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REVIEW ARTICLE

SUDDEN DEATH IN ATHLETES: ETIOLOGY & PREVENTION

Sudden Death in Athletes: Etiology & Prevention

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INTRODUCTION

The sudden death of a young athlete is a tragic event that has devastating effects on families and communities. Despite public perception to contrary sudden death in young athletes is exceedingly rare. Sudden death in athletes defined as "Natural, unexpected, death within one hour of onset of symptoms and subsequent cardiac arrest." Depending on the underlying cause, sudden death can be divided into sudden cardiac death defined as sudden death from a cardiac cause, and Sudden death due to noncardiac causes, for example, intracranial hemorrhage, epilepsy, pulmonary embolism, or asthma. This subdivision is clinically relevant because cardiac causes are inherited in a significant proportion, whereas noncardiac causes usually are not. Death in absence of a diagnosis despite autopsy is generally termed sudden unexplained death (SUD) or autopsy-negative SUD. In countries where autopsy is not mandatory in the case of Sudden death at a young age, cases in which no autopsy is performed are considered to be SUD. sudden death occurs in approximately 1:200,000 athletes annually. The victim is usually male and, in the US. Two/Third occurs in foot ball and basket ball players. This reflects the popularity of these sports and the number of athletes involves in them and elsewhere in world, soccer is most commonly associated. From static's derived from NCAA data, one death per 25000 collegiate athletes over a five year period was attributable to medical cause.

Many observational studies done on sudden death in athletes for cause other than trauma, Philips et-al reported rate of 1:735000 US Air force recruitment age 17-28 years during training, Van camp et-al reported rate of approximately 7:1000000 and 1:1000000 for male & female high school and college students of athletes activity. Age 35 serves as water shed as to the likely cause of sudden death. Before age 35 congenital abnormalities of heart & blood vessels are major etiology. There are usually asymptomatic prior to the fatal event although not invariably so. After age 35 acquired coronary artery disease predominant

(80%). and this is true regardless of the athlete's former level of fitness.

Screening athletes for disorders capable of provoking sudden death is challenge, because of the low prevalence of disease, and the cost and limitations of available screening tests. Current recommendations for cardiovascular screening call for a careful history and physical examination performed by a knowledgeable health care provider. Specialized testing is recommended only in cases that warrant further evaluation.

ETIOLOGY

There are many causes of nontraumatic sudden death in athletes, in which congenital cardiovascular diseases is leading cause of it, Hypertrophic cardiomyopathy being most Common.

Following are causes of nontraumatic sudden death in athletes -

Table ;1

Likely Etiologies for Sudden Cardiac Death in Young Athletes

Hypertrophic cardiomyopathy

Coronary artery anomalies

Atherosclerotic coronary artery disease

Myocarditis

Other etiologies (less common)

Right ventricular dysplasia

Marfan's syndrome

Conduction system abnormalities

Idiopathic concentric left ventricular hypertrophy

Substance abuse (e.g., cocaine, steroids)

Aortic stenosis

Mitral valve prolapse

HYPERTROPHIC CARDIOMYOPATHY

Hypertrophic cardiomyopathy is an autosomal-dominant congenital disorder characterized by left ventricular outflow obstruction with asymmetric septal hypertrophy and marked disarray of ventricular muscle fibers. In a recent review¹³ of 4,111 subjects in the Coronary Artery Risk Development in (Young) Adults (CARDIA) Study, the prevalence of echocardiographically defined hypertrophic cardiomyopathy was estimated at two per 1,000 young adults. It is thought that this condition could predispose persons to malignant ventricular arrhythmias leading to syncope or sudden death.

Hypertrophic cardiomyopathy is often clinically silent, but a personal or family history of unexplained syncope, especially effort syncope or sudden-death events, is an important clinical clue. Chest radiography may show cardiomegaly, and electrocardiography may show left ventricular hypertrophy or other changes, but results of these tests may also be normal. The diagnosis is best confirmed with two-dimensional and M-mode echocardiography.

CONGENITAL AND ACQUIRED CORONARY DISEASE

Congenital coronary anomalies are multiple, the most common being misplaced aortic ostium, in which the left main and right coronary artery arise from the right sinus of Valsalva. These conditions are difficult to identify unless complaints of early fatigue, angina or exercise-induced syncope lead to a directed evaluation. In one review¹⁴ of 78 cases of sudden death thought to be secondary to autopsy-proven coronary anomalies, 62 percent occurred in asymptomatic persons.

Tragically, acquired premature coronary artery disease can appear in the athlete under age 30. Genetic predisposition plus other risk-factor prevalence can sometimes lead to coronary events resulting from typical atherosclerosis. Attention to risk factors and to the early symptoms of ischemia, angina and other effort-related symptoms should be just as aggressively pursued in younger athletes as in older athletes.

