Clinical case

Sudden cardiac death in regularly training young athletes: risk factors and prevention

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Abstract: Sudden cardiac death (SCD) in young athletes engaged in regular intensive training represents a rare but highly significant clinical and social problem. Although the incidence is low, SCD typically occurs unexpectedly during physical exertion or shortly thereafter, often as the first manifestation of previously undiagnosed cardiovascular disease. The main etiological factors include hypertrophic cardiomyopathy, arrhythmogenic right ventricular cardiomyopathy, myocarditis, dilated cardiomyopathy, and primary electrical disorders such as long QT syndrome. Risk stratification is based on a comprehensive approach that involves clinical history, physical examination, resting and exercise electrocardiography (ECG), echocardiography, and, in selected cases, genetic testing. Recent consensus statements highlight the importance of improving ECG interpretation criteria to reduce false-positive results in athletes. Preventive strategies include pre-participation cardiovascular screening, continuous follow-up of high-risk groups, and timely intervention in suspected cases.Equally important are emergency measures, including cardiopulmonary resuscitation and rapid use of automated external defibrillators, which significantly improve survival outcomes. Awareness of potential risk factors, combined with optimized screening protocols and personalized decision-making, may reduce the incidence of SCD in the athletic population.

Keyword: Sudden cardiac death, athletes, cardiovarcular risk, screening, channelopathies.

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Introduction

Although SCD among young individuals (under 35 years of age) engaged in regular sports activities is a relatively rare event, its unexpected occurrence makes it a highly significant phenomenon due to its social resonance, public debate, and the profound psychological impact on the victim's relatives. SCD is defined as an unexpected death resulting from cardiac arrest that occurs within one hour of the onset of symptoms [1,22]. In such cases, the underlying heart disease may or may not have been previously recognized, yet the fatal event is always unanticipated.

In most international publications, athletes younger than 35 years are typically referred to as "young athletes." In this population, SCD often represents the first clinical manifestation of previously undiagnosed, life-threatening cardiovascular disorders, particularly when occurring during intense physical activity. Retrospective analyses indicate that approximately 29% of SCD cases in this group reveal identifiable structural or electrical cardiac abnormalities [2]. The final diagnosis is usually established through postmortem investigations, including pathological, histological, and molecular (genetic) autopsy techniques.

Epidemiology

The incidence of SCD in young individuals engaged in intensive sports activity is estimated at 0.3–3.6 cases per 100,000 persons, with male athletes being affected 13 times more often than their female counterparts [3,29]. Among young individuals who are physically active in competitive sports, the risk of SCD is 2.5 times higher compared to non-athletes [4,9]. Approximately 56–80% of cases

occur during physical activity, while nearly 46% are classified as sudden arrhythmic death syndrome (SADS). The term SADS is applied when pathological and toxicological examinations fail to identify a clear cause of death. Sports that place a particularly high hemodynamic load on the cardiovascular system are more frequently associated with SCD or sudden cardiac arrest (SCA). Epidemiological observations indicate that football and basketball carry a higher frequency of SCD compared to other sports. Geographic variations are also evident: in France, Sweden, Spain, and Luxembourg, cycling accounts for most SCD cases; in the United States, basketball; in Japan, baseball; in Italy and Germany, football; and in Australia, running [5].

From an ethnic perspective, hypertrophic cardiomyopathy (HCM) is more prevalent among Black athletes than in other racial groups [9,24]. Similarly, Brugada syndrome is reported more frequently among athletes in Asia, particularly in Southeast Asia [23]. The highest rates of SCD are seen in the 25–30 age group [4,5,7].

Although the majority of SCDs in athletes occur during exercise, cases are also reported during rest or sleep. A recent study from the United Kingdom showed that 61% of athletes experienced SCD during training, including 4% during fights or physical altercations. Among those who died at rest, one-third experienced SCD during sleep [6].

Etiology

The causes of SCD in young athletes can be categorized into five major groups (see Table 1): cardiomyopathies (HCM, arrhythmogenic right ventricular cardiomyopathy (ARVC), left ventricular non-compaction, and dilated cardiomyopathy (DCM)); primary electrical disorders (channelopathies); valvular diseases; coronary artery anomalies; and aortopathies.

A retrospective forensic study in Spain analyzed epidemiological data of 645 SCD victims aged 1–35 years between 2010 and 2017. Myocardial disease was diagnosed in 33 patients, with ARVC being the leading cause (37%), followed by HCM (24%) and myocarditis (15%). Only five patients had a prior diagnosis of cardiomyopathy. Notably, 85% of cases occurred during recreational sports activity [8]. Although ischemic heart disease is rarely a cause of SCD in this age group, it becomes one of the most common etiologies after the age of 35.

