventional cardiology.

2.2.Demographic factors

Available analyses in athlete populations clearly demonstrate that sex is a significant determinant of SCD risk. A large cohort study of NCAA (National Collegiate Athletic Association) athletes found a higher incidence of SCD in men compared to women, with rates of 1 case per 43,348 person-years and 1 case per 164,504 person-years, respectively [2]. These differences cannot be explained solely by the greater number of male competitive athletes but are also attributable to biological, hormonal, and structural factors.

A comprehensive review reported that while men and women are born with a similar number of cardiomyocytes, adult men have a significantly lower proportion. With respect to hormonal influences, higher circulating concentrations of the predominant form of estrogen, 17β-estradiol (E2), are inversely correlated with the incidence of heart disease, strongly suggesting a cardioprotective role of E2. Conversely, clinical studies on testosterone indicate that both deficient and excessive levels are associated with adverse cardiovascular outcomes. Animal models have demonstrated a reduction in cardiomyocyte count and diastolic dysfunction in testosterone deficiency, while excessive testosterone may promote cardiomyocyte apoptosis, contributing to the lower cardiomyocyte proportion observed in male hearts [15].

Race is another demographic factor associated with differences in SCD incidence among athletes. In the previously cited study, the incidence of SCD among Black athletes was 1 case per 27,217 person-years, compared with 1 case per 74,581 person-years in White athletes [2]. An analysis of the NCAA database from 2003 to 2013 further confirmed racial disparities, with 57% of recorded deaths occurring in White athletes and 38% in Black athletes. Athletes of other racial and ethnic groups, including Hispanic and Asian athletes, accounted for approximately 15% of cases [10]. Although these findings highlight disparities, the available data remain inconclusive and suggest that the risk of SCD is not evenly distributed across racial groups within the athletic population.

2.3.Cardiogenic factors

Differences in the etiology of SCD are closely associated with the age of athletes. Hypertrophic cardiomyopathy (HCM) is the leading cause of SCD in young competitive athletes (<35 years), accounting for 36% of confirmed cases in the American National Registry

of Sudden Death in Athletes [9]. Among older athletes (>35 years), coronary artery disease (CAD) is most frequently identified. However, it remains debated whether SCD in this group results primarily from atherosclerotic plaque rupture or an imbalance between myocardial oxygen supply and demand [3,4]. In a large cohort study, the most frequent autopsy finding was unexplained sudden death with negative autopsy results (AN-SUD, 19.5%), followed by idiopathic left ventricular hypertrophy or possible cardiomyopathy (16.9%) and HCM (12.7%) [2].

Etiological patterns also vary by sex. According to a meta-analysis, HCM was the leading cause of SCD in men (45.1%), followed by congenital coronary artery anomalies (16.0%) and CAD (10.3%). In women, congenital coronary anomalies were most common (33.0%), followed by HCM (13.9%) and arrhythmogenic right ventricular cardiomyopathy (ARVC, 12.2%). Differences in CAD incidence between sexes mirror those observed in the general population, with women typically developing CAD later in life due to the cardioprotective effect of estrogens [4].

Moreover, a large systematic review combining race, sex, and sport type demonstrated that male basketball players had a disproportionately high risk of SCD due to cardiomyopathy or probable cardiomyopathy (including HCM, dilated cardiomyopathy, unspecified cardiomyopathy, and idiopathic left ventricular hypertrophy/possible cardiomyopathy) compared with other male athletes [10].