MYOCARDITIS

Acute myocarditis is a rare but potentially devastating condition that is most commonly caused by viruses. Coxsackie B virus has been implicated in 50 percent of cases.¹⁵ Early symptoms, if present, may include

exercise intolerance and congestive heart failure symptoms with dyspnea, cough and orthopnea. Subtle clinical signs include tachycardia in the absence of fever, pulsus alternans and other clinical signs of heart failure (e.g., S3 gallop, soft apical murmur, distended neck veins, peripheral edema). Most patients with myocarditis present with sudden death secondary to a ventricular arrhythmia and had few, if any, prodromal signs or symptoms. In addition, inflammatory coronary artery aneurysms associated with Kawasaki's disease have also been reported as a cause of sudden death.¹⁶

OTHER CONDITIONS

Finally, many other conditions have much less frequently been associated with sudden cardiac death in the young athlete. Marfan's syndrome, with its lethal association with ruptured aortic aneurysms, deserves particular note because of helpful clinical clues¹⁷ that make it a screenable condition. In addition, other preventable conditions should be noted, such as cocaine use (associated with coronary artery spasm) and anabolic steroid use (potential association with hypertrophic cardiomyopathy).^{18,19} Other such conditions include conduction abnormalities, aortic stenosis, idiopathic concentric left ventricular hypertrophy and, possibly, mitral valve prolapse.

SCREENING STRATEGIES

The impact of sudden death in a young athlete during competition always drives the question as to what more could have been done to identify this person who, apparently, was at higher risk. The use of screening tests, however, should be evaluated by epidemiologic criteria for determining effectiveness, not merely by media and/or public consensus.

Attempts have been made²⁰ to put screening strategies for the prevention of sudden death into perspective by estimating disease prevalence. It has been estimated that 200,000 competitive asymptomatic athletes would need to be screened to potentially identify one athlete who would die as a result of competition. [corrected] If we had a tool to screen for sudden death with a sensitivity and specificity of 99 percent, the low prevalence of disease would yield a positive predictive value of only 0.05 percent. In other words, only one positive test out of every 2,000 would be correctly positive, and 1,999 would be falsely positive.

One of the problems with screening athletes is that "abnormalities" detected during examinations may merely be normal variants. The changes that occur in the heart in response to athletic training are known as "the athletic heart syndrome."²¹ The well-trained athlete often demonstrates electrocardiographic, radiographic and echocardiographic changes of cardiac enlargement and enhanced vagal tone. In addition, the clinical examination may demonstrate

bradycardia, S3 and S4 heart sounds, and innocent flow murmurs. While clinical criteria have been developed to assist in distinguishing athletic heart syndrome from pathologic conditions, differentiation of normal from abnormal changes may be extremely difficult.

USE OF ECHOCARDIOGRAPHY

Because hypertrophic cardiomyopathy is the most common cause of sudden death in the young competitive athlete, health care strategy should focus on detection of this disease. A good history and physical examination are accepted as the minimal standard. While the role of echocardiography in routine screening in the asymptomatic athletic population is limited by cost and the low prevalence of the disease, recent evidence suggests that even echocardiography may not always demonstrate hypertrophic cardiomyopathy. Recent molecular genetic studies²⁷ have demonstrated that clinical features such as arrhythmias, myocardial ischemia and/or diastolic dysfunction may be present in patients in the absence of left ventricular hypertrophy as determined by echocardiography. While the reports of these studies clearly state that current practice is far from incorporating genetic testing into routine clinical practice and screening, they concluded that relying solely on echocardiography provides a restricted view of the prevalence and clinical spectrum of hypertrophic cardiomyopathy.

In a recently published detailed study,²⁸ the demographics of sudden death in young competitive athletes was profiled. A review of the records of victims of sudden death revealed that a standard history and physical examination had been completed in 115 of the athletes (158 cases were reviewed). In only four (3 percent) of these athletes was there any suspicion of a cardiovascular problem. In only one athlete was the correct diagnosis, Marfan's syndrome, made—and that athlete did not withdraw from athletic participation.

In 15 of the 158 athletes, symptoms provoked individualized work-ups. These evaluations led to seven correct diagnoses and two disqualifications from competitive athletics. In reviewing cases retrospectively, 31 percent of the athletes with anomalous coronary arteries had symptoms (syncope or dizziness) and just 21 percent of the athletes with hypertrophic cardiomyopathy had symptoms. These medical evaluations failed to identify 47 of 48 cases of hypertrophic cardiomyopathy.

CURRENT RECOMMENDATIONS

The Science Advisory and Coordinating Committee of the American Heart Association (AHA) appointed a panel to develop a consensus recommendation that was recently published.²⁹ Their recommendations are

consistent with the newly published Preparticipation Physical Evaluation Monograph endorsed by the American Academy of Family Physicians.³⁰ The AHA concluded that some form of preparticipation cardiovascular screening for high school and collegiate athletes is justifiable and compelling, based on ethical, legal and medical grounds. The AHA resolved that a complete and careful personal and family history and physical examination designed to identify (or raise suspicion of) cardiovascular lesions known to cause sudden death or disease progression in young athletes is the best available and most practical approach to screening populations of competitive athletes, regardless of age. The AHA additionally recommends that an examination be performed before participation in organized high school and collegiate sports. Screening should then be repeated every two years; in intervening years, an interim history should be obtained.

