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REVIEW PAPER

Sudden cardiac death in young athletes: current evidence and clinical implications

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Abstract

Sudden cardiac death in athletes, although relatively rare, remains a significant clinical concern due to its occurrence in young, often asymptomatic individuals. The most common causes include inherited cardiomyopathies, coronary anomalies, arrhythmogenic right ventricular cardiomyopathy, and primary electrical disorders (channelopathies). The incidence varies across populations and sporting disciplines, reflecting both epidemiological differences and variations in data collection. Early identification of at-risk individuals is central to prevention. Pre-participation screening, particularly with the inclusion of a 12-lead electrocardiogram (ECG), significantly improves detection of underlying cardiac abnormalities compared with a history and physical examination. Risk stratification should be individualized and may incorporate clinical evaluation, imaging techniques, ambulatory monitoring, and genetic testing. Equally important is effective emergency preparedness. The implementation of structured emergency action plans, including rapid recognition of cardiac arrest, immediate cardiopulmonary resuscitation, and early use of automated external defibrillators, has been shown to markedly improve survival outcomes. Future advances in genetic testing and the development of more sophisticated AI-assisted ECG analysis may further enhance early detection and risk stratification.

Keywords: sudden cardiac death, mass screening, athletes, cardiology, cardiovascular diseases

Introduction

The European Society of Cardiology (ESC) guidelines define sudden cardiac death (SCD) as an unexpected, non-traumatic death that occurs within one hour of symptom onset in individuals with a known or suspected cardiac condition or when autopsy findings suggest a likely cardiac cause, with non-cardiac causes ruled out [1]. Competitive athletes may increase their risk of adverse cardiac events by pushing their bodies to physiological extremes through intense training, supplement use, or doping [2]. SCD can occur in young, apparently healthy individuals, including athletes, who are often considered among the healthiest members of society. In athletes under 35, SCD might be the first clinical sign of an otherwise undetected condition [3]. SCD is the leading medical cause of unexpected death among athletes, although its frequency varies depending on the population studied. Although relatively rare, these events in young, asymptomatic patients have a significant emotional and social impact on their communities [4]. A variety of cardiovascular abnormalities can lead to sudden death in young

athletes, and even minor structural changes can become life-threatening. These conditions are often clinically silent and difficult to detect. Early identification through systematic pre-participation screening may help reduce both the risk and occurrence of sudden death [5].

Aim of the work

This review provides a comprehensive overview of SCD in young athletes, focusing on its epidemiology, underlying causes, current therapeutic approaches, and preventive strategies. It aims to raise awareness among healthcare professionals, coaches, and athletes of potential cardiovascular risks, emphasize the importance of early recognition, and highlight preventive measures to reduce the incidence and consequences of SCD in this population.

Methods

This review synthesizes current evidence on SCD in athletes, with a specific focus on the role of pre-participation cardiovascular screening and preventive measures. A comprehensive literature search was conducted in PubMed and Google Scholar from October 2025 to February 2026. Only peer-reviewed articles covering SCD, including its pathophysiology, risk factors, and evidence-based prevention strategies in athletes, were included. Key findings were extracted and organized to clarify underlying mechanisms, potential triggers, and relevant preventive measures for competitive athletes. Most of the studies reviewed were published between 2016 and 2026. During the search, about 400 articles were retrieved from PubMed and 250 from Google Scholar. After screening titles and abstracts and reviewing full texts, 47 articles were ultimately included. This process ensured the inclusion of studies directly relevant to SCD in athletes with rigorous methodology.

Literature review results

Epidemiology and pathophysiology of SCD in athletes

SCD incidence is estimated at 2-6.3 per 100,000 person-years in physically active adults (~35 years), compared with 0.4-3 per 100,000 in competitive athletes ≤35 years, indicating a lower SCD burden in the latter cohort. Differences in reported rates arise from regional and

