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Clinical paper

Exercise related sudden cardiac death (SCD) in the young — Pre-mortal characterization of a Swedish nationwide cohort, showing a decline in SCD among athletes

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Abstract

Aims: To study the frequency, etiology, and premortal abnormalities in exercise-related sudden cardiac death (SCD) in the young in Sweden.

Methods: All subjects with SCD in 10–35-year olds in Sweden during 2000–10, were included (n = 514). Information about each case was retrieved from death certifications, autopsy- and medical records. The number of SCD in athletes was compared to national figures from 1992-99.

Results: Exercise-related SCD occurred in 12% (62/514) of the SCD-population, a majority being men (56/62; 90%). Cardiopulmonary resuscitation (CPR) was started in 87% (54/62). In total, 48% (30/62), had a cardiac diagnosis, symptoms, family history and/or ECG-changes, before the fatal event. The most prevalent autopsy diagnosis was sudden arrhythmic death syndrome (15/62; 24%). The frequency of hypertrophic cardiomyopathy (HCM) and arrhythmogenic right ventricular cardiomyopathy (ARVC) was significantly higher in exercise-related SCD compared to non-exertional SCD. Exercise-related SCD was more common in athletes (21/29) than in non-athletes (41/485) ($P < 0.0001$). The total number of SCDs/year in athletes 15–35 years old, are approximately halved in 2000-10 compared to the years 1992–99.

Conclusion: The increased risk of exercise-related SCD in HCM and ARVC underlines the importance of early detection and eligibility recommendations. There is a major reduction in deaths among athletes in the 2000s, compared to the previous decade. These results may partly be explained by improved acute preparedness for sudden cardiac arrest (CPR, defibrillation), but as a substantial percentage have preceding risk factors, such as symptoms and ECG-abnormalities, increased cardiac screening and increased general awareness, may also play a role.

Keywords: Sudden cardiac death, Young athletes, Exercise, Risk group, Incidence, Sweden, Autopsy

Introduction

Sudden cardiac death (SCD) is uncommon, but accounts for a majority of natural, unexpected sudden death in the young population. Exercise, although clearly health promoting for the vast majority of individuals, is a well known potential trigger of SCD.[1]

In the Veneto region, athletes in 1979–80 had a four-fold increased risk of SCD compared to non-athletes.[2] The incidence among athletes

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has then been decreasing in this region, generally attributed to the pre-participation screening of athletes, and in 2004 was lower, (0.4 per 100,000 per year), than the incidence among non-athletes, (1.0 per 100,000 per year).[3] In the US, a prospective study from 2009–11, found a 3.6 times relative risk for sudden cardiac arrest (SCA) in high school athletes vs non-athletes.[4] In Sweden, we have previously studied SCD in the young population 15–35 years.[5,6] Since the 1990's focus has been increased on SCD in athletes, both nationally and internationally, with cardiac screening recommendations for athletes, being published by the European Society of Cardiology (ESC) in 2005[7] and by the Swedish National Federation of Sports 2005 and 2014.[8] No follow-up of the incidence of SCD, since the introduction of such measures has been performed.

So far, SCD studies have commonly encompassed the risk of exertional SCD in competitive athletes, while reports on SCD during recreational sports activities are uncommon. This is important, since the borderline between competition and non-competitive sports is sometimes difficult. While competition offers an additional level of stress, training can be of very high intensity and duration, while, conversely, competitive athletes may be competing in non-exhaustive sports (bowling, chess). Gathering information about these deaths may be helpful in the planning of preventive strategies.

The aim of this study was to give an overview of all exercise related SCD in the young Swedish population during the 11-year period 2000–10, regarding the frequency, etiology, and prevalence of previous symptoms, family history and/or ECG-changes in this population. In addition, we aimed to compare the frequency of SCD in athletes in a well- defined national autopsy-based SCD cohort,[9] with similar national data from the previous decade.[6]

Material and methods

Definitions

SCD was defined as a witnessed, natural, unexpected death from cardiac causes occurring within 1 h after onset of symptoms in an apparently healthy person, or an unwitnessed, natural, unexpected death of a person observed to be well within 24 h of being found dead.[10] Subjects who initially survived sudden cardiac arrest (SCA) after cardiopulmonary resuscitation (CPR) and died more than one hour later after life-preserving measures were interrupted, were also included.