HCM is the most frequent inherited heart disease, occurring in 0.2% of the general population. It is characterized by left ventricular hypertrophy (LVH) in the absence of abnormal hemodynamic load. Electrocardiography (ECG) plays a crucial role in diagnosis, revealing abnormalities in over 90% of cases. Echocardiography typically demonstrates LVH, and in two-thirds of patients, left ventricular outflow tract obstruction is observed either at rest or during exercise. HCM accounts for 2–36% of SCD in athletes and has been reported as the leading cause of SCD among young athletes in several U.S. studies.

ARVC is another inherited disorder, defined by progressive replacement of myocardial tissue with fibrous-fatty infiltration, predisposing to fatal arrhythmias. The condition is most commonly associated with mutations in genes encoding cardiac desmosomal proteins and has an estimated prevalence of 1:1000. Research has shown that ARVC is a major cause of SCD in athletes, particularly in the Veneto region of Northern Italy. Geographical differences have been noted: in the U.S., HCM predominates, whereas in Italy, ARVC is more frequent. These findings may also be influenced by differences in screening protocols. For instance, since ECG abnormalities are present in over 90% of HCM cases, widespread ECG screening in Italian athletes likely contributes to higher detection rates.

DCM, defined by dilation and systolic dysfunction of the left or both ventricles in the absence of coronary artery disease sufficient to explain global systolic impairment, is a relatively rare cause of SCD, accounting for 1–3% of cases. Myocarditis is particularly significant among inflammatory causes, being responsible for 2–9% of SCD in young athletes, and diagnosis often relies on cardiac magnetic resonance imaging (MRI) [10].

Another important group of conditions without structural heart abnormalities are channelopathies, which contribute substantially to SCD cases. These disorders are caused by mutations in proteins responsible for sodium, potassium, or calcium ion channel function, leading to abnormal myocardial excitability and conduction. Recognized entities include Brugada syndrome, long QT syndrome, short QT syndrome, catecholaminergic polymorphic ventricular tachycardia (CPVT), and early repolarization syndrome.

Coronary artery anomalies are implicated in up to 17% of SCD cases in the United States. These include anomalous origin of the coronary arteries, interarterial courses, and myocardial bridges. In a retrospective analysis of 30 cases, anomalous origin of the left coronary artery from the right sinus of Valsalva was particularly associated with SCD during exertion (73% of cases occurred during exercise, compared to 18% for anomalous right coronary artery from the left sinus).

Although valvular diseases rarely cause SCD in this population, mitral valve prolapse is commonly observed among athletes and has been associated with arrhythmic events [3,10].

Finally, in recent years, concern has grown regarding the impact of doping substances on the risk of SCD among young athletes. However, reliable data are limited due to underreporting of illicit drug use in sports. Substances such as anabolic steroids, growth hormone, and erythropoietin may increase cardiovascular risk. In particular, under conditions of intense exertion, adverse environment, or acute psychological stress, these agents may predispose to life-threatening ventricular tachyarrhythmias [5].

Although the etiological factors leading to sudden death are diverse, the principal pathogenetic mechanism is the occurrence of life-threatening arrhythmias—most commonly ventricular tachycardia and asystole—which ultimately result in fatal outcomes.

Table 1. Major cardiovascular diseases causing sudden cardiac death in young athletes

Category	Main conditions
Myocardial/structural heart diseases	- HCM - ARVC - Familial/idiopathic DCM - Left ventricular noncompaction cardiomyopathy - Toxic cardiomyopathy (alcohol, anabolic steroids, etc.) - Acute and chronic myocarditis - Simple and complex congenital heart defects
Primary electrical/conduction system diseases	- Wolff–Parkinson–White (WPW) syndrome - Long QT syndrome - Short QT syndrome - CPVT - Idiopathic ventricular tachycardia - Brugada syndrome - Commotio cordis (sudden cardiac arrest due to chest impact)
Coronary circulation disorders	- Congenital anomalies of coronary artery origin and course - Coronary artery disease (CAD)
Valvular heart diseases	- Bicuspid aortic valve (moderate stenosis ± aortopathy) - Pulmonary valve stenosis (moderate stenosis) - Mitral valve prolapse (associated with arrhythmogenicity)
Aortic diseases	- Aortopathy associated with bicuspid aortic valve - Idiopathic aortopathy / thoracic aortic aneurysm - Marfan syndrome, Loeys–Dietz syndrome, Turner syndrome - Ehlers–Danlos syndrome type IV (vascular form)

Clinical Screening for the Prevention of Sudden Cardiac Death in Athletes

Initial stage screening: The primary goal of screening among young athletes is to detect latent cardiovascular diseases and assess the risk of SCD. However, due to the wide diversity of sports disciplines, varying environmental conditions, and differences in athletes' physical preparedness, it is impossible to develop a universally standardized screening protocol and risk-assessment strategy applicable to all athletes.

