Leading causes of sudden death in young athletes

Dr. Andrés R. Pérez Riera

Sudden cardiac death (SCD) is the leading cause of medical death in athletes; however, many studies are significantly flawed in methodology, making accurate estimation difficult. Incidence studies must have a defined study population and must be stratified by sex and age. The risk of ACS/d in college-aged men is 1 in 35,000 person-years, black men 1 in 18,000 person-years, and High-risk sports include men's basketball, men's soccer, and American football.
Hereditary cardiomyopathies and electrical conditions account for approximately 2/3 of the SCA/d off and can be detected with an ECG. More research is needed to provide more robust estimates.

Concept of Sudden Death (SD) and prevalence in athletes

SD is defined as the death that is not traumatic, not violent, unexpected, which occurs within the first 6h without a prior manifestation of cardiac disease (**Maron 1986**).

Prevalence

Estimated between young athletes of secondary school, as 1 in 200,000 per year (**Maron 1996, 1998**).

The causes of arrhythmic sudden death in young athletes (average age: 17 years)

I. **Entities with structural heart disease (98%)**

1. Hypertrophic cardiomyopathy (HCM) whether in its obstructive form or in its non-obstructive form (33%);
2. Congenital anomalies of coronary arteries with increase of ventricular mass (20%);
3. Tumors or cardiac masses (10%);
4. Aorta rupture due to Marfan syndrome (5%). Mutation in the gene in fibrillin-1 (FBN1), in chromosome 15q21.1 and Marfan-like syndrome with no eye anomalies, mapped in chromosome 3p24;
5. Arrhythmogenic cardiomyopathy (3%). Prevalence of 1 in 15,000;
6. Early atherosclerotic coronary artery disease (2%) by familial hypercholesterolemia and dominant mixed hyperlipidemia by alteration in chromosome 6;
7. Mitral valve prolapse syndrome (MVPS) (2%);
8. Myocarditis (2%);
9. Dilated cardiomyopathy
10. Restrictive cardiomyopathy

11. Familial arrhythmogenic syndrome: ventricular and tachyarrhythmia association (syndrome of Wolff-Parkinson-White), progressive disease of conduction system and cardiac hypertrophy by involvement of regulatory subunit gamma-2 (PRKAG2) of AMP- activated by protein kinase (**Gollob 2002**);
12. Aortic stenosis valvar and sub valvar.

II) **Entities without structural heart disease (2%);**

1) Drug abuse, e.g. anabolic agents,
2) Ventricular pre-excitation of the Wolff-Parkinson-White syndrome type, with anomalous pathway of short refractory period, not detected previously;
3) Cardiac concussion or commotio cordis;
4) Channelopathies.

A) Of the sarcolemma or external channelopathies:

1) congenital long. QT syndrome (LQTS);
2) Brugada syndrome (BrS);
3) Progressive familial heart block type I; progressive "idiopathic" disease of the His-Purkinje system or Lenègre;
4) Genuine idiopathic ventricular fibrillation (GIVF);
5) Mixed forms or with overlapped phenotypic aspects:

5a) BrS and LQT3;
5b) BrS and Lenègre disease;
5c) BrS and SND;

5d) Association of BrS, LQTS and PCD;

6) Some sudden unexpected nocturnal death syndromes (SUNDS);

7) Some sudden infant death syndromes (SIDS).

B) Of the channels of the endoplasmic reticulum or intracellular channelopathies:

1) Catecholaminergic Polymorphic Ventricular Tachycardia (CPVT).