epidemiological variation, along with inconsistencies in registry methodologies, including how athletes are defined and how cases are identified [6]. SCD occurs predominantly in males (80-95%). Key risk factors include older age (linked mainly to coronary artery disease (CAD)) and Afro-Caribbean ethnicity, associated with a higher prevalence of hypertrophic cardiomyopathy (HCM) [7]. In the United States, SCD risk is higher among Black athletes compared with White athletes (5.65 vs 3.02 per 100,000 person-years). Similar disparities were observed in a UK cohort of adolescent football players, where incidence was substantially greater in Black athletes than in White athletes (25 vs 3.86 per 100,000 person-years). SCD incidence also varies by sport, with the highest rates reported in ice hockey (4.25 per 100,000 person-years), basketball (2.51), and American football (1.21). In a UK registry of 357 SCD cases, running and football were most frequently affected (25% each), followed by cycling and gymnastics (8% each) [8]. The main causes of SCD in athletes are well documented in literature and include both congenital and acquired heart conditions [9]. SCD is mainly caused by inherited cardiac diseases. In the U.S., the leading cause is HCM (36%), followed by coronary anomalies (17%), with arrhythmogenic right ventricular cardiomyopathy (ARVC) and channelopathies less common. In Italy, ARVC predominates (22%), coronary anomalies follow (12%), while HCM is rare (2%), likely due to mandatory electrocardiogram-based (ECG) screening and population differences [10]. No nationwide registries have been performed in Poland, but due to geographic and racial similarities, the German epidemiology of SCD may correspond better to the Polish situation than the North American or Mediterranean [7]. National registries are important for optimizing screening and prevention strategies. In Germany, the leading causes of sudden cardiac arrest in young athletes are premature CAD, sudden arrhythmic death syndrome (SADS), and myocarditis, highlighting clear international differences in underlying etiology [11]. The leading causes of SCD in young athletes are presented in Table 1.

Table 1. Leading causes of SCD in young athletes. The following sections provide detailed descriptions of the conditions mentioned above, as the study concentrates on these entities [3,9,12-15]

Cardiac structural disorders	Cardiac electrical disorders	Cardiac acquired disorders
Hypertrophic cardiomyopathy	Long QT syndrome	CAD
Anomalous coronary arteries	WPW syndrome	Commotio cordis
ARVC	CPVT	Drug-induced cardiotoxicity

Congenital heart defects	-	Myocarditis
MVP	-	-

Notes: ARVC – arrhythmogenic right ventricular cardiomyopathy, MVP – mitral valve prolapse, CPVT – catecholaminergic polymorphic ventricular tachycardia, CAD – coronary artery disease, WPW – Wolff-Parkinson-White.

Cardiac structural disorders

HCM

HCM is the most common inherited cardiac disorder, characterized by abnormal thickening of the ventricular myocardium. The disease exhibits a wide range of clinical presentations and phenotypes, often resulting from mutations in sarcomeric proteins. Symptoms can include shortness of breath, chest pain, palpitations, and fainting. Additionally, the condition is linked to an increased risk of SCD, heart failure, and thromboembolic events [16]. Transthoracic echocardiography is the primary diagnostic tool, while cardiac magnetic resonance (CMR) imaging also plays an important complementary role. In adults, HCM is diagnosed through imaging showing a left ventricular wall thickness of ≥15 mm that cannot be explained by loading conditions or other systemic or metabolic diseases [17]. Resting left ventricular outflow tract (LVOT) obstruction in HCM indicates a higher risk of adverse outcomes and worsening heart failure. Physical examination may be normal or may reveal characteristic signs, including a fourth heart sound (S4), systolic murmurs (such as an ejection murmur at the left sternal border that worsens with the Valsalva maneuver), paradoxical splitting of S2, a forceful apical impulse, and systolic thrill. The arterial pulse may be bifid with a dome-shaped systolic peak, and the venous pulse might display a prominent “a” wave [18]. HCM has traditionally been regarded as a high-risk disorder for exercise-induced SCD, leading to the standard recommendation to exclude affected individuals from moderate- and vigorous-intensity sports. However, it is now recognized as a heterogeneous disease with variable individual risk, supporting a more personalized approach to participation decisions [19]. Managing HCM is complex and tailored to each patient. The main treatment goals are symptom relief, especially in patients with dynamic outflow tract obstruction, and risk assessment for SCD to facilitate preventive strategies [20].