An athlete was defined as a person who participated in an organized team or individual sport that required regular practice and competitions.[11] Participation in competitions is what differentiated athletes from non-athletes.

Data collection

We have previously identified all cases of SCD in 1–35 years old from the 11-year period 2000–10 in Sweden by systematic search in the Swedish Cause of Death Registry and the database of Forensic Medicine (n = 552).[9] From this cohort, we identified 514 cases in 10–35-year olds, 373 (73%) men, and 141 (27%) women. For each case, information on circumstances at death, pathogenesis, athletic activity, medical history, symptoms, substance use, and family history of sudden death was assembled from autopsy-, police- and medical records, where available. Family history included first-degree relatives, i.e. parents, siblings, children of the deceased, and in addition, grandparents, uncles and aunts of the deceased.

ECG

Twelve-lead ECGs for all subjects were searched for in military conscription- and medical records. The ECGs identified, were classified according to the recent International recommendations for ECG interpretation in athletes into normal, abnormal or borderline.[12]

The study groups

1 All cases of exercise-/sports related SCD in 10–35-year olds during 2000–10 in Sweden, occurring during or shortly after physical exertion. The subjects were characterized as athletes or non-athletes. Athletes also included former competitive athletes, i.e. those who had stopped competing because of symptoms such as pre-syncope/syncope.
2 All SCDs in 15–35-year-old athletes, exercise related and non-exercise related, for comparison with the years 1992–99.

The control group

Was made up of all SCDs in athletes, 15–35 years old, in Sweden, during the years 1992–99, as previously described.[6]

Statistical analysis

The SCD incidence by sex and year, in age group 10–35 years, was calculated from the number of SCD, divided by the number of residents in Sweden for each sex and year, according to Statistics Sweden.[13] The mean population in Sweden was 9.10 million inhabitants during this period. Poisson regression was used to test trends by calendar year. Chi-square tests with Yates' correction or Mid-P tests have been used to detect significant differences between groups, and the level of significance was set at $P < 0.05$. For comparison of the number of SCDs in 15–35-year- old athletes, the total number of SCDs/year was compared, between the 1992–99 and 2000–10 cohorts.

Autopsy

All forensic autopsies are performed or supervised by a limited number[20] of experienced forensic pathologists, located in six regional departments. Autopsy is performed according to several consensus documents and a Swedish accreditation standard protocol. Clinical autopsies are performed in numerous hospitals in Sweden. Histological evaluations were performed in all subjects with forensic autopsy and in most of cases of clinical autopsy.

Ethics

The Regional Ethical Review Board at Umeå University (2012) and at Uppsala University (2017) approved the study (Dnr 2017/430, 2017/431).

Results

Exercise related sudden cardiac death

Cardiopulmonary resuscitation (CPR) was started in all witnessed cases (48/62; 77%), and in some unwitnessed cases when found

Table 1 – Baseline data for 62 cases of exertional sudden cardiac death in persons aged 10-35-years in Sweden during 2000-10.