Screening must be sensitive enough to identify pathological conditions, while at the same time avoiding the unnecessary disqualification of low-risk athletes from sports participation. Moreover, screening should take into account population-specific risk factors that may differ among regions and ethnic groups. For this reason, opinions on athlete screening remain divided. Some authors argue that pre-competition screening is unnecessary, as SCD is a very rare event in young athletes and the overall risk is considerably lower compared with the general population. In contrast, many experts, influenced by the tragic and widely publicized cases of SCD in athletes, advocate for systematic screening programs.

According to research, among approximately 100,000 screened athletes, only about 300 may be identified as being at increased risk of SCD, and in some instances, none of these athletes may experience sudden death within a year. The actual rate of SCD among "high-risk" individuals is estimated at <0.33% (3.3 per 1,000 athletes), representing just 0.001% of the entire screened population [12]. Furthermore, not all athletes flagged as "at risk" are later confirmed to have a clinically significant arrhythmogenic disorder.

Screening programs are also highly costly. In the United States, it has been estimated that saving one life would require screening 33,000 athletes at a cost of approximately 10.5 million USD. In Italy, the cost for the same outcome is about 1.32 million USD. Thus, population-wide screening presents major logistical and financial challenges. To date, only two countries — Italy and Israel — have implemented state-supported nationwide athlete screening programs [3,11].

Currently, the American Heart Association (AHA) and the European Society of Cardiology (ESC) provide differing recommendations regarding athlete screening. The AHA recommends screening based only on medical history and physical examination, whereas the ESC additionally advocates the use of a standard 12-lead electrocardiogram (ECG). Meta-analyses show that ECG is approximately 5 times more sensitive than medical history alone, and 10 times more sensitive than physical examination alone, for detecting conditions associated with SCD [21].

The AHA has developed a 14-element questionnaire that focuses on warning signs of cardiovascular disease in personal history (e.g., chest pain, syncope, dyspnea), as well as family history of premature SCD or disability due to heart disease (see Table 2). While the AHA approach is less costly, its sensitivity for predicting SCD remains limited. Physical examination is also included, comprising bilateral blood pressure measurement, pulse assessment, and auscultation for heart murmurs [13,20].

Including ECG in screening increases the sensitivity of detecting cardiovascular diseases that may lead to SCD. Several guidelines and expert consensuses have been developed to assist physicians in correctly interpreting ECG findings in athletes, with the goal of maintaining sensitivity while improving specificity. However, not all cardiac diseases that predispose to SCD in athletes necessarily present with warning symptoms, even if an ECG is performed. Therefore, the importance of screening tests aimed at identifying occult cardiovascular diseases prior to sports participation has been widely debated.

Nevertheless, interpreting ECG results in athletes is often challenging. This is because physiological adaptations to exercise may cause certain changes that are not pathological, but can mistakenly be classified as abnormal by physicians with limited experience in sports cardiology. Over time, the refinement of ECG interpretation criteria has led to a reduction in false-positive results. The latest consensus has introduced the concept of the "borderline ECG," allowing for more accurate assessment (see figure 1) [5,11].

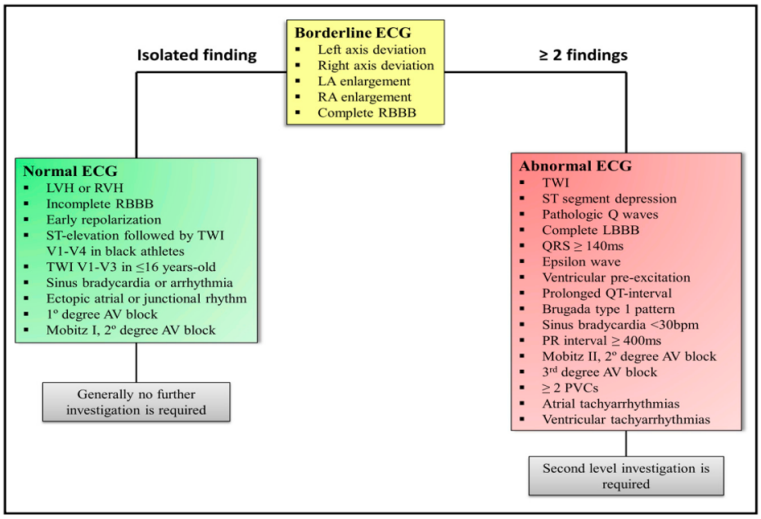

Figure 1. Algorithm for Interpreting ECG Changes in Athletes and Determining Subsequent Evaluation Steps

As noted above, the incidence and causes of SCD in athletes vary greatly depending on age, sex, ethnicity, country, and type of sport. The effectiveness of any screening test is directly determined by the characteristics of the target population. Therefore, it is unlikely that a single standardized screening program would be equally effective across all groups.

