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Sudden Cardiac Death As a Mystery of The Sports World - Current Insight Into Risk Factors, Diagnosis and Management of The Most Common Underlying Causes

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Abstract

Introduction and aim of the study: With the growing popularity of physical activity, sudden cardiac death has become a significant concern among both professional and recreational athletes. This study aims to review current knowledge on risk factors, diagnosis, and management of the most common causes of sudden cardiac arrest and sudden cardiac death in athletes, offering practical insights for healthcare professionals.

Materials and methods: This review is based on literature sourced from databases such as PubMed and Google Scholar. Keywords used included: “sudden cardiac death,” “sudden cardiac arrest,” “sudden cardiac death in sport,” and “sudden cardiac death in athletes.”
Results: Several risk factors for sudden cardiac death were identified, including age, sex, ethnicity, type and intensity of sport, and abnormalities on ECG screening. The causes of sudden cardiac death vary by age; in athletes under 35, the most frequent conditions include hypertrophic cardiomyopathy, arrhythmogenic right ventricular cardiomyopathy, long QT syndrome, and Brugada syndrome. These conditions were analyzed with respect to diagnosis, management, and potential complications.
Conclusion: Sudden cardiac arrest and sudden cardiac death are critical concerns in the athletic population. All discussed conditions are genetically determined, so a family history of unexplained sudden death or cardiogenic syncope should raise clinical suspicion. Early detection, proper risk assessment, and timely intervention can significantly reduce the risk of fatal outcomes. Increasing physician awareness of these conditions is essential to improve recognition and prevention among at-risk athletes.

Keywords: Sudden cardiac death, sudden cardiac arrest, sudden cardiac death in sport, sudden cardiac death in athletes

Introduction and aim of the study

Sudden cardiac death (SCD) is a term which can be defined as death caused by cardiovascular or unknown background approximately within one hour since the onset of symptoms. It is often preceded by sudden cardiac arrest (SCA) - people with SCA in their medical history have a vastly greater chance of experiencing SCD in future [1]. SCD can be observed in a broad spectrum of age groups - both in older and younger populations. The difference in these two groups lies in etiology of SCD - in older patients, the most common

cause is coronary arterial disease (CAD), whereas in younger patients main causes include genetically-determined cardiac arrhythmias and cardiomyopathies, myocarditis and coronary artery anomalies [1]. For some patients, SCA is a result of chronic, symptomatic disease, which was diagnosed many years prior to the incident. However, there are also cases where SCA (followed by possible SCD) was the first symptom indicating a possible underlying cardiac disorder. This type of situation is often observed in athletes participating in recreational or competitive sports. An increase in cardiovascular load and heart strain during physical activity can result in the collapse of hemodynamic stability of the athlete's body.

The incidence of SCD in the young population (defined as less than 35 years old) ranges in the literature approximately from 1 to 6 cases per 100,000 patient-years, whereas the incidence of SCD during sports participation for the general population is estimated at 0.46 per 100.000 person-years [2,3]. Interestingly, even though physical activity directly increases the risk of SCD, most cases of that phenomenon occur while the person is at rest [4]. There are many recognized and possible risk factors for SCA/SCD, which include sex, age, race, comorbidities, family history, type of performed sports and diagnostic findings - these can be categorized into two main groups of inherited and acquired risk factors [5,6]. A better understanding of possible risk factors could allow us to determine which individuals are in need of more strict follow-up and additional testing when competing in sport. Due to the dynamic progress in the availability of various imaging, electrophysiological or even genetic testing, we are nowadays more able to distinguish patients at risk [7], and with the usage of modern techniques and different treatment approaches, we can greatly decrease the possibility of SCA/SCD happening during sport performance.