Postmortem diagnoses No. (% of total)	No. of M/F	Athletes M/F (%)	Mean age (years) M/F	BMI Kg/m^2 M/F	Family history M/F (%)	Cardiac Symtoms M/F (%)	Premortal cardiac diagnoses M/F (%)	Ventricular fibrillation M/F (%)	Comorbidity M/F (%)
SADS 15 (24)	12/3	4/1 (33)	22.5/29	23.2/22.4	1/1 (13)	4/0 (27)	4[b]/0 (27)	5/1 (40)	[i]3/0 (20)
HCM 10 (16)	9/1	5/0 (50)	22.3/22	25.7/24.1	5/0 (50)	1/1 (20)	2[c]/1[d] (30)	5/1 (60)	[k]1/0 (10)
ARVC 8 (13)	7/1	1/0 (13)	25.4/28	23.9/23.9	6/1 (88)	4/1 (63)	1[e]/0 (13)	2/0 (25)	[l]1/0 (13)
Myocarditis 7 (11)	6/1	1/0 (14)	21.1/13.5	23.5/23.9	1/1 (29)	1/0 (0)	0/0 (0)	1/0 (14)	0/0 (0)
CAD 7 (11)	7/0	2/0 (29)	31.8	26.9	3/0 (43)	1/0 (14)	5[f]/0 (71)	2/0 (29)	[m]1/0 (14)
UCM 6 (10)	6/0	3/0 (50)	23	23.2	0/0 (0)	4/0 (67)	1[g]/0 (17)	3/0 (50)	[n]2/0 (33)
DCM 4 (6)	4/0	0/0 (0)	21.3	20.8	0/0 (0)	2/0 (50)	2[h]/0 (50)	1/0 (25)	[o]2/0 (50)
[a]Other 5 (8)	5/0	4/0 (80)	19	20.4	0/0 (0)	1/0 (20)	1[j]/0 (20)	0/0 (0)	0/0 (0)
Total 62	56/6	20/1 (34)	23.3/21.3	23.5/23.2	16/3 (31)	18/2 (32)	16/1 (27)	19/2 (34)	10/0 (16)

SADS, sudden arrhythmic death syndrome; CAD, coronary artery disease; HCM, hypertrophic cardiomyopathy; ARVC, arrhythmogenic right ventricular cardiomyopathy; DCM, dilated cardiomyopathy; UCM, unspecific cardiomyopathy.

[a] Other: thoracic aortic dissection (n = 2); Aortic stenosis (n = 2), coronary artery anomaly (n = 1).

[b] long QT syndrome (n = 1), sick-sinus syndrome (n = 1), Wolf Parkinson White syndrome (WPW) (n = 2).

[c] ventricular septal defect (n = 1), HCM (n = 1).

[d] WPW (n = 1).

[e] ARVC (n = 1).

[f] myocardial infarction (n = 3), familial hypercholesterolemia (n = 2).

[g] myocarditis (n = 1).

[h] WPW (n = 1), floppy mitral valve (n = 1).

[i] aortic stenosis.

[j] autism (n = 1), pollen allergy (n = 1), myotonic dystrophy (n = 1).

[k] nut allergy (n = 1).

[l] myotonic dystrophy (n = 1).

[m] pollen allergy and arthritis (n = 1).

[n] diabetes mellitus and celiaki (n = 1), pachyonychia congenital (n = 1).

[o] Mb Crohn (n = 1), diabetes mellitus and pollen allergy (n = 1).

(6/62; 10%). CPR was started by bystander in most cases (46/62; 74%) and continued by the ambulance crew. Ventricular fibrillation was seen in 34% (21/62) (Table 1). In five cases the victims initially survived after resuscitation, but died later in hospital.

Time trend

In men, suffering from SCD, during the 11-year survey period, 15% (56/373) were exercise-related. There was no change in the rates over time amongst non- exertional or exertional SCD, although the annual mortality rate per 100 000 per year varied between 1.5–2.3 for non-exertional and between 0.1–0.6 for exertional SCD (Fig. 1). In women, only 4% of SCD were exercise-related (6/141), and no comparative analysis of time trend has been made.

Sports activities

Among the 62 exercise-related SCDs, 41 (66%) were non - athletes (five women), and 21 (34%) were athletes (one woman) (Table 1). Eighteen different sports activities were practiced; the most common were running (n = 11) and floor ball (n = 10). Only one was at a national level (basketball). Three former long-distance runners had stopped competing because of syncope at exertion, and only practiced recreational sports.

Etiology

In most cases a forensic autopsy (47/62; 76%) or a clinical autopsy (13/62; 21%), was conducted. In one additional case, an athlete collapsed during a short distance competition due to thoracic aortic dissection and died during surgery. In another subject, echocardiography examination after cardiac arrest showed hypertrophic cardiomyopathy (HCM), and no autopsy was performed. A molecular autopsy was performed in four subjects, but no pathogenic variants causing cardiac disease were found.

