Review Article

Preparticipation Screening of Athletic Participant: A Proposal for the Cardiologist

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Abstract

Hence, the sudden cardiac death (SCD) in sports activity is a rare event, it affects the community deeply. To prevent the SCD on the athletic field, screening of athletic participants is necessary. Lack of randomised controlled trials in sports-cardiology makes the decision-making process hard for the physician. Hereby, a screening model including a family and personal history, an appropriate physical examination of the athlete and 12-lead electrocardiogram, is proposed for athletic participants based on a review of current literature and in accord with the European recommendations.

Keywords: Athletic population, electrocardiogram, preparticipation screening, sudden cardiac death

Introduction

Sudden cardiac death (SCD) itself is a devastating event, especially when it occurs during an athletic activity in a previously healthy individual. Although the exact incidence of SCD is not known, 1–2 of 100,000 athletes die suddenly in the age group between 12 and 35 years as an estimation.[1] Although the incidence of SCD is low in the athletic population, Italian experience suggests the incidence is approximately three times greater than the sedentary counterparts (estimated at 2.3/100,000 vs. 0.9/100,000 individuals). However, the only exception is a recent study from Denmark that did not support these results and showed a reduced SCD incidence in the athletic population.[1-3]

Sports activity itself does not increase mortality but may act as a trigger for lethal arrhythmias in predisposed participants with underlying, even silent, cardiac disease.[1] Because of the catastrophic effects of SCD of an apparently healthy individual, every precaution should be taken to identify the high-risk individuals and prevent SCD on the athletic field.

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Background

Observational evidence about the benefits of regular physical activity and sports participation outweigh by far the risk of SCD,[4] and regular exercise should be encouraged in all ages.

As a result, preparticipation screening (PPS) of the athletic population has become more popular with the growing number of individuals who want to participate in sports activities as part of an active lifestyle and medico-legal issues regarding SCD on the athletic field.

Because of the lack of specific regulation in Turkey, either family physicians or cardiologists are confused about the PPS process. The present work aimed to establish a practical proposal for PPS of the athletic participants of all ages.

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Preparticipation screening process

The most important factor in PPS is the definition of athletic participant. Before 2016, the definition was "competitive athlete," for the person who participates in regular competition as an individual or as a part of an organized team, with regular and intense training under unique pressure to progress in achievement and performance.[5,6]

Recent observations showed the incidence of SCD is similar in both competitive and noncompetitive athletes[7] and it is recognized that athletic participants, either recreational or amateur, usually show the behavior to exert physically up to their limits.[1] Therefore, the definition of an athletic participant has been revised in the latest recommendation of the European Society of Cardiology (ESC). Individuals of any age, either amateur or professional, who exercise on a regular basis, independent of the competitive status, are defined as athletic participants, according to the last European guideline.

The second important issue is the cause of SCD on the field. If the causes of SCD of the athletic population are identified, the screening can be done for these and precautions to prevent SCD on the field can be taken. Age is an important determinant for the cause of SCD. Coronary artery disease (CAD) is the leading factor of SCD in adult and older (age >35 years) athletes, while young athletes (age between 12 and 35 years) die mostly because of a variety of cardiac disease, including mostly genetic diseases such as hypertrophic or arrhythmogenic cardiomyopathy or congenital anomalies. The causes of SCD in young athletes are given in Table 1.[1,6]

The most important debate is about the screening process. The aim here should be not only to prevent SCD but also to avoid unnecessary disqualification and advanced-expensive diagnostic work-up. Given the fact that the majority of older athletes die because of CAD, some additional testing might be beneficial in high-risk older athletes.[8]

Screening protocols vary from country to country, and currently, broad-based systematic screening of athletes of any level is enforced in several countries, including the USA, Italy, and Israel. While in the USA, screening is done with only family/personal history and appropriate physical examination, PPS in Italy and Israel is done with an electrocardiogram (ECG) on top of that. Both of European and American recommendations agreed to include a detailed familial and personal history and an appropriate physical examination because most of the common causes of SCD in the athletic population have either genetic origin or lead to clinical important sign or symptom, but there are still debates on the additional ECG screening.