The incidence of SCD connected to sport, which was mentioned above, may not seem significantly high, however, the rapidly increasing popularity of sport and the number of people deciding to perform one of many available disciplines result in the necessity of expanding the knowledge about SCA/SCD incidents in athletes. The primary aim of this review is to provide current insight into possible risk factors, diagnostic options and management strategies of the most common underlying causes of SCD connected with sport performance, based on the most recent studies available considering this topic.

Materials and methods

For this review, a comprehensive search of articles available in various scientific databases such as PubMed, Google Scholar and others was conducted. The search included terms such as: "Sudden cardiac death", "Sudden cardiac arrest", "Sudden cardiac death in sport", "Sudden cardiac death in athletes" and similar. The articles selected were limited to those published in English. They were then narrowed down to recent and relevant articles.

Results

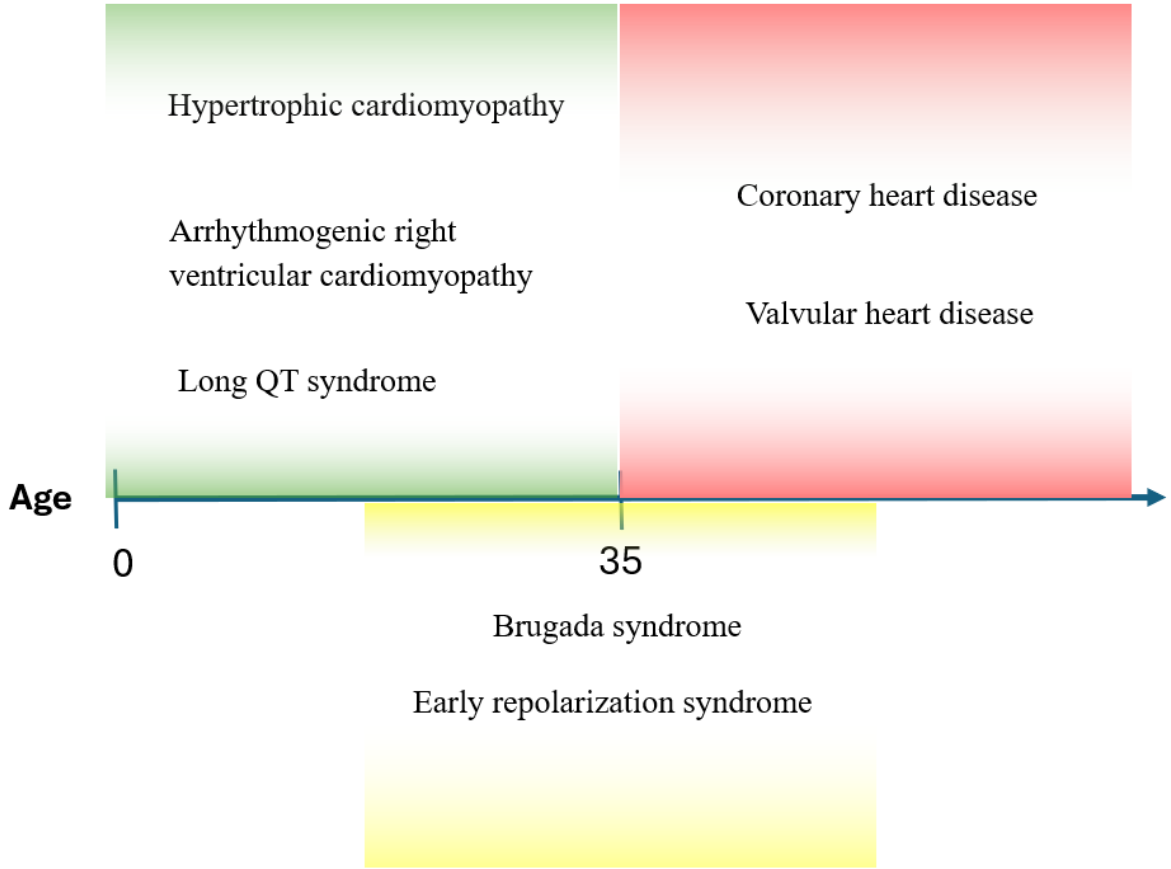
Risk factors

A number of risk factors connected with the potential future development of SCA/SCD have been reported in the available literature [8–16]. One of them is age - studies suggest that the incidence of sport-related SCA/SCD increases with age, both in recreational and competitive athletes [9]. SCD is also more frequently observed in male athletes - the difference in incidence rate between male and female individuals is 2.6 to 1.1 per 100.000 person-years. This trend is still up-to-date despite the still increasing total number of female athletes participating in a variety of different disciplines [10]. Researchers suggest potential background of this correlation, such as higher prevalence of myocardial fibrosis in males, protective influence of estrogens on cardiovascular system in females, higher tendency in males to develop atherosclerotic changes in coronary vessels and psychological factor of higher tendency in males of trying to reach their physical and endurance limits during sport participation [11,12]. SCD is also more common among black athletes compared to other ethnic groups [13,14]. The type of performed sport discipline is also reported as the potential risk factor - studies suggest that most cases of SCA/SCD are connected to soccer (mostly in Europe) [14] and basketball (mostly in America) [10] - this correlation can result both from the popularity and total number of players of these disciplines in corresponding continents and from their overall high intensity and endurance-dependency. The competitive players also have a higher chance of experiencing SCA/SCD compared to recreational ones [15]. Due to the wide accessibility of electrocardiogram (ECG) testing nowadays, different abnormalities may be observed among players undergoing periodic health check-ups. These abnormalities include prolonged QRS