Sudden arrhythmic death syndrome (SADS) (n = 15) and HCM (n = 10) were the most frequent diagnoses (Table 1). The entity UCM (unspecific cardiomyopathy), was used when there were slight and undefined histological findings. In men, HCM was a more common cause of exertional SCD (9/56, 16%) compared to non-exertional SCD (12/317, 4%) (P = 0.0008). Exertional SCD in arrhythmogenic right ventricular cardiomyopathy (ARVC) was more common (7/56, 13%) than non-exertional SCD (7/317, 2%) (P = 0.001) (Fig. 2). In women, no comparisons was made because of the low numbers of exercise-related SCDs (n = 6).

Premortal diagnoses and symptoms

In 17/62 (27%) cases of exercise-related SCD, a cardiac disease or familial hypercholesterolemia (FH) was diagnosed pre-mortally, among them WPW-syndrome (n = 4), myocardial infarction (n = 3), and one case each of ARVC and HCM (Table 1).

In addition, 8/62 (13%) had no previous diagnosis but had cardiac related symptoms, typically within a few months before death. Among them three persons postmortem diagnosed with ARVC, were examined with ECG due to exertional syncope or palpitations, in two cases accompanied by UCG, Holter-monitoring and exercise ECG. Two former long-distance runners, postmortem diagnosed with

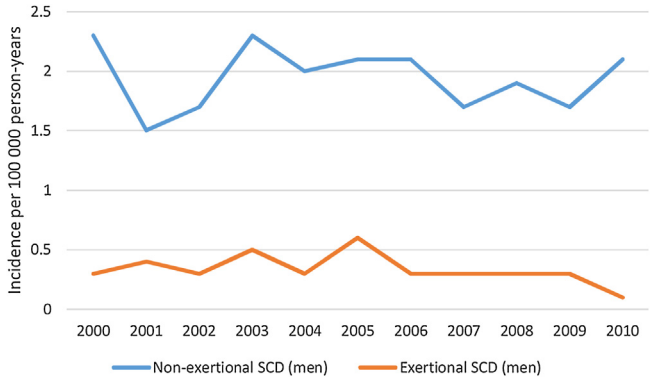

Fig. 1 – Trend analysis of exertional and non-exertional sudden cardiac death in men aged 10–35 years in Sweden during 2000–10.

SADS and UCM, had undergone an extensive in-hospital cardiac examination because of exertional syncope.

Family history

A family history of SCD, heart disease, or FH was present in 19/62 (31%) (Table 1). In ARVC 6/8 (75%) had a family history of SCD, and in four of these families there had been two cases of SCD, all with unknown diagnoses. In myocarditis 2/7 (29%) and in HCM 1/10 (10%) had a family history of SCD. A pre-mortem clinical evaluation in two of the study subjects had been performed because a first degree relative (with HCM and ARVC respectively) had suffered SCD, but no diagnosis had been found.

12-lead resting ECG

40 subjects had at least one previous ECG available, from conscription (n = 22) and/or from earlier visits to the health care (n = 24). In another 7 cases, an ECG was taken at SCA and/or was incomplete. In total, 18/40 (45%) ECGs were classified as abnormal (n = 16) or borderline (n = 2) according to the current International criteria for ECG interpretation in athletes.[12] There were no clear statistically significant differences between different underlying causes of SCD regarding the prevalence of previous ECG-changes, although 2/5 (40%) cases with ARVC showed abnormal ECG and another case was borderline abnormal. For all other diagnoses, the prevalence of abnormal ECG's varied between 25–33%. The most common findings were t-negativity, in 12/18 (67%) cases (Table 2).

Risk group

A "risk group" for future SCD, was constructed by adding together subjects with either cardiac symptoms/diagnoses (n = 25) and/or a family history of SCD (n = 9), and/or ECG abnormalities (n = 18), totally 30 individuals (48%).