Twelve-lead ECG has the potential to detect some lethal conditions such as cardiomyopathies, myocarditis, preexcitation syndromes, and channelopathies, which are responsible for up to 60% of the SCD in athletes.[5] The addition of ECG into the PPS protocol enhances the sensitivity to detect cardiomyopathies or channelopathies from 25% to >90%.[9] With the implementation of ECG into the PPS protocol, SCD in young competitive athletes decreased by approximately 90% in Italy.[10]

On the other hand, the view from the USA highlights the low incidence of SCD in this population and the issue of the false-negative and false-positive ECG results, as well as cost and resource availability.[11,12] The main concern of these recommendations lies in balancing the detection of underlying diseases and not increasing the costs of advanced diagnostic workup with the unnecessary disqualification of athletic individuals, which brings the interpretation of athlete's ECG in a crucial position.

Consequently, for the accurate interpretation of the athlete's ECG, a big effort has been made since 2010. Regular and long-term intensive exercise causes some ECG changes in the heart, which reflects normal physiological adaptation and has to be distinguished from the abnormal findings. The first recommendation on athlete's ECG has been published in 2010 by ESC.[13] After that, these criteria have been updated in Seattle to decrease the false-positive results (Seattle Criteria), and in 2016 ESC has further revised the ECG criteria.[1,14] Because of the limits of the athletic heart can be overlapping those described as pathologic, an international group has defined the international criteria on athlete's ECG in 2017. The main difference of international criteria on athlete's ECG is to bring

Table 1: The causes of sudden cardiac death in young athletes

HCM
AR/LVC
CCAA
Myocarditis and dilated cardiomyopathy
Premature coronary atherosclerosis
Conduction system abnormalities (ventricular preexcitation and channelopathies)
Aortic rupture (Marfan syndrome)
Mitral valve prolapse
Commotio cordis

The causes of SCD in young athletes (age between 12 and 35 years) are given in the Table 1. The ratio of the cause changes from country to country. HCM is the leading cause of SCD in USA, while AR/LVC in Italy. An anomalous origin of left coronary artery from the right aortic sinus with an interarterial course between aorta and pulmonary artery is the most common CCAA related to SCD. Although not a cardiac disease and previously thought extremely rare, commotion cordis reports are increasing especially in adolescent males with a mean age of 14 years.[1,5,6] HCM: Hypertrophic cardiomyopathy, AR/LVC: Arrhythmogenic right or left ventricular cardiomyopathy, CCAA: Congenital coronary artery anomaly, SCD: Sudden cardiac death

a "borderline" definition beside physiologic and pathological features to avoid false-positive results, unnecessary diagnostic workup, and disqualification.[15] Another important tool of daily cardiology practice, transthoracic echocardiography (TTE) is an established imaging modality of athletes to be indicated when there is a suspicion of cardiac disease on physical examination or ECG, but in the mass-screening protocol, the addition of TTE does not increase diagnostic sensitivity to identify cardiomyopathies.[1,16-18]

Family and personal history

Medical history consists of an important part of the PPS; hence, a majority of diseases related to SCD during sport are genetically inherited, mostly with an autosomal dominant pattern. In the presence of any close relative with a premature heart attack or SCD (younger than 50 ages in males and 65 ages in females), or cardiomyopathy, or Marfan, long QT, Brugada Syndromes, severe arrhythmic or coronary or other cardiac disabling disease is considered as positive family history and requires further diagnostics. Chest pain or discomfort, syncope or near-syncope, palpitation with exercise, unexplained dyspnea, or fatigue with the degree of exercise should be accepted as a positive sign, which indicates further work-up. Furthermore, any previous cardiac diagnosis or any restriction from sports should be questioned on individual bases.[5]