complex or QT interval duration, fragmented QRS complex, pathologic Q waves, inverted T-waves or increased R-wave voltage - they can suggest an underlying heart condition and therefore be connected with a higher risk of developing SCA/SCD during sport performance [16].

Etiology

SCD and SCA present a wide range of possible underlying etiologies of these phenomena (see **Figure 1**). As mentioned above, underlying causes can include CAD, genetically-determined cardiac arrhythmias and cardiomyopathies, myocarditis, coronary artery anomalies and many more less frequent conditions. Etiology of SCD can be divided into numerous groups, such as coronary artery-related causes (mostly myocardial infarction, ischemia, coronary spasm or myocardial bridging), cardiomyopathies (mostly hypertrophic cardiomyopathy (HCM), arrhythmogenic right ventricular cardiomyopathy (ARVC) and myocarditis), primary arrhythmogenic diseases (mostly long QT syndrome (LQTS), Brugada syndrome, Wolf-Parkinson-White (WPW) syndrome and early repolarization syndrome (ERS)), valvular heart diseases and congenital heart diseases [1]. These diseases in certain conditions (such as intensive physical exercise), can disrupt correct hemodynamic and electrical heart activity and lead to ventricular arrhythmias or asystole, which are main direct causes of SCA and following death. The underlying etiology differs between age groups and the estimated cut-off point for this observation is 35 years of age. Patients above 35 years of age develop SCA/SCD mostly due to the underlying coronary arterial disease and valvular heart diseases, whereas patients between 35 years of age suffer from HCM, AVRC and LQTS [17]. Hayashi et al. [17] also suggested dividing SCD/SCA etiology into two groups - SCD in patient populations with present or absent structural heart disease, respectively with coronary arterial disease and cardiomyopathies in the former group, and arrhythmic disorders (LQTS, Brugada syndrome, ERS) in the latter. The justification for such division is the fact that in almost 5% of SCD/SCA cases the significant cardiac abnormality cannot be found upon clinical examination in SCA survivors and performed autopsy in patients after SCD. Two studies of autopsy series performed in young patients developing SCD [18,19] showed that the demonstrable structural heart



abnormality could not be found in approximately 29% of cases, however results of Corrado et al. research [20] showed that implementation of detailed histopathological testing after the autopsy noticeably improved detectability of underlying pathology (such as focal myocarditis, regional arrhythmogenic right ventricular cardiomyopathy and conduction system abnormalities). As we can see, the spectrum of SCA/SCD possible etiology varies greatly, however because the main aim of this paper is to discuss underlying causes of SCD/SCA in professional and recreational athletes, we will mostly focus on the conditions present in the younger population due to their proportional higher participation in such activities.