All with cardiac symptoms had been to a doctor, where at least an ECG was taken. One individual with a history of syncope and palpitations had a DDDR-pacemaker since childhood because of sick sinus syndrome. No one had received an ICD, but it had been discussed in four cases with recurrent syncope, diagnosed with ARVC (n = 2) and UCM (n = 2) at autopsy. Individuals with pre-mortem myocardial infarction or FH were treated with lipid-lowering treatment (n = 5), anti-trombotics (n = 3), and beta-blockers (n = 3). Three former long-distance runners with exertional syncope and one individual with ARVC, also had beta-blockers, another subject with syncope, was treated with calcium blockers.

Exercise-related SCD in athletes vs non-athletes

Among athletes aged 10–35 years, 21 individuals suffered exercise-related SCD during the study period, and eight individuals suffered non-exertional SCD. Among non-athletes, 41 persons aged 10–35 years, suffered exercise-related SCD, and 444 individuals suffered non-exertional SCD. Thus, death during physical activity was more common in athletes (21/29; 72%) than in non-athletes (41/485; 8%) ($P < 0.0001$).

Comparison with a previous study in 15-to 35-year-old athletes

In a previous study from 1992-99, we found 32 SCDs among 15–35 year old athletes, nationwide in Sweden, including only cases with forensic autopsy.[6] As about 80% of all SCD cases in Sweden were captured in this study,[5] the adjusted figure is 40 cases, i.e. 5 SCDs per year during the eight–year period 1992–99. In the current

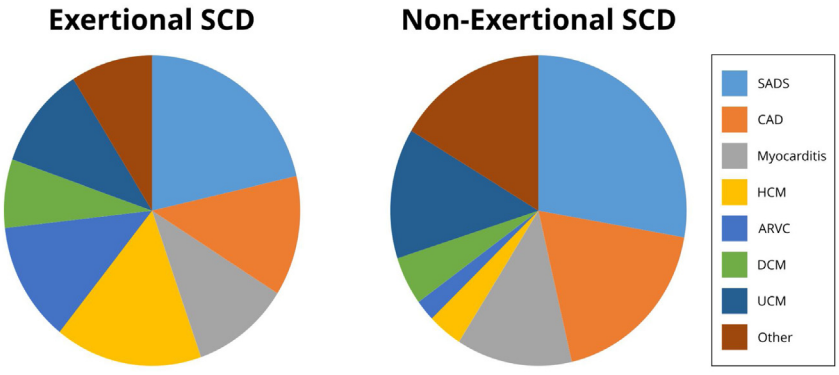

Fig. 2 – Pathogenesis in exertional sudden cardiac death (n = 56) and non-exertional sudden cardiac death (n = 317) in men aged 10–35 years in Sweden during 2000–10. SADS, sudden arrhythmic death syndrome; CAD, coronary artery disease; HCM, hypertrophic cardiomyopathy; ARVC, arrhythmogenic right ventricular cardiomyopathy; DCM, dilated cardiomyopathy; UCM, unspecific cardiomyopathy. Other: congenital heart disease; thoracic aortic dissection; coronary artery anomaly.

Table 2 – Pathological ECG findings and premortal risk profile in 18 cases of exercise related SCD in persons aged 10–35 years in Sweden during 2000–10.