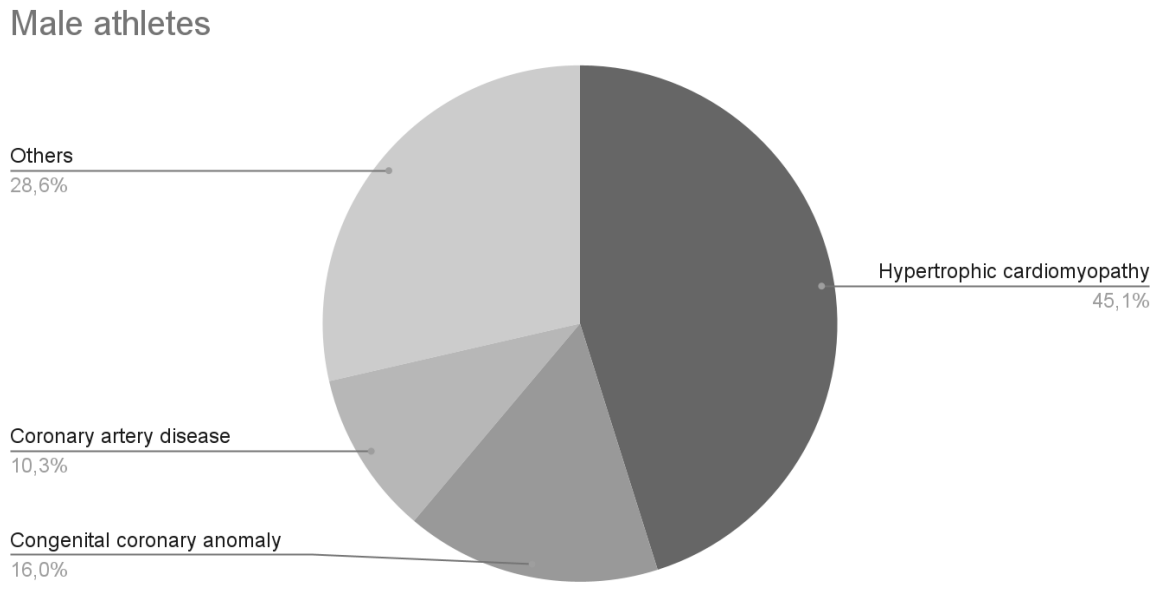

Table 1. Etiologies of SCA in male athletes [4].

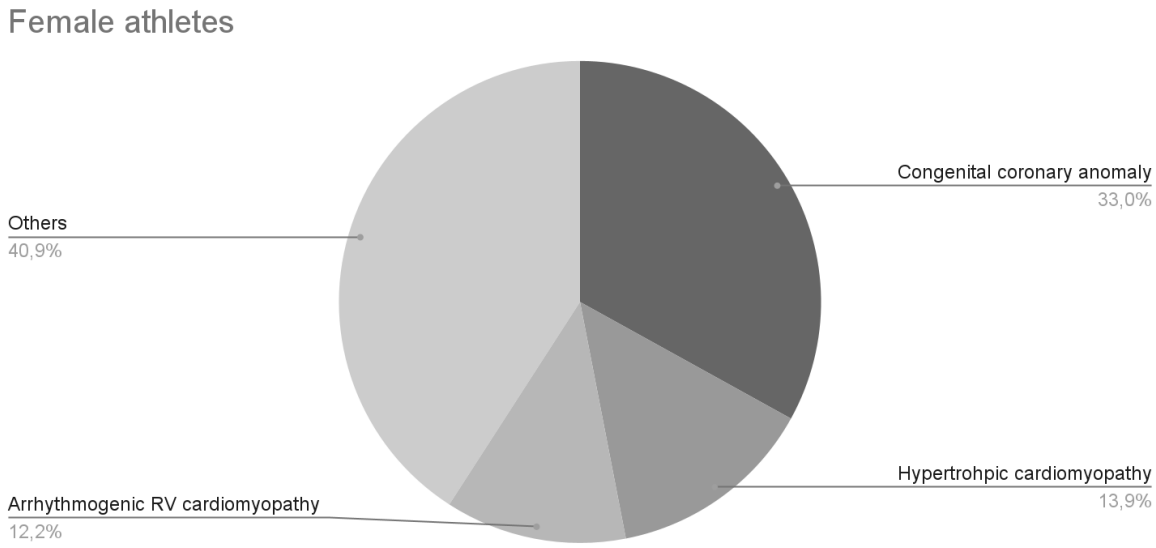

Table 2. Etiologies of SCA in female athletes [4].

2.4.Type of sport

The incidence of SCD in athletes should be interpreted in relation to the general population. Studies suggest that physical exertion associated with sports and training increases the risk of sudden cardiac arrest (SCA) and SCD by 2.4–4.5% compared with non-athletes and recreational athletes. While athletic activity elevates relative risk, the absolute number of cases remains higher in non-athletic populations. In the 10-year Oregon Sudden Unexpected Death Study, 1,184 cases of non-sports-related SCA were recorded, compared with only 63 sports-related cases among individuals aged 35–65 [9].