Figure 1. The spectrum of the most common SCA/SCD etiology in different age groups with the cut-off point of 35 years of age.

Hypertrophic Cardiomyopathy

Hypertrophic cardiomyopathy (HCM) is a genetically-determined heart condition that results in hypertrophy of the left ventricle, which cannot be explained with excessive strain and pre- and afterload of the left ventricle [21]. It is considered to be one of the most common inherited cardiovascular disorders.

Mutations in numerous genes responsible for coding sarcomere proteins (mostly MYH7 and MYBPC3 genes) result in pathological myocyte hypertrophy, myocyte displacement and

myocardial fibrosis [22]. HCM can also lead to various arrhythmias (such as atrial fibrillation or ventricular tachycardia), which can be present before the development of diastolic heart failure (HF) and complicate HF with time. These mutations are most commonly inherited in autosomal dominant trait, however current knowledge confirms that there are many possible inheritance ways with variable expressivity and incomplete mutation penetrance. Although sarcomere-coding genes play the main role in pathogenesis, in around 10 to 15% of HCM cases pathological variants were detected in genes independent from the proper sarcomere structure [23].

The most common symptoms in HCM patients include dyspnea, fatigue, syncope, chest pain, physical activity intolerance and SCA followed in some cases with SCD. The severity of experienced symptoms does not regularly correlate with the level of morphological changes in heart muscle, such as left ventricular outflow tract obstruction (LVOTO) or left ventricle hypertrophy. HCM is the most common underlying cause of SCD among young athletes - both recreational and professional ones. Diagnosing HCM in athletes can cause significant problems due to the fact that exercises performed regularly are often resulting in development of adaptive changes in heart muscle, to which the term “athlete's heart” is used. Changes in the athlete's heart and heart of HCM patients can be similar and misleading (see **Table 1)** [24]. Wall thickness in recreational athletes is most commonly in the normal range of slightly elevated to approximately 12mm, however, in some professional athletes these dimensions can reach up to 16mm. In patients with HCM wall thickness values oscillate around 20mm with rare extreme cases of 50mm. Significant issues can be observed in the minority of HCM cases where wall thickness is in the “grey zone” of 13 to 15mm. In such situations the pattern of hypertrophy can be helpful - in trained athletes wall hypertrophy mostly include anterior part of ventricular septum, however the rest of left ventricle muscle is similarly enlarged with a deviation of 1-2 mm, whereas in HCM other parts of left ventricle beside the anterior part of ventricular septum are heterogeneously and irregularly thickened. It is also observed that the thickness of left ventricle wall can decrease during deconditioning in athlete’s heart, which is unusual for pathological hypertrophy caused by HCM. Another variable helpful in the differentiation process between an athlete's heart and HCM is cavity dimension. End-diastolic cavity dimension of left ventricle is usually enlarged above 55mm in almost one-third of male trained sportsmen, whereas HCM patient are often presenting decreased left ventricular cavity

dimension of approximately less than 45mm due to the heart muscle hypertrophy - in HCM patients this dimension can also be elevated, but mostly during development of end-stage transformation of diastolic to systolic HF. Physiological left ventricle hypertrophy is also more common in male athletes – therefore the female athletes with borderline left ventricle wall thickness are more likely to be diagnosed with HCM. When diagnosing patients with potential HCM, their family history should also be taken into consideration - cases of unexplained early deaths during physical activity should be an important warning sign for the physician [24].