Autopsy diagnosis	Premortal symtoms/premortal diagnosis/heredity	ECG class (International criteria)	Age at ECG (years)	Age at death (years)	ECG comments
SADS	Syncope, dizziness/LQT-syndrome	Abnormal	12	12	SR 56, T-negativity V1–5
SADS	Family history of SCD	Abnormal	13	13	SR 101, LQT (after resuscitation)
SADS	Palpitations/WPW-syndrome	Abnormal	14	17	SR 60, delta wave
SADS	Family history of SCD	Abnormal	22	22	SR 52, weak R-progression, T-negativity V1–3
SADS	Syncope, palpitations/sick sinus syndrome	Abnormal	14	22	SR, T-negativity V1-4+III. Pacemaker.
SADS	Palpitations/WPW-syndrome	Abnormal	17	24	SR 57, RBBB, short PQ, T-negativity V1–4 + inferior
CAD	Chest pain/myocardial infarction	Abnormal	31	32	SR 67, Q-wave III, T-negativity aVF, III + V6
CAD	Chest pain/myocardial infarction	Abnormal	18	35	SR 70, T-negativity V2–5
Myocarditis	Fatigue/mononucleosis recently	Borderline	18	26	SR 100
HCM	Dyspnoea, palpitations/HCM/family history of cardiac disease	Abnormal	11	11	SR 73, T-negativity V1–3, ST-depression aVF + III
HCM	–	Abnormal	17	22	SR 77, T-negativity V3–6, QRS-voltage increased
HCM	Palpitations/suspected WPW	Abnormal	19	22	Paroxysmal atrial-fibrillation
ARVC	Palpitations, dizziness/family history of SCD	Abnormal	12	12	SR 79, low voltage, T-negativity V1–5
ARVC	Palpitations, dizziness/ARVC/ family history of SCD	Abnormal	16	18	SR 51, T-negativity V1–4, inferior Q, VES bigeminy
ARVC	Syncope, palpitations/ family history of SCD	Borderline	28	28	SR 53, unspecific ST-depression
DCM	Palpitations/WPW	Abnormal	17	24	Paroxysmal atrial flutter, delta wave
UCM	Syncope/myocarditis	Abnormal	25	25	SR 55, T-negativity V5–6, VES/VT
UCM	Syncope	Abnormal	18	31	SR 64, T-negativity V1–4

SCD, Sudden cardiac death; SADS, sudden arrhythmic death syndrome; CAD, coronary artery disease; HCM, hypertrophic cardiomyopathy; ARVC, arrhythmogenic right ventricular cardiomyopathy; DCM, dilated cardiomyopathy; UCM, unspecific cardiomyopathy; SR, sinus rythm; LQT, long QT time; RBBB, right bundle branch block; VES, ventricular extrasystole.

study there were 25 SCDs among 15–35-year-old athletes or 2.3 SCDs per year (25/11), i.e. a halving compared to the years 1992-99. As the percentages of athletes and the mean population have been stable during these two decades, we consider these figures to reflect a real decrease.[6,13,14]

Discussion

The main results of the present study were, that among all subjects 10–35 years old, suffering from SCD in Sweden 2000–10, 12% were exercise-related, and affected mainly men (90%). SADS was the most common etiology. Death during exercise was more common in athletes than in non-athletes ($P < 0.0001$). Specifically, exercise seemed to trigger SCD in men with ARVC and HCM. 48% of the subjects had a premortal risk profile, potentially detectable by recommended cardiac screening strategies.[7] One third of the affected were athletes, and two-thirds non-athletes. SCD among athletes 15–35 years old, approximately decreased by half compared to the years 1992-99, from on average five cases per year to 2.3 cases of SCD/year.[5,6]

A high frequency of SADS, (24%), is a result consistent with several reports,[15–18] suggesting primary arrhythmia diseases as an important cause of SCD in athletes. The increased risk of exercise-related SCD in HCM ($P = 0.0008$) and ARVC ($P = 0.001$) is also shown in other studies. In a large forensic study from the U.S., HCM accounted for one third of SCD among athletes.[19] A strong association of ARVC with sports-related death has been shown in the United Kingdom and Italy.[18,2] The results of the present study confirm that exercise may be an important trigger factor for SCD in susceptible individuals, with underlying cardiovascular abnormality. These results also supports the current recommendations on restrictions in sporting activities, among athletes with known HCM and ARVC.[20]

The proportion of women suffering from exercise-related SCD compared to men was 1:9, in non - exertional SCD the figures were 1:2. This large male predominance in athletes is consistent with other studies.[4,17,18] It has been proposed that male gender, in itself, is a risk factor because of men having a higher prevalence of cardiomyopathies and premature CAD. Partly the difference may also be explained by a higher degree of training intensity and participation in competitions in male athletes. In the US college setting (17–24 years), despite a similar sporting participation, the gender difference is still present, albeit lower, 1:3.2.[15]