Anomalous coronary arteries

Congenital coronary artery anomalies are rare, occurring in less than 1% of the population, and include abnormal coronary artery origins, as well as developmental anomalies such as fistulae and aneurysms. They may occur alone or in combination with other congenital heart defects, such as transposition of the great arteries, truncus arteriosus, or tetralogy of Fallot. While many patients remain asymptomatic, some are at risk for serious complications, including myocardial infarction, ventricular arrhythmias, and SCD. These anomalies are increasingly identified through advanced imaging techniques, especially computed tomography (CT) and magnetic resonance imaging (MRI) [21]. With the rising use of coronary computed tomography angiography (CCTA) in evaluating acute chest pain, coronary artery anomalies and variants are detected more often. Although most are clinically insignificant, some can cause myocardial ischemia due to flow limitation. Therefore, it is important to distinguish clinically significant anomalies that may require intervention from incidental anatomical variants with little or no clinical relevance [22].

ARVC

ARVC is a genetic heart disorder characterized by fibrofatty replacement of the ventricular myocardium, which leads to ventricular arrhythmias and SCD [23]. Electrocardiographic abnormalities in ARVC highlight the importance of systematic cardiovascular screening in athletes to identify high-risk individuals early and decrease mortality [24]. Managing the condition is challenging due to disease progression and limited clinical evidence. Anti-arrhythmic medications have uncertain effectiveness. Radiofrequency ablation, especially when combining endocardial and epicardial approaches, can reduce the burden of arrhythmias. New therapies, like stereotactic radiotherapy and gene-based strategies, are currently being studied. Implantable cardioverter-defibrillators (ICDs) decrease mortality but pose risks such as inappropriate shocks and device-related complications [23]. Intense and prolonged exercise can accelerate ARVC progression and complicate diagnosis, since right ventricular enlargement can also occur in athletes. Exercise restriction is essential for both affected patients and those with genetic mutations [25]. Currently, there is no cure for ARVC. Treatment focuses on improving quality of life and reducing the risk of SCD through lifestyle changes, medications, ablation, ICD implantation, and heart transplantation [26].

Congenital heart defects

Heart development is influenced by multiple intrinsic and extrinsic factors and involves interactions among various cardiac cell populations. Notably, interference at any stage of organogenesis can lead to congenital heart malformations caused by genetic, epigenetic, or environmental factors [12]. As understanding of the cardiovascular system and its pathophysiology has advanced, care for patients with congenital heart disease has improved, significantly increasing survival rates. Nevertheless, these improvements have introduced new challenges, with complications such as arrhythmias, endocarditis, pulmonary hypertension, and heart failure emerging. To address these issues, new surgical techniques, catheter-based interventions, electronic devices, and pharmacological treatments have been developed based on current knowledge and available evidence. In addition, growing attention is being paid to sports participation, pregnancy, work, and the social challenges these patients face [27]. Importantly, people with congenital heart disease tend to have reduced cardiorespiratory fitness (CRF), which declines faster than in healthy individuals. Since CRF is a predictor of mortality and morbidity, this highlights the importance of assessing physical activity interventions for clinical guidance [28].

MVP

MVP is a valvular heart disease, usually with a benign course, affecting about 3% of the population. It involves the prolapse of the mitral valve leaflet into the left atrium during systole. MVP is most commonly detected on cardiac auscultation, and the diagnosis is confirmed by echocardiography. In rare cases, it can lead to SCD, endocarditis, or stroke. MVP can be classified as primary or secondary. Primary MVP results from myxomatous degeneration of the valve, while secondary MVP is linked to systemic disorders such as Marfan syndrome, Ehlers-Danlos syndrome, polycystic kidney disease, Graves' disease, or pectus excavatum [13]. Clinical signs and complications are generally unrelated to physical exertion; therefore, individuals with MVP are usually able to participate in sports. However, in individuals with high-risk arrhythmogenic phenotypes, personalized assessment and monitoring are necessary in sports cardiology [29]. Echocardiography remains the primary diagnostic tool, while CMR is increasingly important due to its high accuracy and reproducibility. Indications for surgical

treatment of mitral regurgitation are continually updated, taking into account predicted risk, repair feasibility, and early signs of cardiac impairment [30].