Treatment of HCM consists of two main strategies - symptoms management and prevention of SCD with the use of risk assessment [21]. The first-line treatment for symptomatic obstructive HCM is pharmacological therapy. One of the key drugs used in HCM are beta-blockers, which cause heart drop to decrease and, therefore, increase ventricular filling time and preload. The preferable beta-blockers are propranolol, metoprolol and bisoprolol due to the lack of vasodilator effect, which could worsen the LVOTO state [25]. Nondihydropyridine calcium channel blockers (NDHP-CCBs), such as verapamil and diltiazem, are used in patients with contraindications to beta-blockers, adverse effects caused by beta-blockers or inefficiency of therapy. Disopyramide (class 1a antiarrhythmic drug) causes negative inotropic effect, a decrease in myocardial contractility and LVOTO pressure gradient and can be added to beta-blockers or NDHP-CCBs, when more intense symptoms management is required [26]. There are numerous studies and clinical testing performed regarding a new modern drug group - cardiac myosin inhibitors, such as mavacanten. These drugs work by decreasing the number of myosin-actin cross-bridging interactions and therefore causing the significant reduction of resting and provoked LVOTO pressure gradients and alleviate patient's symptoms [27]. In case of infectivity of conservative approach, invasive treatment can be incorporated in HCM therapy. Invasive treatment causes the reduction of active cardiac muscle and is mostly achieved with alcohol septal ablation or surgical septal myectomy [25]. Aside from symptom management, the risk assessment and prevention of SCD is also vitally important - especially in the context of athletes with HCM. The risk can be assessed using the HCM Risk-SCD calculator, which gives us an approximate risk of sudden death in the next 5 years in patients above 16 years of age. This tool takes into account variables such as age, maximum wall thickness, LA dimensions, LVOT pressure gradient, positive familial history for unexplained SCD, non-persistent ventricular tachycardia and unexplained syncopes. Patients with the risk of <4% do not need to

have ICD, in 4-6% the device implantation may be considered and >6% it is necessary to consider ICD primary prevention therapy. The SCD risk should be evaluated at the initial diagnosis and every 1-2 years or whenever there is significant change in patient's clinical status [28]. Athletes with HCM are recommended to perform low-to-mild-intensity exercises regularly due to its beneficial impact on overall health - high-intensity and competitive sports should not be performed in patients with confirmed HCM. These types of activities can sometimes be considered in patients with genotype-positive/phenotype-negative status or in patients with asymptomatic low-risk individuals with morphologically mild hypertrophic cardiomyopathy. The implantation of ICD solely to allow patients to perform exercises with this level of intensity is not recommended [28].

	HCM heart	**Athlete's heart**
End-diastolic LV cavity dimension <45 mm	+	-
End-diastolic LV cavity dimension >55 mm	-	+
Atypical pattern of LV hypertrophy	+	-
Enlargement of LA	+	-
Athlete's female gender	+	-

LV thickness decrease during deconditioning	+	-
Family history of HCM	+	-

Table 1. Comparison of HCM and athlete's heart in terms of specific characterictics.

Arrhythmogenic Right Ventricular Cardiomyopathy

Another example of SCD etiology in athletes below 35 years of age is arrhythmogenic right ventricular cardiomyopathy (ARVC) - a genetically-determined heart condition in which the proper myocardium of the right ventricle is being replaced with fibrofatty tissue. As a result, the heart muscle is susceptible to the generation of various arrhythmias.

The condition is caused by the mutation in one of 8 determined genes, from which 5 genes is responsible for coding desmosomes (PKP2, DSP, DSG2, DSC2, and JUP) and 3 genes are non-desmosomal (TMEM43, DES, and PLN) [29]. The main clinical manifestations of ARVC include arrhythmogenic syncopes, spontaneous sustained ventricular tachycardia, SCA and SCD. Around 10% of ARVC cases are being diagnosed after experiencing resuscitated SCA. Studies suggest that ventricular fibrillation is more common in the early stage of disease, whereas sustained ventricular tachycardia develops later secondary to the formation of scar tissue [30]. Atrial arrhythmias (such as atrial fibrillation, atrial tachycardia and atrial flutter) are also present in the ARVC in approximately 20% of cases [31].

