Sudden Cardiac Death and Exercise among the Young

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The Magnitude of the Problem

Regular, moderate exercise has several beneficial effects on health, cardiovascular and all-cause mortality. However, in certain circumstances, in predisposed individuals, an acute bout of exertion may cause an adverse health effect and this can include sudden death. The sudden death of a young person or athlete is a dramatic event with devastating impact on the family, sports community and medical personnel.[1-3]

Exercise induced sudden death (ESD) refers to an unexpected witnessed sudden cardiac arrest occurring within an hour of a bout of exercise in an apparently healthy person. The incidence is rare; estimated at 1 per 100 000 to 1 per 300 000 per year, in males. This is also the reason why we still discuss the usefulness of a routine medical pre-participation screening in preventing ESD. It has largely been demonstrated that cardiovascular diseases play a dominant role in ESD accounting for about 85% of cases. Often, the fatal cardiac arrest happens during or just after strenuous exercise, suggesting that the autonomic nervous system changes related to effort, may trigger malignant arrhythmias in the young person with cardiac disease.[2-4]

Causes of ESD

Among cardiovascular diseases, atherosclerotic coronary artery disease is the leading cause amongst older athletes (>35 years). In the younger group (<35 years), it is often caused by rare and often silent heart diseases. In about 2% of these athletes with sudden death, no identifiable causes are found at post-mortem examination.

Some of the possible causes of ESD include:

Hypertrophic Cardiomyopathy (HCM)

This is a genetic disease with a prevalence in the general population of about 1 in 500 people. HCM can be detected by routine medical preparticipation screening. It is characterized by an abnormal hypertrophy of the left ventricular walls with spatial disarray of the myocardial fibres at microscopic level and a propensity to life-threatening arrhythmias. There is usually an extreme symmetrical left ventricular hypertrophy with wall thickness greater than 13 mm, a reduced cavity diameter and impaired diastolic filling. HCM may be totally asymptomatic and physical examination of the person may be completely normal. However, the diagnostic suspicion will arise from the electrocardiography (ECG), which tends to be abnormal frequently. Once this is suspected, a well conducted echocardiography (ECHO) and/ or MRI examination generally confirms the diagnosis.

Myocarditis

This is an inflammatory condition of the heart muscle, most frequently due to a viral infection. Some people with myocarditis may be completely asymptomatic whilst others may show subtle symptoms like mild exercise intolerance, palpitations, resting tachycardia or excessive increase in heart rate with exercise. These can appear days to weeks after a viral illness. For this reason, the incidence of myocarditis amongst athletes is often underestimated. The ECG may be abnormal and dysrhythmias may occur, especially when these young people are being investigated with a stress ECG and Holter monitoring. Malignant ventricular arrhythmias may be the cause for ESD either during active or healing myocarditis. Therefore, it is important to make an early diagnosis, based on a high index of suspicion, and to allow full recovery before returning to the sport/ exercise.

Congenital Coronary Artery Anomaly (CCAA)

This is very rare in the general population but in young athletes with CCAA, there is a 70-fold increase in relative risk of dying during effort than at rest. This condition is rarely diagnosed during one's lifetime. The most common abnormality linked to ESD is a left main coronary artery arising from the right sinus of Vasalva (this should normally arise from the aorta). The physical examination as well as rest and stress ECG are usually normal and only about a third of subjects may have symptoms which are non specific such as chest pains, palpitations and syncope/ pre-syncope (fainting or near fainting). ECHO, in experienced hands

can pick up the diagnosis and these days MRI or angio-coronary computed tomography (angio-CT) scans can solve doubts on coronary artery anatomy.[5]

The other possible causes that have been linked to ESD include:

Wolff-Parkinson White syndrome, a disorder of the cardiac conduction system characterized by the congenital presence of one or more additional electrical pathways.

Congenital electric heart diseases, a group of diseases with genetic anomalies of the ion channels ("channelopathies"), which include long or short QT syndrome and Brugada syndrome (these are conditions which may manifest with ECG changes).