Cardiac electrical disorders

Long QT syndrome (LQTS)

Chronic exercise in athletes can lead to QT interval prolongation, which is generally a benign physiological adaptation. However, similar QT prolongation occurs in congenital LQTS, which increases the risk of SCD, so its detection requires further evaluation. In athletes, this makes it harder to distinguish physiological adaptation from pathology [31]. Untreated LQTS carries a risk of potentially fatal ventricular arrhythmias, particularly during exercise, which in the past led to the recommendation of strict restrictions on athletic participation. However, with a growing understanding of the complex interactions between specific genotypes, modifying factors, and individual risk, current evidence indicates that such limitations are not always necessary. In many cases, patients who remain asymptomatic during exercise and are compliant with β-blocker therapy may safely engage in physical activity [32]. Treatment of congenital LQTS includes both pharmacologic and interventional approaches, such as left cardiac sympathetic denervation and ICDs, while novel, more targeted strategies, including gene-specific treatments, are also being developed [33].

WPW syndrome

Individuals are born with accessory atrioventricular conduction pathways. Most remain asymptomatic, but some display the WPW electrocardiographic pattern. A minority develop symptomatic WPW syndrome, with symptoms such as palpitations, presyncope, or dyspnea. WPW syndrome poses a risk of malignant tachyarrhythmias and SCD. Early identification, accurate diagnosis, and thorough risk assessment are crucial [34]. Although often symptom-free, life-threatening arrhythmias can be the first sign. Routine 12-lead ECGs in athletes can detect pre-excitation patterns in healthy individuals. Catheter ablation provides a definitive and safe treatment option, with a low complication rate [35].

CPVT

CPVT syndrome is characterized by bidirectional or polymorphic ventricular tachyarrhythmias triggered by exercise or emotional stress. The condition is mainly associated with pathogenic variants affecting the cardiac ryanodine receptor. Symptoms, often presenting as syncope, typically occur between 7 and 12 years of age, although late-onset cases in adulthood have also been reported. Without treatment, CPVT carries a high risk of death: about 30% of affected individuals experience at least one cardiac arrest, and up to 80% have one or more fainting episodes, with SCD possibly being the first sign. Recent studies demonstrate that nonselective beta blockers are the most effective treatment for CPVT and are more effective than selective beta blockers. Despite this, a substantial risk of life-threatening arrhythmias persists following left cardiac sympathetic denervation. Implantable cardioverter defibrillators provide effective intervention when pharmacological therapy fails to adequately control arrhythmias. Advances in treatment options over recent years have lowered the rate of arrhythmic events, and ongoing research is investigating additional therapies [36,37].

Cardiac acquired disorders

CAD

In older athletes, SCD is often caused by CAD. Accordingly, screening may be justified, and considering both traditional risk factors and additional CAD markers can help improve risk assessment [14]. However, it should not be presumed that an athlete is protected from CAD, despite optimal management of traditional cardiovascular risk factors. Furthermore, prolonged, high-intensity endurance exercise may contribute to the development of coronary atherosclerosis [38]. In recent years, advances in imaging technology have improved the accuracy of CCTA, allowing clinicians to detect not only significant coronary stenoses but also high-risk plaque characteristics, such as low-density fibro-lipid cores, positive arterial remodeling, and microcalcifications. As a result, among older athletes with higher cardiovascular risk or ambiguous stress test results, CCTA offers crucial anatomical insights that functional tests alone may not reveal [39]. For patients with CAD, revascularization therapy is required, which involves restoring normal blood flow in the coronary arteries. Available methods include coronary artery bypass grafting (CABG) or percutaneous coronary

intervention (PCI). Ultimately, the choice of method depends on the complexity of coronary anatomy, patient characteristics, and individual preferences [40].

Commotio cordis

Commotio cordis is defined as SCD precipitated by a blunt impact to the precordial region and is more prevalent in children and adolescents due to the pliability of the chest wall. Experimental evidence indicates that impacts occurring at the onset of the T-wave in the cardiac cycle can induce ventricular fibrillation and cardiac arrest [3]. Commotio cordis is one of the leading causes of SCD in young athletes, and the vast majority of cases occur during both organized and recreational sporting activities. Survival depends on immediate cardiopulmonary resuscitation (CPR) and defibrillation, which is why adequate medical coverage at sporting events, including trained personnel and readily available automated external defibrillators (AEDs), is essential [41]. Preventive strategies moving forward should focus on secondary measures, such as using protective padding during contact activities, because this condition is most commonly observed in young athletes engaged in contact or organized sports [42].