The diagnosis of this condition is mostly based on fulfilling major and minor Task Force Criteria (TFC) - definite diagnosis can be stated if the patient fulfilled 2 major criteria, 1 major and 2 minor criteria or 4 minor criteria from different groups. TFC are based on results of different tests and patients' medical history - cardiac imaging, electrophysiological testing, histopathological examination of myocardium, genetic testing and familial history. Cardiac imaging is performed with the usage of echocardiography and cardiac magnetic resonance

(CMR) - physicians are mostly looking for regional akinesia, dyskinesia or aneurysm, which are one of the major TFC. Electrophysiological examination of the heart includes 12-lead ECG and Holter-ECG. - Typical findings in ARVC include Epsilon wave in V1, V2 or V3 leads (one of the major TFC - it represents ventricular depolarization delay), T-wave inversions in V1-V3 or VT with LBBB configuration registered in Holter-ECG. Endomyocardial biopsy is preferably collected from the free wall of the right ventricle - microscopic changes include myocyte atrophy with fibrous and fatty tissue replacement within right ventricular myocardium. Despite the high sensitivity and specificity of endomyocardial biopsy and it being one of the major TFC, nowadays this test is not recommended to be routinely performed due to the increase of high-quality CMR availability and because of its invasive character it should be performed in case of diagnostic difficulties and uncertainty. The individuals with definite diagnosis of ARVC should undergo genetic testing - identification of specific mutation allows for cascade screening of first-degree relatives of the proband. Those with present mutation should be clinically assessed with ECG, Holter-ECG, echocardiography and CMR.

Similarly to HCM, the risk stratification in ARVC is also vital to determine the necessity for ICD implantation. The International Task Force created 4 groups of specific indications for ICD implantation - high-risk group (ICD is indicated), major-risk group (ICD should be considered), minor-risk group (ICD may be considered) and low-risk group (ICD is not indicated). For example, the high-risk group include indications such as SCA, sustained ventricular tachycardia and severe LV or RV dysfunction - those situations lead to the annual risk of approximately 3.8% of experiencing serious arrhythmic events [32]. Management of the ARVC is also divided into conservative and invasive treatment. The exercise restriction is one of the most crucial components of treatment, however studies are inconsistent regarding the specific recommendations for this restriction - mostly it is recommended that ARVC patients should limit their physical activity to less than 650 MET-hours per year (around 30 minutes of walking per day aside from routine daily activities) [33,34]. Pharmacotherapy with the usage of beta-blockers, sotalol, amiodarone or flecainide in ARVC patients is the topic of various studies and the results are sometimes conflicting when it comes to its efficacy. Invasive treatments include preventive ICD implantation, ablation of arrhythmias foci or cardiac transplantation in highly advanced ARVC cases.

Long QT Syndrome

In contrast to HCM and AVRC, long QT syndrome (LQTS) does not present with the visible structural changes in cardiac muscle. This condition is characterized by prolonged QT interval in the ECG, which is caused by congenital mutations in genes coding ion channels participating in repolarization of cardiac muscle. Although throughout the years around 17 genes were connected to LQTS, nowadays 7 genes are considered to have strong causality in this condition. Prolonged QT interval can lead to development of life-threatening arrhythmias, such as ventricular fibrillation or Torsade de Pointes [35].