The ESC Working Group on Sports Cardiology, the Working Group on Cardiac Rehabilitation and Exercise Physiology, as well as the Working Group on Myocardial and Pericardial Diseases, recommend systematic cardiovascular screening for young athletes. Such screening should be carried

out at the initiation of sports participation, typically at the age of 12–14 years, and should be repeated at least every two years thereafter. This approach provides the opportunity to detect the development of certain conditions in a timely manner [25].

Table 2. AHA Questionnaire for Assessing the Probability of Sudden Cardiac Death in Athletes [20]

Item	Question	Clinical Significance
A. Personal Medical History		
1	Have you ever experienced chest pain, palpitations, or shortness of breath?	May indicate underlying cardiac ischemia or arrhythmia.
2	Have you experienced fatigue, dizziness, or severe weakness during exercise or at rest?	Suggestive of cardiovascular compromise or arrhythmogenic disorders.
3	Have you ever had fainting episodes?	Syncope can indicate life-threatening arrhythmias or structural heart disease.
4	Have you ever fainted during exercise?	Strong predictor of SCD risk in athletes.
5	Have you ever had palpitations, tachycardia, or episodes of arrhythmia?	May indicate primary arrhythmic disorders.
6	Do you experience excessive fatigue or reduced exercise capacity?	Can signal cardiomyopathy or other functional cardiac limitations.
7	Do you experience chest pain suggestive of angina?	May indicate ischemic heart disease.
B. Family History		
8	Has any family member experienced SCD before the age of 50?	Strong hereditary risk factor for SCD.
9	Is there a history of hypertrophic cardiomyopathy (HCM), Marfan syndrome, or other hereditary cardiovascular conditions in your family?	Genetic predisposition to structural heart disease.
10	Have there been cases of unexplained cardiac arrest or death in your family?	Suggests possible inherited arrhythmogenic or structural cardiac conditions.
C. Physical Examination		
11	Are there any cardiac murmurs?	May indicate valvular or structural heart disease.
12	Are femoral pulses diminished or absent (suggesting possible aortic coarctation)?	Detects congenital vascular abnormalities.
13	Are there clinical signs of cardiomegaly?	Indicates possible cardiomyopathy or chronic cardiac pathology.
14	Are there external physical features suggestive of Marfan syndrome?	Identifies risk for aortic disease and associated complications.

This approach is of critical importance for the management of athletes' cardiovascular systems, including pre-participation screening, clinical diagnosis, and risk stratification.

A study conducted in Italy provided the most reliable evidence regarding the effectiveness of ECG screening. It demonstrated that identifying athletes with high-risk cardiac conditions and excluding them from competitions could save lives. In the Veneto region, the annual incidence of SCD among athletes decreased from 3.6 cases per 100,000 person-years before the implementation of screening to 0.4 cases afterward, representing an 89% reduction [5].

However, it should be noted that in Israel, screening conducted with ECG and exercise testing did not reduce the incidence of SCD among athletes.

Second- and Third-Stage Examinations

If the initial screening reveals positive findings suggestive of SCD risk factors, the athlete should undergo additional invasive and/or non-invasive investigations. These may include Holter monitoring, echocardiography, exercise testing, cardiac MRI, coronary CT angiography (CCTA), invasive angiography, electrophysiological studies, electroanatomic mapping, and endomyocardial biopsy. In some cases, these results may be necessary to determine whether the athlete can safely participate in competitive sports [11,13].

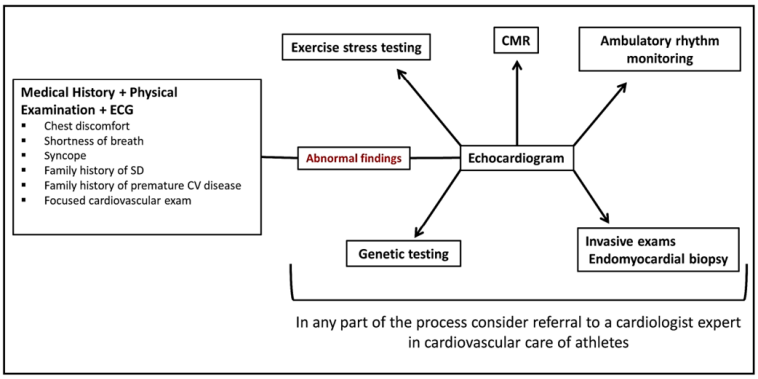

Figure 2. Pre-Competition Cardiovascular Screening Algorithm for Athletes

Holter Monitoring — Useful for patients with recurrent or reproducible symptoms such as palpitations or syncope. In athletes with infrequent symptoms, continuous loop recorders are used. These devices continuously record 1–3 minutes of ECG and save several minutes preceding an event when a button is pressed.