Sport-specific analyses demonstrate that basketball players (both men and women) have the highest risk of SCD, at 1 case per 15,462 athlete-years (AY). Elevated risks are also observed in men's soccer (1:23,689 AY), men's American football (1:35,951 AY), and men's and women's cross-country running (1:44,973 AY). In terms of etiology, male basketball players are more likely to die from congenital coronary artery anomalies than other male athletes, while Black athletes are more frequently affected by coronary anomalies compared with White athletes [10].

2.5. COVID-19

COVID-19 infection has emerged as a potential factor influencing the incidence of SCD. A 2022 meta-analysis of retrospective studies comparing patients hospitalized with

COVID-19 to those with other respiratory infections found new-onset myocarditis in 0.12% of COVID-19 patients versus 0.04% of controls, representing a 2–3-fold increased incidence after SARS-CoV-2 infection [5]. Based on these findings, researchers investigated whether the risk of SCD among athletes differed before and after the COVID-19 pandemic. A meta-analysis encompassing 347,092,437 person-years (PY) reported that SCD attributable to myocarditis was extremely rare (≈0.047/100,000 PY), with comparable incidence rates before and after the pandemic [6].

Another global study of professional football players reported that in those aged >35 years, coronary artery disease (CAD) was the predominant diagnosis both before (69.8%) and during (54.8%) the pandemic. Among players aged ≤35 years, myocarditis was diagnosed in four cases (4%) before and one case (1%) during the pandemic [7].

2.5. Doping substances

Doping substances and stimulants significantly increase the risk of cardiovascular disease, both directly—through structural and functional damage to the myocardium—and indirectly, by pushing the body beyond physiological limits. The World Anti-Doping Agency (WADA) identifies anabolic agents and oxygen-carrying modulators as particularly dangerous. Despite frequent claims of “clean sport,” routine testing often fails to detect many of these substances.

Anabolic agents represent the oldest and most extensively studied group of doping substances. When combined with training, they increase muscle mass and strength while reducing body fat. Their use, however, is not limited to strength sports; they are also employed in endurance disciplines to enhance recovery. Cohort studies estimate that mortality among athletes using anabolic steroids is 6–20 times higher than in non-users, with approximately one-third of deaths attributable to cardiovascular disease. The most common complications include cardiomyopathy, myocardial infarction, and coagulation disorders [16].

A large study by the European Association of Preventive Cardiology (EAPC) reported that testosterone directly impairs vascular reactivity by enhancing the response to noradrenaline [17]. Similarly, a systematic review of studies published between 1991 and 2019 concluded that anabolic steroids can disrupt coagulation, promote thrombotic complications and myocardial infarction, and trigger life-threatening arrhythmias leading to sudden cardiac death [18].

Substances that increase oxygen availability enhance performance by improving cardiac output and oxygen uptake in peripheral tissues. This type of doping is difficult to detect, as it often involves transfusion of previously collected autologous blood or the use of recombinant erythropoietin (EPO). Population studies have demonstrated that even a twofold increase in EPO concentration is associated with a 25% higher risk of heart failure. Similar effects can be achieved with cobalt chloride, which stimulates erythropoiesis but may also induce dilated cardiomyopathy [16].

β_2-agonists, such as salbutamol, are commonly administered via inhalation for the treatment of bronchial asthma. However, when taken orally, they enhance muscle strength and mass and, in some cases, reduce body fat. Their use has been linked to an increased risk of cardiovascular disease, including congestive heart failure, arrhythmias, and sudden cardiac death [17].

A review examining the relationship between doping and sudden cardiac death analyzed 13 studies. The data confirmed that doping is more prevalent among men, particularly bodybuilders. Among the reported cases of sudden cardiac death, 33 individuals had no relevant personal or family history. The most frequent autopsy findings were cardiomegaly (33%) and left ventricular hypertrophy (30%). In all cases, death was confirmed as SCD, with a strong correlation to the use of anabolic agents [19].

3. Screening and prevention

Although rare, cases of sudden cardiac death (SCD) among young competitive athletes receive considerable media attention. Identifying individuals at risk has therefore become a priority for both sports and medical communities, as many of the underlying conditions can be diagnosed during life and targeted interventions implemented to reduce mortality. Several major organizations, including the European Society of Cardiology (ESC), the American Heart Association (AHA), the International Olympic Committee (IOC), and the Fédération Internationale de Football Association (FIFA), recommend pre-participation cardiovascular screening (PSS) in young athletes. However, the uptake of such screening remains limited, mainly due to high costs and the lack of unequivocal evidence regarding its effectiveness [11].