Prevention of SCD in general is linked to the recognition of any existing symptoms and subsequently an accurate pre-mortal diagnosis. 6/8 subjects with ARVC were investigated, but only one had a correct diagnosis. In addition to a standard investigation with ECG, UCG, Holter-monitoring and exercise ECG, other diagnostic methods should also be used, when suspecting ARVC.[20] In light of the high percentage (42/62; 68%) without symptoms, also strategies to detect asymptomatic individuals at risk are needed. An asymptomatic elite basket player, died before screening was recommended in elite athletes in Sweden 2006. In subjects with ARVC, 88% had heredity, which probably had been investigated to a higher extent today.

The fact that 45% of available ECGs was abnormal or borderline, is a very important finding in regard to screening. Resting ECG has shown to have a high sensitivity for underlying disease associated with SCA/SCD, while symptoms and family history has shown a lower sensitivity.[21] Major cardiac associations (European Society of Cardiology, American Heart Association) agree on the principle

and importance of cardiac screening of athletes, but disagree on the inclusion of the ECG in screening.[7,22] While having a high sensitivity, the major problem with the ECG, has been the low specificity and thus the need for further evaluations. With development of ECG interpretations criteria, this problem is now much lower, with higher specificity and preserved high sensitivity.[12] The results of the present study, showing that 48% of victims of SCD associated with exercise, had risk factors, prior to the fatal event, supports the use of cardiac screening, as recommended by the ESC (family history, symptoms, physical examination and resting-ECG).[7] Appropriate treatment, eg. beta- blockers, ablation (WPW-syndrome), surgery (anomalous coronary artery origin), pacemaker (brady-arrhythmias), or an ICD in high risk patients may, in some cases, allow the athlete to continue sports.[23] Recommendations from 2005 for various cardiovascular diseases are now being updated. An individualized approach and involvement of the athlete in the decision-making process is suggested. Selected patients with ICD may continue with sports with exception of sports with bodily contact.[20]

In the present study we found a higher percentage of exercise-related SCD in athletes than in non-athletes, a result in accordance with other studies.[6,18] A prospective French study, reported a 4.5 times higher risk in athletes (10–35 years) than in recreational sports participants.[16] Also these results may have implications for cardiac screening, which is currently recommended for competitive athletes only.[7]

Importantly, the yearly mean number of SCD in young competitive athletes in Sweden has been approximately halved in the 2000's compared to 1992–99.[6] No such decrease was found in the general population of the same age in Sweden, when the 2000's was compared to 1992–99.[9] Increased public awareness of SCA/SCD in sports, safety measures at sports arenas/facilities (medical action plans with defibrillators and CPR), and the introduction and increased use of cardiac screening, may all have contributed to this positive development in athletes.[24–26] In a British study of cardiovascular screening in 11,000 adolescent soccer players from 1996 to 2016, 42 (0.38%) had diseases associated with SCD and 225 (2%) had other cardiac disorders. SCD occurred in 8 cases, i.e. in 0.7/1000 screened athletes, 2 of which had been diagnosed by screening.[27]

Strengths and weaknesses of the study

This is a comprehensive, national study, and we have a solid knowledge about all cases. We may have underestimated the number of athletes, especially if the athlete died during rest or sleep. Our data is almost 10 years old, but is from a period when SCD began to receive much more attention than before and is interesting to compare with the previous decade.

Conclusion

12% of SCD cases in the young Swedish population are exercise-related, with SADS being the most common etiology. The risk of exercise-related SCD is higher for athletes compared to non-athletes. Specifically, exercise seems to trigger SCD in men with ARVC and HCM. About 50% of the subjects have a pre-mortal risk profile, highlighting the possibility to detect these individuals prior to the fatal event, by cardiac screening. Importantly, the number of SCDs/year in young athletes has been approximately halved in the 2000's compared to the 1990's. To further increase the present knowledge, we advocate a uniform post-mortem evaluation of all exercise-related SCD in young athletes, including the use of optimal histology and genetic analysis at autopsy, as recently outlined.[28]

Conflict of interest statement

There is no conflict of interest.

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