Exercise Testing — Should be tailored to the type of sport that triggers arrhythmias in the athlete, as standard exercise tests may not always reproduce clinical conditions. Arrhythmias may appear at the start of exercise, disappear at peak exertion, and reappear during recovery, often indicating benign extrasystoles. However, if arrhythmias increase with exercise, it may indicate cardiomyopathy or channelopathy, raising the risk of dangerous arrhythmias during sports.

Echocardiography (ECHO) — Often the first step for morphological assessment. However, in some cases, such as channelopathies, ECHO findings may be normal.

Cardiac MRI — Over the past decade, it has become a key non-invasive method for detailed myocardial evaluation. It can detect hypertrophy not visible on ECHO, especially localized thickening of the left ventricular wall. Gadolinium-enhanced MRI is considered the gold standard for diagnosing myocarditis and arrhythmias caused by fibrosis. In a study by Andreini et al. (946 patients), CMR identified structural cardiac pathology in 25.5% of cases and non-specific changes in 19.7%, even when ECHO was normal. Myocarditis was the most frequently identified cardiomyopathy [15].

Contrast CT and Coronary Catheterization — Necessary for diagnosing congenital coronary anomalies or atherosclerotic disease.

Electroanatomic Mapping, Endomyocardial Biopsy, and Transcatheter Ablation. If myocardial pathology is suspected in second-stage examinations, electrophysiological studies and electroanatomic mapping can be employed. New imaging technologies allow better characterization of arrhythmogenic substrates, e.g., high-density mapping can localize ectopic impulse sources.

Endomyocardial biopsy (EMB) is necessary in certain cases because some myocardial diseases can only be diagnosed this way, and their treatment strategies differ. However, due to technical limitations, this method is currently not applied in Uzbekistan. In a study by Narducchi et al., 33 young patients (18 athletes, 15 non-athletes) were examined. Even when ECHO and CMR were normal, EMB enabled a definitive diagnosis in 50% of athletes. Diagnoses included myocarditis (2), arrhythmogenic right ventricular cardiomyopathy (1), and localized fibrosis (1) [17].

Genetic Testing

When no specific disease is identified through examinations, genetic testing is recommended. Genetic analyses help uncover molecular mechanisms of SCD, identify new genetic markers, improve diagnosis and prognosis, and develop preventive and therapeutic strategies. Ventricular arrhythmias leading to SCD are often influenced by multiple genetic mutations. Genetic testing methodology is also referred to as “molecular autopsy.” In athletes who experienced SCD without identifiable pathological or histological cause, genetic testing is advised for relatives to detect primary cardiac electrical disorders [26,27].

However, routine genetic testing is not conducted in all cases due to low yield or the discovery of variants of uncertain or unknown clinical significance, particularly in individuals without suspicious phenotypes or a positive proband [28,31].

Athletes and Cardiac Pathology

The detection of a cardiac disorder in active athletes does not automatically necessitate a complete cessation of physical activity; however, it requires careful ongoing monitoring and management. In cases of sudden cardiac arrest (SCA), the prompt initiation of cardiopulmonary resuscitation and the timely use of an automated external defibrillator can be decisive for survival. Even a short delay of a few minutes following syncope can reduce the likelihood of successful rhythm restoration by 7–10% [16]. Equally important is the skill level and preparedness of the first responder. In many developed countries, sports facilities—particularly coaches—are trained through specialized courses to provide immediate life-saving assistance. Athletes who survive an SCA are generally recommended to receive an implantable cardioverter-defibrillator as a secondary preventive measure to minimize the risk of recurrence [1,14,18].