One of the most frequently cited examples comes from Italy, where legislation requires all competitive athletes in the Veneto region to undergo cardiovascular screening. The protocol includes a detailed personal and family history, physical examination, and a 12-lead ECG, with

further tests reserved for those with abnormal initial findings. Over 26 years of follow-up, statistical analyses demonstrated a significant decline in the incidence of SCD. The annual rate decreased by 89%, from 3.6 to 0.4 per 100,000 person-years. This reduction was driven primarily by a decline in deaths due to cardiomyopathies, which fell from 1.50 to 0.15 per 100,000 person-years [12].

In the United States, pre-participation cardiovascular screening has traditionally relied on personal and family medical history along with physical examination, without routine 12-lead ECG. This approach was recommended by the American Heart Association's Committee on Sudden Death and Congenital Heart Disease, based on the assumption that ECG is not cost-effective for screening large populations of young athletes due to its limited specificity. However, evidence indicates that this method has low sensitivity for detecting serious cardiovascular conditions. An analysis of 134 high school and collegiate athletes who suffered sudden death revealed that only 3% had abnormalities suspected during pre-participation screening, and fewer than 1% received a correct diagnosis [13].

Hypertrophic cardiomyopathy (HCM), defined morphologically as left ventricular hypertrophy in the absence of other causes such as hypertension, is considered the leading cause of sudden death among athletes. Echocardiography is the most effective diagnostic tool for HCM but is expensive and impractical for large-scale screening. The 12-lead ECG offers a more cost-effective alternative. Studies demonstrate that when combined with medical history and physical examination, ECG effectively identifies HCM in young athletes [13].

The modern approach to sports cardiology increasingly considers the use of genetic testing in the prevention of sudden cardiac death (SCD). DNA analysis enables the identification of individuals carrying pathogenic mutations, allowing for tailored training modifications and appropriate cardiological care. However, genetic testing in sports also raises several limitations and controversies, including high costs and legal and ethical concerns regarding genetic privacy and potential discrimination.

Since the late 20th century, numerous studies have demonstrated that a family history of SCD increases individual risk, suggesting a genetic predisposition. The primary hereditary conditions predisposing to arrhythmias can be divided into two categories: (1) primary hereditary arrhythmia syndromes, known as “channelopathies,” which include long QT syndrome (LQTS), Brugada syndrome (BrS), and catecholaminergic polymorphic ventricular tachycardia (CPVT); and (2) cardiomyopathies, including hypertrophic, dilated, and arrhythmogenic cardiomyopathy [20].

Scientific societies such as the European Society of Cardiology (ESC) and the American Heart Association (AHA) recommend genetic testing for individuals with a documented family history of hereditary disease or with abnormalities detected in screening tests suggestive of cardiomyopathy or arrhythmic syndromes. Routine genetic testing of asymptomatic athletes without a relevant family history is not advised.

Protocol of pre-participation cardiovascular screening

The initial cardiovascular assessment should include a detailed personal and family history, a physical examination with blood pressure measurement, and a 12-lead electrocardiogram (ECG). This evaluation should be conducted by a physician with appropriate training, medical expertise, and awareness of cultural considerations, enabling accurate identification of symptoms and signs of cardiovascular disease that may predispose to sudden death during physical exertion. In some countries, postgraduate training in sports medicine with a focus on sports cardiology is required.

These examinations should be performed at the onset of sporting activity, which often occurs at an average age of 13, and should be repeated regularly at least every two years.

The personal history interview should include questions about exertional chest pain or discomfort, episodes of syncope, or irregular heartbeat. The family history should cover genetic conditions to which the athlete may be predisposed. A family history is considered positive if a close relative has experienced a premature myocardial infarction or sudden death (before age 55 in men and 65 in women), or if there is a history of cardiomyopathy, Marfan syndrome, long QT syndrome, Brugada syndrome, severe arrhythmias, coronary artery disease, or other significant cardiovascular disorders.