Drugs and doping: stimulants such as epinephrine, ephedrine and cocaine all have a pro-arrhythmia effect, particularly in the presence of dehydration and in association with other heart anomalies. At times, deaths have been linked to the use of performance enhancing agents such as erythropoietin, possibly from blood hyperviscosity and increased thrombogenesis. Anabolic steroids may cause cardiac hypertrophy, myocardial fibrosis and accelerated atherosclerosis with medium to long term use.

Commotio cordis (CC)

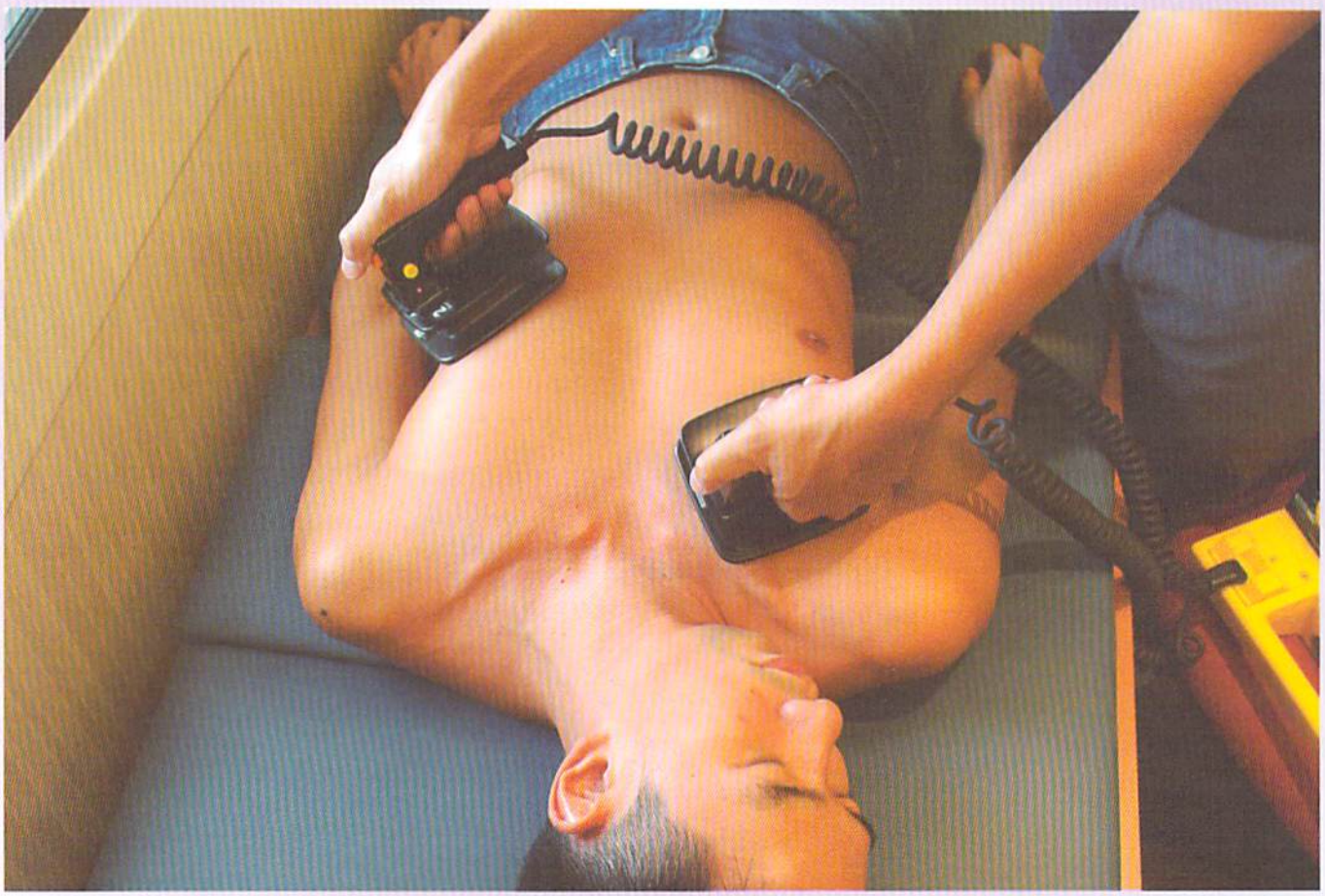
This is an electrophysiological event caused by precordial chest impact in individuals who are free of heart disease. When the chest impact is delivered within a narrow, electrically vulnerable period of the cardiac cycle, it may start an abnormal malignant rhythm called ventricular fibrillation (VF). CC can occur in contact sports or sports involving balls such as base ball or football.