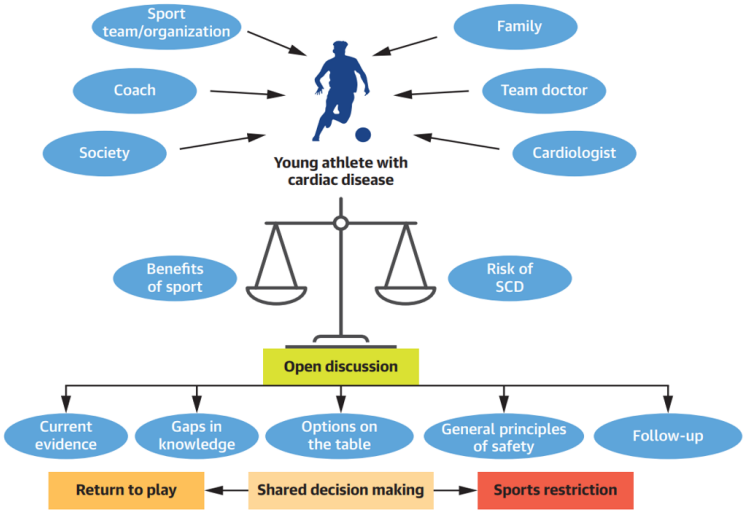

Figure 3. Decision-Making Algorithm for Athletes with Cardiovascular Disease

Recent evidence indicates a trend toward relaxing restrictions on intensive exercise for athletes diagnosed with cardiac conditions. For example, patients with hypertrophic cardiomyopathy (HCM) may now be permitted to participate in higher levels of physical activity, provided certain risk factors are absent. Conversely, athletes with dilated cardiomyopathy (DCM) who carry an LMNA mutation are strongly advised to avoid high-intensity sports, reflecting a Class III, Level C recommendation [19].

Older, asymptomatic HCM patients without established SCD risk factors may, in selected circumstances, engage in higher-intensity physical activity (Class IIb, Level C) [19].

In athletes with long QT syndrome, intense physical activity can precipitate arrhythmogenic syncope or sudden cardiac arrest, particularly if the corrected QT interval (QTc) exceeds 500 ms; in such scenarios, exercise is generally contraindicated [30]. However, individuals with a mild, asymptomatic phenotype, controlled under beta-blocker therapy, may be considered for activity on a case-by-case basis following expert evaluation. Similarly, athletes with positive genetic markers but without clinical manifestations should still limit high-intensity exertion. Decisions regarding participation must integrate multiple factors: the individual's risk of SCD, the benefits of physical activity, and the athlete's personal goals and preferences. Dynamic monitoring may also be warranted to ensure ongoing safety (Figure 3).

Conclusions

While SCD remains relatively uncommon among athletes, the increasing number of participants in competitive sports elevates the significance of this issue. The types of underlying cardiac conditions leading to SCD differ across populations, necessitating sport-specific and resource-appropriate screening strategies. In general, a combination of clinical examination and ECG-based screening is often sufficient, as demonstrated by the Italian experience, which led to a substantial reduction in SCD incidents. Recent advances in imaging technologies, such as cardiac magnetic resonance (CMR) and EMB, have further enhanced the ability to establish accurate diagnoses in athletes. For individuals with identified cardiac system disorders, collaborative, individualized decision-making is critical to

determine whether continued participation in physical activity is safe or whether restrictions should be implemented.

Authors' contribution.

Conceptualization, R.K. and N.Z.; formal analysis, E.T.; investigation, E.T.; resources, N.Z and A.S.; data curation, E.T.; original draft writing, E.T.; writing—review and editing, N.Z.; visualization,E.T.; supervision, R.K.; project administration, R.K. All authors have read and agreed tothe published version of the manuscript.

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Ethics approval.

The study was conducted in accordance with the Declaration of Helsinki and was approved by the Republican Specialized Scientific-Practical Medical Center of Cardiology (RSSPMCC).

Data Availability Statement

Statement All data sources used are listed in the references section.

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Conflict of interest

The authors declare that they have no conflict of interest.

Abbreviations

AHA	American Heart Association
ARVC	Arrhythmogenic right ventricular cardiomiopathy
CAD	Coronary artery disease
CMR	Cardiac magnetic resonance
CCTA	coronary CT angiography
CPVT	Catecholaminergic polymorphic ventricular tachycardia
CVD	cardiovascular disease
DCM	Lylated cardiomiopathy
ECG	Electrocardiography
ECHO	Echocardiography
EMB	Endomyocardial biopsy
ESC	European Society of Cardiology
HCM	Hypertrophic cardiomiopathy
LVH	Left ventricular hypertrophy
MRI	Magnrtic resonance tomography
SCD	Sudden cardiac death
SADS	sudden arrhythmic death syndrome
USD	United states dollar
WPW	Wolff–Parkinson–White

References

[1] Maron B.J., Udelson J.E., Bonow R.O., Nishimura R.A., Ackerman M.J., Estes N.A.M., Gersh B.J. (2025). Clinical considerations for competitive sports participation for athletes with cardiovascular abnormalities: A scientific statement from the American Heart Association. Circulation, 151(6), e256–e280. https://doi.org/10.1161/CIR.0000000000001297.

[2] Finocchiaro G, Papadakis M, Robertus JL, et al. Etiology of sudden death in sports insights from a United Kingdom regional registry. J Am Coll Cardiol 2016; 67:2108-15.