The physical examination should include assessment of systolic and diastolic murmurs, blood pressure measurement, and evaluation of heart rate in the upper and lower limb arteries [13]. The ECG is considered abnormal if any of the aforementioned findings are present. The specific criteria are summarized in Table 3 [14].

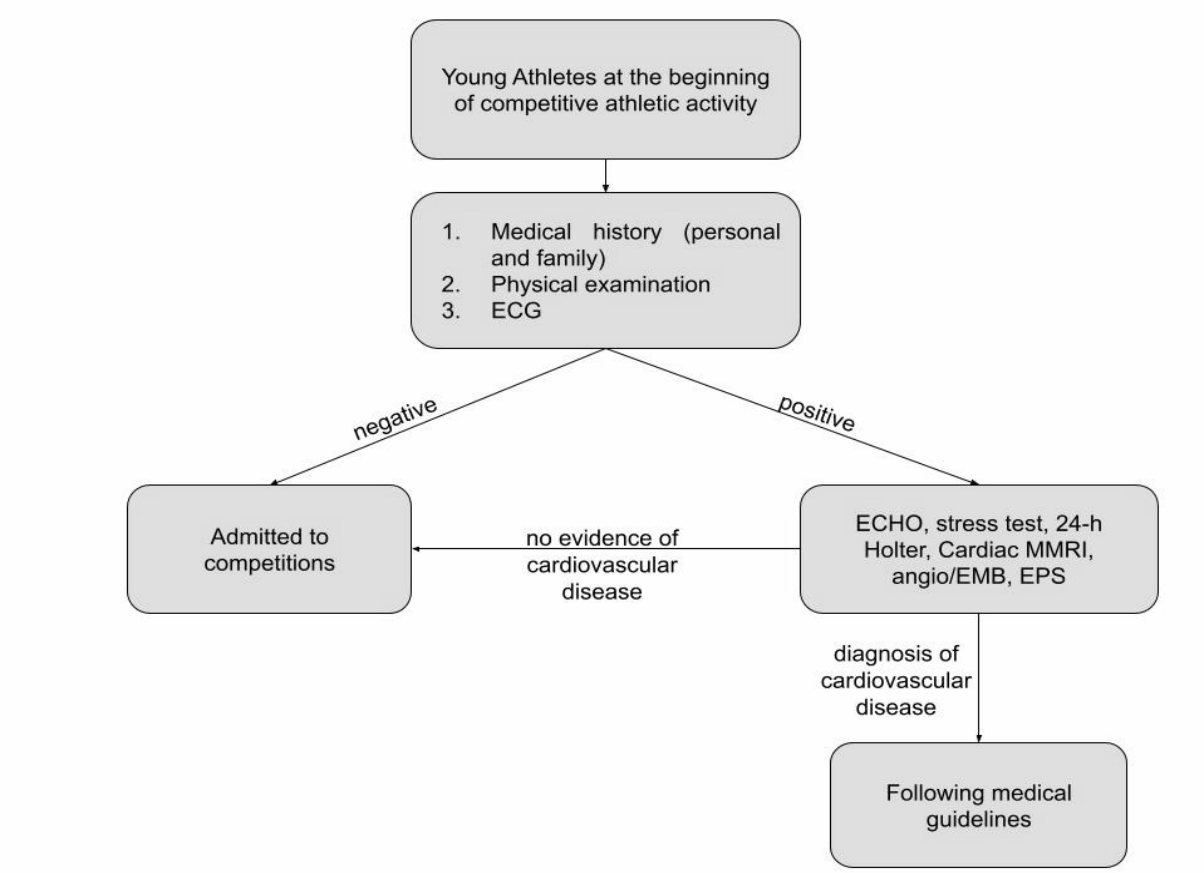

Figure 1.Protocol of pre-participation cardiovascular screening [13].