Due to the high variability of QTc in different individuals the ECG testing alone is not enough for the diagnosis. The LQTS diagnostic criteria (also referred to as "Schwartz-score") were described to determine which patients are at high risk of having congenital LQTS. These criteria include different ECG findings, clinical history of syncope or congenital deafness and family history of relatives with diagnosed LQTS or unexplained SCD under 30 years of age in first-degree family members - the result of 3.5 and above points indicates the high probability of LQTS presence, which should be confirmed in genetic testing [36]. Standard 12-lead ECG should also be followed by Holter-ECG and exercise test-ECG. There are 3 main subgroups of LQTS - LQTS1 (KCNQ1 gene), LQTS2 (KCNH2 gene) and LQTS3 (SCN5A gene), which differ in ECG appearance, frequency, arrhythmias triggers and management. LQTS1 is the most common type of LQTS, affects mostly male patients in their childhood and the main trigger for arrhythmias is exercising (especially swimming). LQTS2 is the second most common type, affecting female patients in their puberty and arrhythmias are typically caused by sudden auditory stimuli (for example an alarm clock) or emotional stress. LQTS3 is the rarest of the 3 mentioned types and, similarly to LQTS2, also affects female patients in their puberty. Interestingly, arrhythmic incidents in LQTS3 are mostly triggered during rest or sleep [37]. In LQTS possible therapy can also be divided into conservative and invasive treatment. When it comes to sports participation in patients diagnosed with LQTS, moderate physical activity is recommended - studies show that patients who are diagnosed, stable and appropriately treated have a low risk of experiencing SCA/SCD during exercising [38]. The only exception is the patients with LQTS1 subgroup, for whom swimming is strongly contraindicated due to the high rate of SCA and drownings during this activity in this particular group of patients. LQTS

patients also need to be aware of the necessity of avoiding drugs usage, which can prolong QTc interval.

Pharmacological therapy is based on beta-blockers in almost all symptomatic patients. Non-selective beta-blockers (such as propranolol and nadolol) are preferred and they are most effective in LQTS1 patients, however, they are also widely used in LQTS2 and LQTS3 subgroups. It is suggested that propranolol has a higher potential for reduction in QTc interval, however, nadolol has shown greater risk reduction in the general LQTS population [39]. Drugs such as mexiletine and flecainide, which are blockers of the late sodium inward current, can be used in addition to beta-blockers (especially in LQTS3 patients). Invasive treatment in LQTS patients include ICD implantation (in secondary prevention after SCA or in primary prevention with QTc >500 ms despite pharmacological therapy) [40], atrial overdrive pacing (during acute ventricular arrhythmias) or left cardiac sympathetic denervation (in patients with intolerance of beta-blockers or ineffectiveness of previous treatment) [41].

	LQTS1	**LQTS2**	**LQTS3**
Gene	KCNQ1	KCNH2	SCN5A
Altered ion current	K+	K+	Na+

Frequency in LQTS patients	~ 35%	~ 30%	~ 10%
Triggers of arrhythmic events	Exercise (especially swimming)	Sudden arousal (auditory stimuli)	Rest/sleep
Period of first arrhythmic event	Childhood	Puberty	Puberty
Gender	Mostly male	Mostly female	Mostly female

Table 2. Comparison of three most common types of LQTS.

Brugada Syndrome

Another condition connected to SCA/SCD, which does not present with the visible structural changes in cardiac muscle, is Brugada Syndrome (BrS). It is characterized by typical ST-segment elevation with T-wave inversion in the right precordial leads (type 1 of BrS) or saddleback ST-segment configuration with variable ST-segment elevation (type 2 and 3 of BrS). It is caused by a mutation in SCN5A gene, which is responsible for coding alpha-subunit of sodium channels - it is the only gene in which mutation is considered disease-causing for BrS [42].