[3] Finocchiaro G, Westaby J, Sheppard MN, Papadakis M, Sharma S. Sudden Cardiac Death in Young Athletes: JACC State-of-the-Art Review. J Am Coll Cardiol. 2024 Jan 16;83(2):350-370. https://doi.org/10.1016/j.jacc.2023.10.032.PMID:38199713.

[4] Skeete J, Nair G, Huang HD, Volgman AS, Tracy M. Preventing sudden cardiac death in young athletes - A perspective from the United States. Am Heart J Plus. 2021 Oct 7;11:100054. https://doi.org/10.1016/j.ahjo.2021.100054.PMID:38549739; PMCID: PMC10978109.

[5] Borjesson M, Pelliccia A. Incidence and aetiology of sudden cardiac death in young athletes: an international perspective. Br J Sports Med. 2009 Sep;43(9):644-8. https://doi.org/10.1136/bjsm.2008.054718. PMID: 19734497.

[6] Harmon KG, Drezner JA, Wilson MG, Sharma S. Incidence of sudden cardiac death in athletes: a state-of-the-art review. Heart. 2014 Aug;100(16):1227-34. https://doi.org/10.1136/heartjnl-2014-093872.rep. PMID: 25049314.

[7] Corrado D, Zorzi A. Declining Risk of Sudden Cardiac Death in Young Athletes. Circulation. 2024 Jan 9;149(2):91-94. https://doi.org/10.1161/CIRCULATIONAHA.123.067243. Epub 2023 Nov 13. PMID: 37955558.

[8] Bayés de Luna A, Massó van-Roessel A, Escobar-Robledo LA, Arimany-Manso J. Update of sudden cardiac death: Epidemiology and risk stratification. Rev Esp Med Legal. 2018;44(1):5–12. https://doi.org/10.1016/j.remle.2017.10.002.

[9] Petek BJ, Churchill TW, Moulson N, Kliethermes SA, Baggish AL, Drezner JA, Patel MR, Ackerman MJ, Kucera KL, Siebert DM, Salerno L, Zigman Suchsland M, Asif IM, Maleszewski JJ, Harmon KG. Sudden Cardiac Death in National Collegiate Athletic Association Athletes: A 20-Year Study. Circulation. 2024 Jan 9;149(2):80-90. https://doi.org/10.1161/CIRCULATIONAHA.123.065908. Epub 2023 Nov 13. PMID: 37955565; PMCID: PMC10843024.

[10] Tsang DC, Link MS. Sudden Cardiac Death in Athletes. Tex Heart Inst J. 2021 Sep 1;48(4):e207513. https://doi.org/10.14503/THIJ-20-7513. PMID: 34669953; PMCID: PMC8717749

[11] Kochi AN, Vettor G, Dessanai MA, Pizzamiglio F, Tondo C. Sudden Cardiac Death in Athletes: From the Basics to the Practical Work-Up. Medicina (Kaunas). 2021 Feb 14;57(2):168. https://doi.org/10.3390/medicina57020168. PMID: 33673000; PMCID: PMC7918885.

[12] Fanous Y, Dorian P. The prevention and management of sudden cardiac arrest in athletes. CMAJ. 2019 Jul 15;191(28):E787-E791. https://doi.org/10.1503/cmaj.190166. PMID: 31308007; PMCID: PMC6629536

[13] Han J, Lalario A, Merro E, Sinagra G, Sharma S, Papadakis M, Finocchiaro G. Sudden Cardiac Death in Athletes: Facts and Fallacies. J Cardiovasc Dev Dis. 2023 Feb 5;10(2):68. https://doi.org/10.3390/jcdd10020068. PMID: 36826564; PMCID: PMC9965876.

[14] Agut-Busqueta A, Galtés I. Sudden cardiac death and sport. Review and key trends. Rev Esp Med Legal. 2018;44(4):158–168. https://doi.org/10.1016/j.reml.2017.12.004.

[15] Andreini D., Dello Russo A., Pontone G., Mushtaq S., Conte E., Perchinunno M., Guglielmo M., Coutinho Santos A., Magatelli M., Baggiano A., et al. CMR for Identifying the Substrate of Ventricular Arrhythmia in Patients With Normal Echocardiography. JACC Cardiovasc. Imaging 2020, 13, 410–421Egger F, Ukaj A, Hollander K. Sudden cardiac death in sports. Dtsch Z Sportmed.2023, 74: 14-18. https://doi.org/10.5960/dzsm.2022.550.

[16] Drezner J.A., Rao A.L., Heistand J., Bloomingdale M.K., Harmon K.G. Effectiveness of emergency response planning for sudden cardiac arrest in United States high schools with automated external defibrillators. Circulation 2009, 120, 518–525. [CrossRef]

[17] Narducci M.L., Pelargonio G., La Rosa G., Inzani F., d'Amati G., Novelli V., Marano R., Perna F., Bencardino G., Pinnacchio G., et al. Role of Extensive Diagnostic Workup in Young Athletes and Nonathletes with Complex Ventricular Arrhythmias. Heart Rhythm 2020, 17, 230–237.