Drug-induced cardiotoxicity

Athletes are not more likely than non-athletes to engage in recreational drug abuse; however, they demonstrate a higher propensity to misuse substances classified as ergogenic aids. Consequently, the provision of accurate, evidence-based information is essential in athlete education, and sports medicine professionals are increasingly responsible for delivering this guidance [43]. In addition to enhancing performance and athletic results, these substances can cause numerous harmful effects on an athlete's health, adversely impacting the cardiovascular, hormonal, and metabolic systems. Anabolic-androgenic steroids (AAS) are associated with left ventricular hypertrophy, electrophysiological disturbances, and impaired myocardial function. Erythropoietin (EPO) elevates blood viscosity, thereby increasing the risk of thromboembolic events and heart failure. Stimulants can induce hypertension and arrhythmias. Chronic misuse of growth hormones are linked to cardiac hypertrophy and metabolic dysregulation, including insulin resistance. Collectively, these alterations substantially elevate the risk of SCD, heart failure, and other severe cardiovascular complications. Athletes require professional support

for safe training, with healthcare professionals helping to prevent misuse, provide education, interpret test results, and guide research on ergogenic agents [44].

Myocarditis

Acute myocarditis is an inflammatory disease of the heart muscle. It is most often triggered by viral infections but can also result from autoimmune disorders, certain medications, and, rarely, vaccination. The most common symptom is chest pain, followed by shortness of breath and, less frequently, syncope. Diagnosis is based on clinical presentation, elevated cardiac biomarkers, ECG changes, and echocardiographic findings, while cardiac MRI or endomyocardial biopsy are needed for definitive confirmation [15]. In acute myocarditis, ventricular arrhythmias are caused by inflammation and ion channel dysfunction, leading to disrupted signaling, oedema, and fibrosis that impair conduction. In chronic myocarditis, they are mainly due to scar-related re-entry circuits. Treatment is mostly supportive and focuses on relieving symptoms [45].

Screening and prevention

In athletes, a comprehensive cardiac history and physical examination are fundamental. Detecting any abnormal findings or a positive family history of SCD before the age of 35 should immediately prompt a cardiology referral for further evaluation. Pre-participation cardiovascular screening, together with timely specialist referral, remains the most effective strategy for the early identification of asymptomatic individuals at risk. Screening in young competitive athletes enhances detection of cardiovascular disease linked to SCD, with repeat evaluations improving diagnostic yield and early identification supporting favorable long-term outcomes [46]. Both cardiac and non-specific symptoms are observed in approximately 74% of individuals prior to sport-related SCD (Sr-SCD), with syncope, chest discomfort, palpitations, and dizziness being the most frequent. Recognition of these clinical manifestations, combined with non-invasive predictive models, may facilitate the identification and management of athletes at risk and contribute to Sr-SCD prevention [47]. During pre-participation cardiovascular screening, the physician should primarily inquire about symptoms that occur during physical exertion, including chest pain or pressure, syncope or near-syncope, excessive shortness of breath, disproportionate fatigue, and palpitations or an irregular heartbeat. It is also

essential to determine whether the individual has previously been diagnosed with heart disease, hypertension, a heart murmur, or lipid disorders, as well as whether they have ever been restricted from sports participation for medical reasons or undergone prior cardiac testing. Equally important is the family history, particularly regarding premature SCD before the age of 50 and the presence of cardiomyopathies, clinically significant arrhythmias, LQTS, or Marfan syndrome in close relatives. The evaluation should be complemented by a focused physical examination, including cardiac auscultation, blood pressure measurement, and assessment for phenotypic features suggestive of inherited cardiovascular disorders [48]. Pre-participation screening should also include an ECG, echocardiography, and CMR, with hematologic and biochemical tests as indicated. Intense exercise can induce physiological cardiac changes, necessitating differentiation from pathology. Inherited cardiac disorders may be detected through genetic testing. Interventions include lifestyle modification, arrhythmia management, ICD implantation, and revascularization procedures. Programs should enable safe participation in sports while promoting awareness of warning signs [49]. Effective prevention and management also rely on implementing an emergency action plan (EAP), which should include staff training, rapid AED access, prompt activation of emergency medical services, and transfer to advanced care. Early CPR and defibrillation significantly reduce mortality, making regular training and preparedness essential. A widely publicized case of sudden cardiac arrest occurred during a Euro 2020 football match, when a player collapsed on the field. Immediate CPR and use of an AED enabled return of spontaneous circulation (ROSC). This event highlighted the critical importance of EAPs, including rapid recognition, early CPR, prompt defibrillation, and efficient activation of emergency medical services, all of which are essential for improving survival in sudden cardiac arrest [6].

