Commentary Article

Interventional Cardiology

Commentary on athletes' sudden cardiac death

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Description

Athletes' Sudden Cardiac Death (SCD), although rare (0.5 to 1 per 100,000 athlete's years), but SCD in known athletes causes general shock. In order to better understand and reduce SCD, we collected 360 athletic SCDs (including only 14 women) from 32 physical sports from registers and newspaper articles found on the Internet, where the sport, age, and place/time of death (during training/competition/after) could be clearly determined. From these cases we prepared a uniform database in order of the year of SCD (see Annex/at the end of which the data of 5 successfully revived physical athletes and 15 competitive chess players SCD). In this database, we also provided other available information about each athlete (medical history, pre-SCD symptoms, suspected diagnosis, and post-mortem report).

The vast majority of deaths among physical athletes (239 cases) occurred in competitions (matches), while 99 occurred during training and 22 SCDs occurred after training/competition. The cases were grouped by sport. The majority of SCDs (248 cases, 69%) were team sports with a high proportion of dynamic components (football, American football, basketball, ice hockey), while 85 SCDs (25%) were found in individual endurance sports (marathon and half marathon, triathlon, cycling, swimming, rowing, canoeing). The age of those who died in team sports was significantly lower ($p<0.005$) compared to individual endurance athletes. There was a significant difference in the proportion of team athletes and individual endurance athletes who died in training or competition ($p<0.01$). In the former sports, more people died during training, in the latter during competition. The exception was American football, where many more people died in training (31 *versus* 12). The number of deaths in training is remarkably high in team sports (79/248 SCD), therefore we recommend resuscitation readiness in team sports as well. The high SCD rate of participants in team athletes' trainings may be explained by their younger age compared to the competitors ($p<0.01$), therefore their possible under-fitness or significant stress due to getting into the team can contribute to their SCD, so young people should be monitored more closely from medical, psychological and (under) fitness points of view. SCD after training/matches can be caused by increased vagotonia when the load is stopped, which can provoke malignant arrhythmia, or circulatory failure or collapse due to the sudden cessation of increased muscle tone during competition. This is supported by the fact that the proportion of deaths after training/competition was higher in sports requiring high static effort (in wrestling and weightlifting there were a total of 6 SCD, half of which 3 SCD occurred after competitions), while in other sports the ratio was 19/354. Some marathon runners also died after running (4/39). For this reason, in high-effort sports, athletes should continue to be monitored for at least 1 hour after the competition.

Stress can also play a major role in athletes' SCD, which can be proved by the fact that there was an SCD during warm-ups without significant physical exertion (5 SCD) and chess (15 SCD), and that, compared to 66 SCDs in marathon/half marathon/triathlon

competitions, there was no SCD at all in their stakeless training. Due to the increase in catecholamine levels due to stress, the already higher tension and heart rate due to physical exertion further increases, which can lead to deterioration of microcirculation and the development of microvascular dysfunction. Due to the supposed high stress, we recommend talking to a psychologist before high-stakes races, and for marathon runners (where there are many non-professional athletes who do not have a sports medicine examination) a medical examination is recommended before the race and an Electrocardiogram (ECG) within 1 month should also be required.

Although we found preliminary medical information about athletes in only a few cases (42/360) in the media, we found one eye-catching data: In 5 cases of SCD, sickle cell anemia was known beforehand. No evidence of possible drug use was given anywhere.

In total, only 87 post-mortem data were provided. Most acquired abnormalities were left ventricular hypertrophy (28 cases), right ventricular hypertrophy and dilatation (28 cases, but often associated with left ventricular hypertrophy), and coronary calcification (12 cases). Most congenital anomalies found: 9 cases of hypertrophic cardiomyopathy, 6 cases of arrhytmogen right ventricular cardiomyopathy.

Conclusion

The topics of sports medicine screening tests are still not uniform (in fact, in several countries there is none at all, and the prohibitive scope of the diseases found also differs). Accurate medical history may be of great importance, but the recommended protocols do not go into the details of this. We recommend a uniform protocol for anamnesis and physical/instrumental examination, and accurate documentation of the results available to those concerned.

Successful resuscitation without permanent neurological damage occurs optimally only in 40%-50% of cases (successfully resuscitated cases are not classified as SCDs, but are labeled aborted cardiac death). There are certainly two reasons why the resuscitation of otherwise generally healthy athletes is successful only in so few cases: The usually late or unprofessional resuscitation, and the adverse effect of biochemical changes occurring during sports. The material (defibrillator) and personal condition (qualified personnel) for resuscitation are usually present at competitions, but we recommend basic resuscitation education for all athletes, since they would usually be the first to start resuscitation, and we recommend blood sampling (pH, lactate, bicarbonate, K, Mg, and Ca ion levels) as soon as possible, because early knowledge of this could improve the prognosis for later hospitalization.