[18] Böhm P, Meyer T, Narayanan K, Schindler M, Weizman O, Beganton F, Schmied C, Bougouin W, Barra S, Dumas F, Varenne O, Cariou A, et all. Sports-related sudden cardiac arrest in young adults. Europace. 2023 Feb 18;25(2):627–633. https://doi.org/10.1093/europace/euac172.

[19] Zeppenfeld K, Tfelt-Hansen J, de Riva M, et all ESC Scientific Document Group. 2022 ESC Guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death. Eur Heart J. 2022 Oct 21;43(40):3997-4126. https://doi.org/10.1093/eurheartj/ehac262.

[20] Petek BJ, Baggish AL. Pre-participation Cardiovascular Screening in Young Competitive Athletes. Curr Emerg Hosp Med Rep. 2020 Sep;8(3):77-89. https://doi.org/10.1007/s40138-020-00214-5. Epub 2020 May 21. PMID: 33552703; PMCID: PMC7863976.

[21] Maron BJ, Haas TS, Ahluwalia A, Murphy CJ, Garberich RF. Comparison of the sensitivity of history, physical examination, and electrocardiography for detecting cardiovascular abnormalities in athletes. J Am Coll Cardiol. 2014;64(14):1481–1488. https://doi.org/10.1016/j.jacc.2014.06.1173.

[22] American Heart Association/American College of Cardiology Foundation/ Heart Rhythm Society Scientific Statement on Noninvasive Risk Stratification Techniques for Identifying Patients at Risk for Sudden Cardiac Death. // Circulation. - 2008. - 118. - p. 1497-1518

[23] Vutthikraivit W, Rattanawong P. et all. Worldwide Prevalence of Brugada Syndrome: A Systematic Review and Meta-Analysis. Acta Cardiol Sin. 2018 May;34(3):267-277. https://doi.org/10.6515/ACS.201805_34(3).20180302B. Erratum in: Acta Cardiol Sin. 2019 Mar;35(2):192. PMID: 29844648; PMCID: PMC5968343.

[24] Gazdag P., Oravecz K., Acsai K. et al. Increased Ca2+ content of the sarcoplasmic reticulum provides arrhythmogenic trigger source in swimming-induced rat athlete's heart model. Sci Rep 10, 19596 (2020). https://https://doi.org/10.1038/s41598-020-76496-2.

[25] Maron B.J., Udelson J.E., Bonow R.O.et all. (2023). Sudden cardiac death in young athletes: JACC state-of-the-art review. Journal of the American College of Cardiology, 82(15), 1419–1433. https://doi.org/10.1016/j.jacc.2023.10.032.

[26] Fadoni J, Santos A, Amorim A, Cainé L. Sudden Cardiac Death: The Role of Molecular Autopsy with Next-Generation Sequencing. Diagnostics (Basel). 2025 Feb 13;15(4):460. https://doi.org/10.3390/diagnostics15040460. PMID: 40002611; PMCID: PMC11854515.

[27] Karen L. Kelly MDa, , Peter T. Linb , Cristina Bassoc et all Sudden cardiac death in the young: A consensus statement on recommended practices for cardiac examination by pathologists from the Society for Cardiovascular Pathology Cardiovascular Pathology 63 (2023) ISSN 1054-8807, https://doi.org/10.1016/j.carpath.2022.107497.

[28] Stiles MK, Wilde AAM, Abrams DJ, Ackerman MJ, et all 2020 APHRS/HRS expert consensus statement on the investigation of decedents with sudden unexplained death and patients with sudden cardiac arrest, and of their families. J Arrhythm. 2021 Apr 8;37(3):481-534. https://doi.org/10.1002/joa3.12449. PMID: 34141003; PMCID: PMC8207384.

[29] Weizman O, Empana J, Blom M. et al. Incidence of Cardiac Arrest During Sports Among Women in the European Union. JACC. 2023 Mar, 81 (11) 1021–1031. https://doi.org/10.1016/j.jacc.2023.01.015.

[30] Davies Ross A. et al.The 2023 Canadian Cardiovascular Society Clinical Practice Update on Management of the Patient With a Prolonged QT Interval Canadian Journal of Cardiology, Volume 39, Issue 10, 1285 - 1301

[31] Salzillo C, Sansone V, Napolitano F. Sudden Cardiac Death in the Young: State-of-the-Art Review in Molecular Autopsy. Current Issues in Molecular Biology. 2024; 46(4):3313-3327. https://doi.org/10.3390/cimb4604020.