Diagnostic modalities in SCD

A meta-analysis in young athletes showed that modern ECG criteria greatly improve detection of cardiac disease compared with history and physical exam alone. Pre-participation 12-lead ECG significantly increased detection of cardiac disease (OR≈60, $p<0.001$) and SCD-related conditions (OR≈159, $p<0.001$), while history and exam had low, non-significant yield [50].

Holter monitoring is used in athletes with abnormal ECG findings or symptoms to detect intermittent arrhythmias and quantify arrhythmic burden over time, particularly premature ventricular complexes (PVCs), which may be significant in conditions such as ARVC.

Exercise stress testing is used to provoke exercise-induced arrhythmias that may not be present at rest, while echocardiography provides assessment of cardiac structure and function.

In suspected cardiomyopathy, cardiac MRI helps distinguish physiological athlete's heart from pathological hypertrophy. It provides detailed tissue characterization and can detect myocardial fibrosis (late gadolinium enhancement), fatty infiltration in ARVC, and other structural abnormalities not seen on echocardiography. It is particularly useful for diagnosing cardiomyopathies and improving arrhythmic risk stratification.

Additional investigations, including coronary angiography (diagnosis of CAD and ARVD) and electrophysiological studies (arrhythmias), are reserved for cases where diagnosis remains uncertain or further clarification of arrhythmic substrate is required [51].

Recent advances have made genetic testing widely available in clinical practice. It is mainly used in patients with confirmed diagnosis to enable cascade screening of first-degree relatives. Next-generation sequencing enables broad, cost-effective panels for cardiomyopathies and channelopathies, increasing their use in athletes with borderline features. In selected cases, pathogenic variants in genes encoding desmosomal proteins or lamin A/C, provide additional prognostic information on disease progression and SCD risk [3]. Major clinical warning signs of cardiac disorders associated with SCD are shown in Figure 1.

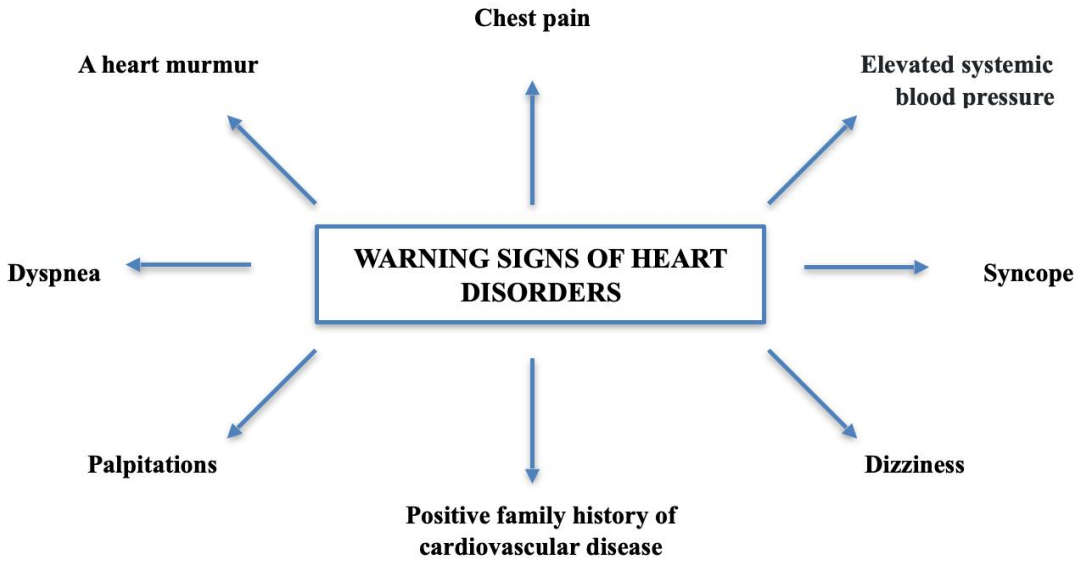

Figure 1. Major clinical warning signs of cardiac disorders associated with SCD [47,48]

ESC recommendations for sports participation in patients at risk of SCD

In HCM, 5-year SCD risk is assessed using the ESC HCM Risk-SCD model, combining clinical, echocardiographic, and Holter data. Key risk markers include prior cardiac arrest, syncope, symptoms, abnormal blood pressure response to exercise, and exercise-induced ventricular arrhythmias; their presence generally contraindicates high-intensity sports, while low to moderate recreational activity may be allowed in stable cases.