Criteria for a positive 12-lead ECG
P wave • left atrial enlargement: negative portion of the P wave in lead V1 0.1 mV in depth and 0.04 s in duration; • right atrial enlargement: peaked P wave in leads II and III or V1 0.25 mV in amplitude.
QRS complex • frontal plane axis deviation: right þ 1208 or left –308 to –908; • increased voltage: amplitude of R or S wave in in a standard lead 2 mV, S wave in lead V1 or V2 3 mV, or R wave in lead V5 or V6 3 mV; • abnormal Q waves 0.04 s in duration or 25% of the height of the ensuing R wave or QS pattern in two or more leads; • right or left bundle branch block with QRS duration 0.12 s; • R or R' wave in lead V1 0.5 mV in amplitude and R/S ratio 1.
ST-segment, T-waves, and QT interval • ST-segment depression or T-wave flattening or inversion in two or more leads; • prolongation of heart rate corrected QT interval .0.44 s in males and . 0.46 s in females.
Rhythm and conduction abnormalities • premature ventricular beats or more severe ventricular arrhythmias; • supraventricular tachycardias, atrial flutter, or atrial fibrillation; • short PR interval (,0.12 s) with or without 'delta' wave; • sinus bradycardia with resting heart rate 40 beats/mina; • first (PR 0.21 sb), second or third degree atrioventricular block.

Table 3 Criteria for a positive 12-lead ECG [14].

4. Discussion

An analysis of available epidemiological data indicates that the incidence of sudden cardiac death (SCD) in athletes results from a complex interaction between individual factors, the characteristics of the sport practised, and the training environment. Although precise estimates are challenging, the most frequently cited incidence of SCD in athletes ranges from 1 case per 40,000 person-years to 1 case per 80,000 person-years. It is evident, however, that men have a significantly higher incidence of SCD than women. This difference is not solely attributable to the greater number of men participating in competitive sports, but also reflects biological, hormonal, and structural factors. Studies examining racial differences have not demonstrated a clear distribution pattern.

The underlying causes of SCD depend on gender, age, and the type of sport. In athletes under 35 years of age, hypertrophic cardiomyopathy is the most common condition, whereas in athletes over 35, coronary artery disease predominates. Data on the impact of the specific sport are less definitive, but the highest rates of SCD are observed among male basketball players at the professional or collegiate level, as well as in football. This may be related to the substantial cardiovascular load associated with combined dynamic and static exercise, compounded by genetic predispositions such as hypertrophic cardiomyopathy or arrhythmogenic right ventricular cardiomyopathy.

Regarding the impact of COVID-19, research remains relatively limited and preliminary. Large meta-analyses do not indicate a significant increase in SCD risk following SARS-CoV-2 infection. In contrast, doping is associated with a marked increase in mortality. Autopsy studies frequently identify hypertrophic cardiomyopathy and other cardiac abnormalities as complications linked to the use of anabolic agents, erythropoiesis-stimulating drugs, or other performance-enhancing substances. Many of these agents are deliberately modified to evade detection, which complicates research; nonetheless, the association between doping and SCD remains robust.

Pre-participation cardiovascular screening is consistently emphasized in the literature as a critical preventive measure. Although recommendations vary across sports organisations due to considerations of cost, feasibility, and ethical concerns, the overall evidence supports the value of screening. Equally important for survival during sudden cardiac events is the rapid availability of automated external defibrillators (AEDs) and immediate cardiopulmonary

resuscitation (CPR), highlighting the need for preventive and educational strategies across all levels of athletic participation.

Presenting precise epidemiological results is challenging due to methodological heterogeneity among studies, variations in event definitions, and differences in the populations analysed. Moreover, the exact incidence of sudden cardiac events remains uncertain because of incomplete mandatory reporting and limited infrastructure for capturing all cases in the general population.

5. Conclusions

Sudden cardiac death in athletes remains a rare but dramatic event, with risk unevenly distributed across different sports and age groups. In younger athletes, congenital and genetically determined heart diseases predominate, whereas ischaemic heart disease is more prominent in older athletes. The highest relative risk is observed in competitive sports such as basketball, while the greatest absolute number of cases occurs in widely practiced team sports like football. Effective prevention requires a multi-level strategy, encompassing pre-participation screening, targeted health education, individualized risk assessment, and ensuring rapid access to life-saving interventions, including automated external defibrillators and trained first responders.

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AI Statement

In this study, ChatGPT was used to improve the linguistic accuracy and readability of the text and to refine the English language to ensure consistency and compliance with academic standards of scientific writing. The aim was also to maintain the grammatical correctness of the text. It should be emphasised that artificial intelligence tools were used solely as auxiliary instruments under the supervision of the authors. The AI tools were used by the authors primarily to increase data processing efficiency, pattern recognition and linguistic refinement, rather than to replace human judgement in the analytical process.

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