Physical examination for preparticipation screening

Physical examination of the athletic population for PPS should be especially focused on the causes of SCD in this population. Musculoskeletal findings for Marfan Syndrome, reduced femoral artery pulses suggestive for aortic coarctation, any clicks or abnormal heart sounds or murmurs >2/6 grade indicative for valvular abnormality are considered positive and require further diagnostics. Furthermore, irregular heart rhythm or high blood pressure should be investigated further.[5]

Athlete's electrocardiogram

Hence an appropriate evaluation of an athlete's ECG is fundamental in the PPS process including ECG, anyone interested in this area should be familiar with the ECG changes in this special population. ECG changes related to regular exercise are defined "normal," while ECG changes suggestive of cardiomyopathy should be recognized early and treated properly. There are also some ECG changes, which should not be considered pathological if they present in isolation and defined as borderline. Normal, borderline, and pathological ECG findings, according to the latest international recommendations, are defined below and summarized in Tables 2-4.[15]

Normal electrocardiogram changes in athletes

Regular, intense, and long-term exercise induces electrical and structural remodeling in the heart. The knowledge of physiological changes is crucial for all physicians taking care of the athletic populations. Isolated QRS voltage criteria for left or right ventricular hypertrophy are considered physiologic and do not require further assessment. If there is an underlying disease, additional pathological features are present. Early repolarization (J-point elevation >0.1 mV) is common in athletes, especially in young participants, males, and of black

Table 2: Normal electrocardiogram changes in athletic population

Normal ECG changes (physiological changes due to exercise)
Increased QRS voltage for LVH or RVH
Incomplete RBBB
Early repolarization/ST segment elevation
ST elevation followed by T wave inversion V1-V4 in black athletes
T wave inversion V1-3 age <16 years old
Sinus bradycardia or arrhythmia
Ectopic atrial or junctional rhythm
1° AV block
Mobitz Type I 2° AV block

Normal ECG changes in an asymptomatic athlete with no family history do not require further diagnostic workup.[15] LVH: Left ventricular hypertrophy, RVH: Right ventricular hypertrophy, RBBB: Right bundle branch block, AV: Atrioventricular, ECG: Electrocardiogram

Table 3: Pathological electrocardiogram changes in athletic population

Pathological ECG changes (not related to exercise)
TWI
ST segment depression
Pathological Q waves
Complete LBBB
QRS ≥140 ms duration
Epsilon wave
Ventricular preexcitation
Prolonged QT interval
Brugada Type 1 pattern
Profound sinus bradycardia <30 bpm
PR interval ≥400 ms
Mobitz Type II 2° AV block
3° AV block
≥2 PVCs
Atrial tachyarrhythmias
Ventricular tachyarrhythmias

Pathological ECG changes in athletic population always require further evaluation for pathological cardiovascular diseases associated with sudden cardiac death.[15] TWI: T wave inversion, LBBB: Left bunble brunch block, PVCs: Premature ventricular contractions, ECG: Electrocardiogram, AV: Atrio-ventricular

Table 4: Borderline electrocardiogram changes

Borderline ECG changes in isolation do not require further evaluation
Left axis deviation
Left atrial enlargement
Right axis deviation
Right atrial enlargement
Complete RBBB

Borderline ECG changes in isolation do not require any further evaluation, but in the presence of 2 or more findings, further evaluation is required to investigate any pathological cardiovascular disorder related to sudden cardiac death in athletes according to the International Consensus Standards for Athlete's ECG.[15] RBBB: Right bundle brunch block, ECG: Electrocardiogram