In ARVC/ACM, high-intensity and endurance exercise promotes disease progression and arrhythmias; thus, significant activity restriction is recommended, and high-intensity sports are contraindicated even in genotype-positive, phenotype-negative individuals. In patients with ICDs, exercise is possible but must be individualized due to risks such as lead dislodgement and device malfunction, especially in contact or high-risk sports, with return to activity considered after recovery and reassessment.

Management of patients with arrhythmic disorders and channelopathies should be individualized and incorporate education, shared decision-making, and structured recommendations regarding sports participation [52,53].

Future directions

The implementation of a standardized program aimed at preventing SCD in children and adolescents constitutes a substantial challenge owing to the intricate nature of the issue. Insights derived from previous initiatives suggest that effective collaboration among diverse stakeholders facilitates the acquisition of consistent data and the development of cardiac screening systems. Such coordinated efforts underpin the establishment of standardized cardiac screening protocols, which are reinforced by consensus guidelines and the engagement of key stakeholders across the medical, educational, and administrative sectors. Future strategies should concentrate on standardized, easily interpretable screening tests that enable the early identification of individuals at risk of SCD [54]. The medical community also faces the challenge of enhancing SCD prediction and prevention, including through AI-assisted algorithms and the integration of data from electronic health records (EHRs), ECGs, and digital devices [55]. Several AI-based ECG and wearable monitoring systems have obtained Food and Drug Administration (FDA) clearance for cardiac rhythm classification, while others aimed at

detecting structural and functional cardiac diseases, such as cardiac amyloidosis, pulmonary hypertension, and left ventricular dysfunction, have received breakthrough device designation. Further clinical implementation will require robust validation, regulatory oversight, and appropriate integration into clinical workflows to mitigate the risk of false-positive results and information overload. In addition, evidence demonstrating improved diagnostic efficiency and patient outcomes is necessary prior to widespread clinical adoption [56]. Additionally, advances in genetic testing have markedly improved the ability to detect pathogenic mutations and disease-modifying variants associated with SCD, thereby enabling the early identification of individuals and families at increased risk. Moving forward, the integration of comprehensive genetic testing with post-mortem evaluations may further augment diagnostic accuracy and elucidate the underlying causes of SCD in young individuals. The systematic collection of clinical and genetic data through national and regional SCD registries would facilitate the evaluation of preventive strategies and support research efforts aimed at reducing SCD-related mortality [57,58].

Conclusions

SCD in athletes is a rare but devastating event with serious medical and societal consequences. Although the overall incidence is low, the impact justifies ongoing prevention efforts, including thorough pre-participation cardiovascular screening and continuous education for athletes, coaches, and medical staff. Early identification of congenital or acquired cardiac conditions is crucial, as many athletes show no symptoms until they engage in intense physical activity. Improving screening methods and raising awareness are vital to lowering the risk of SCD in athletes.

The field of cardiology is rapidly evolving, with ongoing advances in diagnostics, treatments, and preventive strategies for conditions that increase risk. In particular, modern cardiac imaging techniques, such as advanced echocardiography and CMR, together with genetic testing, enable earlier detection of structural and inherited abnormalities, more precise risk stratification, and targeted clinical management. These innovations make it increasingly possible to identify at-risk individuals before the onset of symptoms and implement appropriate interventions in a timely manner. It is also important to emphasize the role of immediate response strategies: rapid access to AED and prompt initiation of cardiopulmonary resuscitation are critical determinants of survival in cases of cardiac arrest during sports activities. With

proper medical management, regular follow-up, and personalized treatment plans, many athletes with identified conditions can lead normal lives and often continue to participate safely in sports, while early recognition of even subtle symptoms remains essential for effective prevention.

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