The Athlete's Heart: Prevention of Sudden Cardiac Death

Ramin Manshadi, MD, FACC, FSCAI, FAHA

Division of Cardiovascular Medicine, University of California Davis School of Medicine, Sacramento, CA

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Sudden cardiac death (SCD) in athletes has been with us for thousands of years, and it continues to be a newsmaker even to this day. The first known incidence of SCD in an athlete occurred to a Greek runner, Pheidippides, in 490 BC.[1] He was the long-distance messenger who brought the glorious news of the defeat of the Persians to Athens and then died suddenly. SCD in athletes is not an uncommon event and can be seen at all levels of athletics; it transcends borders and occurs in all parts of the world. Just within a recent 2-month period, 2 young high school athletes in Sacramento, California, experienced SCD. The precise incidence of SCD in athletes is very difficult to estimate and underscores the lack of a national or global registry to monitor such events. There is a great discrepancy among different sources, with incidence rates ranging from 1 in 10,000 to 1 in 100,000.[2]

Few, if any, studies have provided guidance that would help design protocols to screen for athletes at risk of SCD. What are we doing on a global, national, and local level to collect the information to help combat this problem? There has been a push by the American Heart Association (AHA) to utilize standard health assessment forms for preparticipation of athletes, but the results have not been very promising. A review of data from the National Collegiate Athletic Association (NCAA) suggested that only 26% of AHA forms are adequately completed. The incompletion rate was 30% in NCAA Division II and III schools, and 14% in Division I schools.[3]

Although there has been some movement to address this lack of screening, we are still hearing news reports about elite athletes dying suddenly. Researchers in Italy have significantly reduced the incidence of SCD in their athletes with a national mandatory screening program, which includes a standardized history form and a physical examination performed by a competent clinician who is well versed in performing preparticipation physical examinations and electrocardiograms (ECGs).[4]

In Italy, the most common cause of SCD in athletes is arrhythmogenic right ventricle cardiomyopathy. In contrast, in the United States, SCD in athletes is most often secondary to hypertrophic cardiomyopathy (24%). In general, most of the SCD events that occur in athletes younger than 35 years are due to structural heart disease, such as muscular, valvular, or anomalous coronary arteries. For athletes older than 35 years, atherosclerosis is the most common underlying cause of SCD (seen in 80% of cases). Marfan syndrome may also lead to SCD in athletes. Coronary artery disease (CAD) is commonly observed in athletes older than 35 years.

Recommendations

Because there is little clinical research available to help guide the development of specific recommendations, suggestions are based on general

population studies. If an athlete older than 35 years has no symptoms suggestive of heart disease, a coronary calcium score less than 15, and a normal physical examination and ECG, then he or she is allowed to participate in athletics without further testing. When the coronary calcium score is greater than 100, the athlete requires a full examination, including a stress evaluation.

Athletes with a known diagnosis of CAD who have been treated with revascularization (percutaneously or with coronary artery bypass surgery) should not participate in competitive sports, given their higher likelihood of SCD. They may participate in mild-to-moderate physical activity. All patients who have been diagnosed with hypertrophic cardiomyopathy should be disqualified from any further sport competition, even if an implantable defibrillator is placed. Athletes known to have hypertension (HTN) should be approached as follows:

- Stage 1 HTN with left ventricular hypertrophy: blood pressure must be controlled before competitive sports are resumed.
- Stage 2 HTN, with or without signs of end-organ dysfunction, such as left ventricular hypertrophy: blood pressure must be controlled prior to participation in athletics.
- Athletes with Marfan syndrome without aortic root dilatation, mitral insufficiency, or family history of SCD may participate in low dynamic/static exercise, such as golfing and bowling.

Athletes with known myocardial bridging of an epicardial coronary artery without objective evidence of coronary ischemia may participate in competitive sports. However, if there is evidence of coronary ischemia, revascularization must be performed. After 6 months, participation in all sports can be resumed.

There is no scientific evidence showing that performance-enhancing substances increase athletic achievement in a safe and effective way. The athletic governing bodies should provide a comprehensive list of prohibited drugs to all athletes, develop a rigorous approach to prevent the use of these drugs, and provide education, counseling, treatment, detection, and enforcement.[5]

Screening and Prevention

In what ways can clinicians maximize the detection and prevention of SCD and avoid some of the common roadblocks? First, we must standardize the AHA forms used in the United States and around the world, so that the relevant information is easily captured. Currently, in the 43 states that have forms, only 17 forms can be considered adequate. There should also be standards regarding which clinicians should perform the physical examinations. At this time, these examinations can be performed by nurse practitioners and physician assistants in 21 states, by chiropractors in 10 states, and by naturopathic doctors in 1 state. Interestingly, 75% of team physicians for NCAA teams are orthopedic surgeons, who may not be trained to perform an adequate examination and assessment of SCD risk.[6] There should be a standardized, mandatory global tracking system and registry for all sudden cardiac arrest events, whether they are ultimately fatal or not. We should not rely only on the lay media for our "clinical" data.

After proper screening, the next step is to intervene in a timely manner in those athletes who are at higher risk for SCD. Time to defibrillations is the most important factor in survival from out-of-hospital cardiac arrest. If the shock is delivered within 2 to 3 minutes, the survival is 50%, but by 4 minutes, survival drops to 25%, and after 10 minutes, it is 10%. The solution is to have automatic external defibrillators (AEDs) in all locations where athletic activity takes place, including stadiums, arenas, and tennis courts. There should be trained individuals who know how to activate and apply the AED, and protocols for managing a sudden cardiac arrest event, such as contacting 9-1-1 and performing cardiopulmonary resuscitation (CPR). If an AED can be accessed within 5 minutes of the event, 9-1-1 should be called immediately before the AED is applied. If AED access is longer than 5 minutes away, then 9-1-1 should be called and 60-second CPR performed before the AED is applied.[5]

Conclusion

This brief review of the state of SCD in athletes serves to raise awareness of this condition. Cardiovascular physicians should become more proactive to be sure that our high schools, universities, and professional teams are taking the necessary steps to prevent this event, and, if sudden cardiac arrest does occur, to have the protocols in place to effectively treat it. ■

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