ethnicity. Early repolarization with concave ST-segment elevation and peaked T-wave inversion (TWI) is seen in up to 45% of Caucasian and 63%–91% of black athletes.[15,19,20] J-point elevation and convex ST-segment elevation in the anterior (V1–4) leads with TWI in black athletes are considered normal variants related to ethnicity and do not require further assessment.[15,20-22] The juvenile ECG pattern is defined as TWI in the anterior precordial leads (V1-V3) in an adolescent athlete, who's age is between 12 and 16 years and does not require further evaluation in the absence of symptoms, signs, or familial history.[15] The incomplete right bundle branch block (RBBB) does not require further diagnostic workup if family/personal history and physical exam are normal.[13] The increased vagal tone in athletes is associated with some arrhythmias, such as sinus bradycardia, sinus arrhythmia, and less commonly junctional or ectopic atrial rhythms, first- and second-degree type 1 (Wenckebach) atrioventricular (AV) block.[15,19,23-26] Recently, not only the increased vagal tone but also the electrical remodeling of the sino-atrial node is considered to cause those arrhythmias. In the absence of symptoms, the heart rates ≥30 beats/minute are accepted normal, and bradycardia should resolve with the physical activity.[15]

Borderline electrocardiogram changes in athletes

The latest International Athlete's ECG recommendation has defined a borderline category to balance sensitivity (disease detection) and specificity (false-positive). Axis deviation and atrial enlargement are responsible for >40% of abnormal ECG patterns but do not correlate with underlying cardiac disease.[15,27]

While complete RBBB is present in 1% of the general population, the prevalence in athletes is reported as 0.5%–2.5%.[15,28-31] A study from the USA reported athletes with complete RBBB demonstrated larger right ventricular dimensions but no pathological structural cardiac disease.[15,32] Based on the data, left or right axis deviation, left or right atrial enlargement and complete RBBB defined if identified in isolation, are considered as borderline changes and do not require further work-up.[15]

Pathological electrocardiogram changes in athletes

Pathological ECG changes in athletes are not related to the intensive exercise and always require further diagnostic workup with a temporary restriction of the athletic individual from participation.

TWI ≥1 mm in depth in 2 or more contiguous leads is abnormal, except TWI in V1–4 in black athletes and TWI in V1–3 in athletes younger than 16 years.[15] TWI in inferior or lateral leads is common in HCM.[33-36] Data showed TWI in the lateral or inferolateral leads in athletes is seen with the presence of cardiomyopathy.[15,20,37-39] Similarly, an anterior TWI beyond V2 in a nonblack athlete older than 16 years should raise suspicion for arrhythmogenic right ventricular cardiomyopathy. ST-segment depression also is not a consequence of athletic training and should be investigated. While interpreting pathological Q-waves, lead misplacement should be taken into account. A high lead placement can result in a pseudo-septal infarct pattern, as pathological Q-waves in V1–V2.[15,40] For asymptomatic athletes, pathological Q-waves definition has been modified as a Q/R ratio ≥0.25 or ≥40 ms in duration in 2 or more contiguous leads except III and aVR, to decrease false-positive results.[15] Complete left bundle branch block is always abnormal and requires further workup. Regardless of QRS morphology, profound nonspecific intra-ventricular conduction delay (IVCD) defined as ≥140 ms is also an abnormal finding.

In an asymptomatic athlete, while a short PR interval without wide QRS or delta wave does not require further workup, Wolf-Parkinson-White pattern (PR <120 ms and delta wave and QRS >120 ms) warrants further testing.[15] Prolonged QT interval also important hence congenital long QT syndrome affects 1 in 2000 individuals.[41] Accurate measurement of corrected QT (QTc) interval is crucial and to balance the false positive and false negative results, the QTc's cut-off is chosen for male ≥470 and female ≥480 ms. The exact cut-off and clinical significance of a short QT interval in the athletic population is not known. According to the latest recommendation, a QTc <320 ms is considered as short QT, but it should only be investigated if there are some concerning clinical markers.[15] Athletes with type 1 Brugada pattern should be investigated even in the absence of symptoms, but potential factors for Brugada-like ECG patterns such as hyperkalemia, fever, some medications, and lead misplacement should be taken into account.[15]