In addition, the AHA made specific recommendations about the content and performance of the preparticipation cardiovascular examination. The history should include the following: previous occurrence of chest pain; discomfort or syncope; near syncope; excessive, unexpected and unexplained shortness of breath or fatigue associated with exercise; past detection of a heart murmur or increased systemic blood pressure; family history of premature death or significant disability from cardiovascular disease in close relatives younger than 50 years of age; and specific knowledge of the occurrence of certain conditions (e.g., hypertrophic cardiomyopathy, dilated cardiomyopathy, Marfan's syndrome, long QT syndrome or clinically important arrhythmias).

The cardiovascular examination should include the following: precordial auscultation in both the supine and standing positions to identify, in particular, heart murmurs consistent with dynamic left ventricular outflow obstruction; assessment of the femoral arteries to exclude coarctation of the aorta; recognition of the physical stigmata of Marfan's syndrome and brachial blood pressure measurement in the seated position (Table 3).

TABLE 2

AHA Recommendations for Preparticipation Cardiovascular Examinations

History

Previous occurrence of chest pain

Discomfort or syncope

Near syncope

Excessive, unexpected and unexplained shortness of breath or fatigue associated with exercise

Past detection of a heart murmur or increased systemic blood pressure

Family history of premature death or significant disability from cardiovascular disease in close relatives younger than 50 years of age

Specific knowledge of the occurrence of certain conditions:

Hypertrophic cardiomyopathy

Dilated cardiomyopathy

Marfan's syndrome

Long QT syndrome

Clinically important arrhythmias

Examination

Precordial auscultation in supine and standing positions to identify heart murmurs consistent with dynamic left ventricular outflow obstruction

Assessment of femoral arteries to exclude coarctation of the aorta

Recognition of the physical stigmata of Marfan's syndrome

Brachial blood pressure measurement in seated position

AHA = American Heart Association.

The "focused" examination should seek to rule out historical or physical examination features that have been associated with common causes of sudden death in young athletes (Table 4). Athletes who demonstrate abnormalities on either the history or the physical examination warrant a more detailed examination. This evaluation may include electrocardiography, echocardiography, exercise stress testing and/or cardiac catheterization. In addition, the athlete may require temporary disqualification from athletic participation pending further evaluation. Because further testing has certain limitations, it is the authors' opinion that cardiac consultation is warranted in an athlete suspicious for cardiac disease.

TABLE 3

Screening for Sudden Death in Young Athletes

Condition	Historical features	Physical examination
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Hypertrophic cardiomyopathy

Family history of hypertrophic cardiomyopathy, premature sudden death, recurrent syncope and/or lethal arrhythmias requiring urgent management. Personal history of exertional chest pain and/or syncope.

Wide range of auscultatory findings, from normal examination to harsh midsystolic murmur that accentuates with Valsalva maneuver and/or with standing.

Coronary artery diseases, congenital and acquired

Family history of early coronary artery disease, premature death and/or coronary anomalies. Personal history of exercise-induced chest pain, syncope and/or fatigue.

Physical examination is anticipated to be normal.

MYOCARDITIS

Personal history of fatigue, exertional dyspnea, syncope, palpitations, arrhythmias and/or acute congestive heart failure.

Examination may be normal. Palpable or auscultated extra systoles, S3 and/or S4 gallops and other clinical signs of failure should arouse suspicion.

AORTIC STENOSIS

Personal history of exercise-induced chest pain, breathlessness, light-headedness, syncope or dizziness.

Constant apical ejection click. Harsh systolic ejection murmur heard maximally at the upper right sternal border, crescendo/decrecendo profile, normally \geq grade 3.

MARFAN'S SYNDROME

Family history of Marfan's syndrome or unexpected premature sudden death.

Arachnodactyly, tall stature, pectus excavatum, kyphoscoliosis and lenticular dislocation. Murmur of mitral valve prolapse and/or aortic regurgitation.

Note: These conditions may be asymptomatic and may present without any clues on physical examination. The history and physical examination are complementary and not mutually exclusive.

d for preparticipation assessment. Since electrocardiography and treadmill stress testing are plagued by a high degree of false-positive results (primarily because of athletic heart syndrome) most attention in the literature has focused on the use of

echocardiography for diagnosis. The echocardiogram is considered to be sensitive and specific in detecting hypertrophic cardiomyopathy. The principal concerns with echocardiography are the cost and the risk of misdiagnosing a healthy athletic heart and, therefore, restricting an otherwise-healthy athletes.

PREVENTIVE MEASURES

Ensure that athletes with chest pain, shortness of breath, palpitations or passing out undergo a thorough medical evaluation. Train athletes with a graded program gradually building up their fitness level. Keep athletes well hydrated. Respond immediately to an athlete who has collapsed and be aware that the athlete may have suffered a cardiac arrest. Train your staff to perform cardiac life support and have trained staff available at every practice and sporting event. Purchase a defibrillator and ensure it is readily accessible.

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Sudden Death in Young Athletes: Screening for the Needle in a Haystack

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