Mechanism and Management

There are three postulated mechanisms by which vigorous physical activity may precipitate myocardial ischaemia in athletes where the etiology is coronary artery disease:

a. The initial surge of blood pressure with initiation of exercise might precipitate the fissuring of a vulnerable atherosclerotic plaque with the subsequent thrombus formation transforming previously non-occlusive lesion into total occlusion.

b. A non-occlusive plaque may precipitate transient ischaemia by inducing an imbalance between myocardial oxygen demand and supply. As fewer than a third of resuscitated athletes subsequently develop evidence of acute myocardial infarction, it is thought that this is the commonest mechanism of sudden exercise induced death.



c. Myocardial ischaemia during exercise might result from coronary artery spasm which is commonest at sites of established atherosclerosis.

Irrespective of which mechanism is responsible, the event immediately preceding all cases of sudden cardiac death during exercise is a massive reduction of cardiac output (secondary to either myocardial ischaemia or arrhythmia), decreased cerebral blood flow and loss of consciousness. It is believed that a combination of the triggering event and a susceptible myocardium combine to initiate a potentially lethal arrhythmia. The fall in cardiac output upon rapid cessation of exercise and the presence of arterial dilatation are additional factors that might explain the relatively frequent occurrence of sudden death in the immediate post-exercise period.

Physical examination and cardiac auscultation plays a limited role but can identify athletes and individuals with aortic stenosis and approximately about 25% of those with HCM. The 12-lead ECG is useful in the diagnosis of WPW, short or long QT syndrome and Brugada syndrome. However, the classic ECG pattern in Brugada syndrome may be transient but can be unmasked by pharmacological provocation using certain drugs .The 12-lead ECG is non-specifically abnormal in up to 90% of individuals with heart muscle disorders, but ECHO is the most practical gold standard investigation. A coronary angiogram can be done if there are indications suggesting coronary artery disease. Most deaths are triggered by exertion, therefore the most pragmatic approach in preventing such catastrophes would be to recommend abstinence from medium and high intensity competitive sports. Decision to disqualify cannot be legally enforced in some countries such as the UK.[6]

Certain drugs can be considered for use in controlling symptoms such as in HCM. The prophylactic use of implantable cardiac defibrillators is recommended in some patients with HCM, congenital long QT syndrome and Brugada syndrome who are at high risk of life-threatening arrhythmias. Athletes with myocarditis are advised to stay away from sports and exertion until they and their cardiac function are fully recovered.

CONCLUSION

Sports physician should be able to recognize clinical symptoms in athletes and err on the side of caution when advising them to stay away from practice.

The identification of cardiac disorders in an athlete can prevent ESD. A cost effective initial pre-participation screening programme that comprises of health questionnaire survey with careful medical and family history, physical examination and a resting 12 lead is necessary to have. Exercise test can then be done on those where certain abnormalities are detected. This combination can identify subjects with potentially lethal disease. Further instrumental investigations (ECHO, MRI, Holter monitoring and CT-angio etc) may be useful when any diagnostic doubt arises.

Since a large majority of the diseases responsible for ESD in young athletes are hereditary and are usually evident in the post-pubertal age group, pre-participation screening should be applied to young players starting competitive amateur careers.

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