Sinus bradycardia is common in conditioned athletes, but a marked sinus bradycardia <30 beats/minute or a PR ≥400 ms should be investigated. High-grade AV blocks such as Type II second-degree (Mobitz type 2) or third-grade AV blocks warrant further assessment. AV dissociation without block is not pathological, but the expression of autonomic mismatch. Atrial and ventricular tachyarrhythmias require further evaluation. Multiple premature ventricular contractions (PVCs ≥2 in 12 lead ECG) are considered abnormal and should be investigated for underlying disease, although they are usually benign.[15] A recent report identified PVC according to their morphology in 12-lead ECG into common (usually benign) and uncommon (may represent the expression of underlying cardiac disease) groups and suggests using the morphology other than the number of PVC.[42]

Preparticipation screening of older athletes

Hence, the main cause of SCD in athletes older than 35 years is the CAD; screening should be done accordingly. Both European and American guidelines recommend using screening questionnaires to identify high-risk individuals.[8,43] The aim of the questionnaire is to reveal an underlying heart disease and decrease the need for physician interaction. The questionnaire proposal for older athletes is given in Table 5. To encourage exercise and screen a bevy of people, if a low-intensity activity is aimed and self-assessment with the questionnaire went well, no further testing is recommended. If

any the questionnaire is positive or moderate to high exercise activity is aimed, screening by a physician is recommended. Screening should be done with history, physical examination, ECG, and cardiovascular risk assessment such as SCORE risk charts. High-risk profile is summarized in Table 6. If screening is positive, then a maximal exercise testing is recommended, and according to its result, approval for exercise or further testing is determined. It is important to keep in mind here the exercise test is applied as a part of a risk stratification strategy and not simply for rule-in or rule-out CAD.[8]

Conclusion

The need for a national PPS modality is a growing issue according to the request for PPS of athletic participants such as professional athletes and athletes from either schools or sports federations or sports clubs in Turkey in line with the rest of the world. Hereby, a common national PPS model similar to the European recommendations is proposed with the hope to standardize the care for athletes of any age and help colleagues in the decision-making process.

A family and personal history, appropriate physical examination, and ECG are recommended in PPS of young athletes. For older athletes, an additional risk assessment is needed. If the athlete has high-risk profile, an exercise test is recommended as part of risk stratification. It is important to keep in mind and share the knowledge with the athletic participants, that the risk of SCD (1–2/100,000) still remains despite all the screening process, but the risk is very low, and the beneficial effects of exercise are overwhelming.

Table 5: Questionnaire for older athletes

Has a doctor ever said that you have a heart disease and recommended only medically supervised activity? (Did you ever have a heart attack, coronary angiogram, coronary stent or heart surgery?)
Yes/no
Has a doctor ever recommended you medication for heart disease or hypertension?
Yes/no
Do you ever have chest pain or palpitation with physical activity?
Yes/no
Do you ever have dizziness, fainting, blackouts or unreasonable shortness of breath?
Yes/no
Are you aware, through your own experience or a doctor's advise, of any other reason that would you prohibit from you exercising?
Yes/no

Proposed questionnaire for older athletes. If all the answers are no, there is no need for further work-up in individuals aiming low intensity activity. Adopted from Ballady. Circulation 1998;97:2283-93

Table 6: High risk profile for older athletes

The presence of multiple risk factors with an estimated 10-year risk >5% according to score risk chart
Significant high blood cholesterol (LDL >240 mg/dl) or blood pressure (>180/110 mmHg)
Diabetes mellitus with microalbuminia
Individuals with a strong family history for premature CVD in first-degree relative younger than 50 years or individuals with a BMI more than 28

High risk profile for older adults. In the presence of even 1 factor maximal exercise testing is recommended for risk stratification.[8] CVD: Cardiovascular disease, BMI: Body mass index, LDL: Low-density lipoprotein

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Conflicts of interest

There are no conflicts of interest.

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