Diagnosis is based on criteria, which are collectively referred to as “Shanghai Score”. These criteria include ECG findings (typical type 1 or type 2/3 ECG changes), clinical history

(SCA, documented ventricular fibrillation or polymorphic ventricular tachycardia, syncopes or atrial arrhythmias), family history (first- or second-degree family members with definite BrS or cases of suspected SCD) and genetic testing. At the time of diagnosis approximately one-third of BrS patients are symptomatic. The most common first symptoms of BrS are syncope and SCA resulting from triggering of ventricular fibrillation. Monomorphic and stable ventricular tachycardia is also less frequently observed in BrS patients. The first symptoms typically affect patients between 30 and 50 years old, however first arrhythmic events can also appear in childhood (especially in female patients) [43]. When it comes to baseline ECG, only type 1 is considered diagnostic, whereas for type 2 or 3 it is recommended to perform provocation testing. This test includes administration of sodium channel blockers (SCB) drugs, which can temporarily reveal the type 1 ECG pattern, and therefore help to establish correct diagnosis - SCB used in this testing include ajmaline, flecainide (mostly in Europe) and procainamide (mostly in North America). Genetic testing is mostly recommended in patients with Type 1 ECG due to the possibility of performing family screening. Nowadays genetic testing involves only variants in SCN5A gene, because the significance of other potential BrS-causing genes (such as SCN10A) is disputed. It is vital to remember that the sole presence of pathogenic variants in SCN5A gene cannot be considered diagnostic for BrS without fulfilment of other criteria [44].

The management of this condition is also divided into conservative, pharmacologic and invasive treatment. Conservative treatment includes avoiding of factors that have the potential to induce type 1 ECG changes, such as different drugs (antiarrhythmic, psychotropic, anesthetic, antihistamines drugs), fever (which should be effectively and rapidly treated in BrS patients) or alcohol (due to the increase in the risk of syncope in BrS patients [45]). Pharmacologic treatment is based on quinidine due to its compound antiarrhythmic effect - it should be considered in patients who qualify for ICD implantation, but decline it; for additional support in ICD therapy [46]; for the management of atrial arrhythmias [47]. Usage of quinidine can result in development of side effects, especially gastrointestinal intolerance [48]. Another drug used in BrS is isoproterenol, which is mainly used intravenously during acute and malignant ventricular tachyarrhythmias [49]. ICD implantation is indicated in BrS patients after resuscitated SCA in secondary prevention. Primary prevention should be considered in BrS patients with a history of cardiogenic syncope. When it comes to choice between transvenous

and subcutaneous ICD, a few factors should be considered - transvenous ICD also has the ability of atrial pacing in patients with atrial arrhythmias, whereas subcutaneous ICD lowers the risk of intravenous infections, therefore it should be considered in young patients without atrial arrhythmias history [50].

Conclusion

Due to the increased popularity of different sports disciplines and a higher rate of young people participating in recreational and competitive sports, the knowledge about the causes of exercise-connected SCA/SCD in this age group is crucial for physicians. In this paper, current insight regarding risk factors, diagnosis and the therapeutic management of the four most common underlying causes of SCA/SCD in athletes below the age of 35 years was reviewed. Two of these conditions - HCM and ARVC - present with structural heart changes, whereas the other two - LQTS and BrS - are syndromes disrupting proper heart function without visible structural changes in the myocardium. All of the described conditions are caused by congenital mutations in defined genes, therefore, the medical history of unexplained SCD or cardiogenic syncopes in first-degree family members of a symptomatic patient should raise concerns of the physician and urge him to perform more profound diagnostic procedures when trying to exclude or confirm possible underlying etiology. The expanded diagnostic approach with the usage of different testing methods (such as electrophysiological, imaging, and genetic testing) is necessary to establish the correct diagnosis. Early diagnosis of athletes with the first manifestation (or even before first symptoms due to the positive family history) of these conditions can significantly speed up the potential implementation of proper management and therefore lower the risk of development of life-threatening complications. Risk stratification following the diagnosis is also a crucial step in treatment, which allows to decide whether individual patients can be managed with a conservative, pharmacologic or invasive approach. The awareness of potential underlying causes of SCA/SCD connected to the sport and the proper management of these conditions should be raised among physicians dealing with patients from all age groups to increase the chance of implementation of a proper strategy to minimize the risk of SCA/SCD from happening in athletes.

Disclosure

Author's contribution

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