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Left Ventricle Fibrosis Associated With Nonsustained Ventricular Tachycardia in an Elite Athlete: Is Exercise Responsible? A Case Report

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Objective: To emphasize the potentially harmful effects of high-intensity exercise on cardiac health and the fine line between physiologic and pathologic adaptation to chronic exercise in the elite athlete. This case also highlights the crucial need for regular evaluation of symptoms that suggest cardiac abnormality in athletes.

Background: Sudden cardiac death (SCD) of young athletes is always a tragedy because they epitomize health. However, chronic, high-intensity exercise sometimes has harmful effects on cardiac health, and pathologic changes, such as myocardial fibrosis, have been observed in endurance athletes. In this case, a highly trained 30-year-old cyclist reported brief palpitations followed by presyncope feeling while exercising. Immediate investigations revealed nonsustained ventricular tachycardia originating from the left ventricle on a stress test associated with myocardial fibrosis of the left ventricle as shown with magnetic resonance imaging. Despite complete cessation of exercise, life-threatening arrhythmia and fibrosis persisted, leading to complete restriction from competition.

Differential Diagnosis: Hypertrophic cardiomyopathy, arrhythmogenic right ventricular cardiomyopathy, myocarditis, postmyocarditis, use of drugs and toxic agents, doping, and systemic disease.

Treatment: The arrhythmia could not be treated with catheter ablation procedure or drug suppression. Therefore, the athlete was instructed to withdraw completely from sport participation and to have a medical follow-up twice each year.

Uniqueness: To our knowledge, no other report of left ventricle exercise-induced fibrosis associated with life-threatening arrhythmia in a living young elite athlete exists. Only postmortem evidence supports such myocardial pathologic adaptation to exercise.

Conclusions: To prevent SCD in young athletes, careful attention must be paid to exercise-related symptoms that suggest a cardiac abnormality because they more often are linked to life-threatening cardiovascular disease.

Key Words: myocardial fibrosis, high-intensity exercise, sudden death

Since Pheidippides died just after running 35 km from Marathon to Athens to declare victory over the Persians, sudden cardiac death (SCD) has continued to occur in modern times. These deaths usually are from unsuspected cardiovascular diseases,[1,2] and SCD is the first clinical expression of such a disease in most patients. High-intensity exercise might have harmful effects on cardiac health by potentially generating myocardial fibrosis[3–5] and thereby causing arrhythmias[6] and SCD. The hypothesis for such a pathologic adaptation is emerging and is based largely on postmortem evidence. The purpose of our report was to illustrate the unique case of left ventricle exercise-induced fibrosis associated with life-threatening arrhythmia in a young, elite athlete that led to restriction of competition to avoid exposing the athlete to a very likely competition-induced SCD. In the report, we emphasize the key role of systematic evaluation of exercise-related symptoms that suggest a cardiac abnormality, which warrants exhaustive cardiac evaluation as recommended by current guidelines organizing the standardized procedures of the medical follow-up of athletes.[1,2]

CASE REPORT

Currently involved in the standardized medical follow-up of athletes, our department has examined a highly trained male athlete annually since 1998. This white, 30-year-old athlete began cycling at the age of 10 years and, since at least 1998, had completed an average of 23000 km per year. He had no medical family history of underlying heart disease. No previous concerns were identified from his personal history, and all physical examinations were normal. The athlete did not report taking medication or prohibited substances. His resting blood pressure (both upper extremities) was 125/70 mm Hg. Resting 12-lead electrocardiogram (ECG) showed a training-related incomplete right bundle branch block (QRS duration = 102 milliseconds) associated with sinus bradycardia[7] (39 beats per minute).

From 1998 to 2006, 2-dimensional transthoracic echocardiography performed systematically at 4-year intervals showed intracardiac dimensions consistent with an athlete's heart without evidence of hypertrophic cardiomyopathy.[8] Echocardiographic study revealed a left ventricular wall thickness

(LVWT) of 10 mm, with a ratio of the interventricular wall thickness to the left ventricular posterior wall thickness in end diastole equal to 1. Left ventricular end-diastolic (LVED) and end-systolic (LVES) diameters were 56 mm (29 mm/m^2) and 38 mm (19 mm/m^2), respectively, and left atrium diameter was 43 mm. He did not have mitral valve systolic anterior motion or evidence of left ventricle outflow obstruction. During annual graded exercise testing, the athlete completed a maximum workload of 450 W without symptoms, yet he experienced monomorphic ventricular premature beats (VPBs) during both exercise and recovery. No abnormalities were identified on complete blood profiles (4 times per year), including complete blood count, reticulocyte count, iron panel, C-reactive protein, cortisol, testosterone, and insulin-like growth factor 1.

In 2007, the incremental exercise test showed nonsustained (3 consecutive VPBs) ventricular tachycardia (VT) arising at a workload of 420 W without clinical symptoms. Therefore, we ordered exhaustive cardiovascular examinations, including echocardiography, 24-hour Holter ECG monitoring, and cardiac magnetic resonance imaging (MRI), but we did not find substantial structural or functional disease. According to recommendations, competition was not limited.[9,10]

In 2009, the athlete presented with initial symptoms of brief palpitations followed by presyncope feeling while exercising. He was investigated immediately (within 24 hours) and demonstrated nonsustained VT (7 consecutive VPBs) originating from the left ventricle at a workload of 360 W without clinical symptoms (Figure 1). Electrophysiologic study (EPS) with isoproterenol did not induce VT. Cardiac MRI showed focal fibrosis, which was seen on the late gadolinium enhancement, of the left ventricle (Figure 2) and demonstrated intracardiac dimensions consistent with physiologic remodeling. On MRI, LVED and LVES diameters were 55.45 mm and 37.60 mm, respectively. The LVWT was 11.46 mm, and MRI cardiac mass calculation was 78 g/m^2. End-diastolic and end-systolic volume indices were 121 mL/m^2 and 52 mL/m^2, respectively (Figure 3). Six months later, after complete cessation of exercise, control stress test and cardiac MRI showed persistence of nonsustained VT (3 consecutive VPBs) and myocardial fibrosis. Therefore, the athlete was restricted definitively from competition.[10,11]

DISCUSSION

Researchers have established that regular sport training improves fitness and reduces cardiovascular morbidity and mortality.[12] Therefore, exercise is generally encouraged by the medical community.

The heart is capable of remodeling as a physiologic adaptation to chronic exercise,[13] so structural and electrical cardiac changes usually are assumed to be normal. The latter assumption for a benign adaptation is based mainly on the regression of these changes after cessation of exercise, and the myocyte hypertrophy resulting from myocardial injury repair is thought to be the leading mechanism involved.[14] However, the upper limit of exercise volume for cardiac health is unknown, and the hypothesis that chronic high-intensity exercise might have harmful consequences for the heart is emerging.[3,4] Indeed, the cardiac damage occurring after intensive exercise possibly represents a potential for generation of reactive scar tissue.[5] Therefore, pathologic changes, such as myocardial fibrosis that is associated with an increased risk for arrhythmias,[6] have been described in endurance athletes.[15] The findings of numerous researchers support fibrotic replacement of the myocardium either by biochemical[16] or more often by postmortem evidence,[17] highlighting the potentially deleterious response to exercise in some veteran athletes. In our case report, successive cardiac

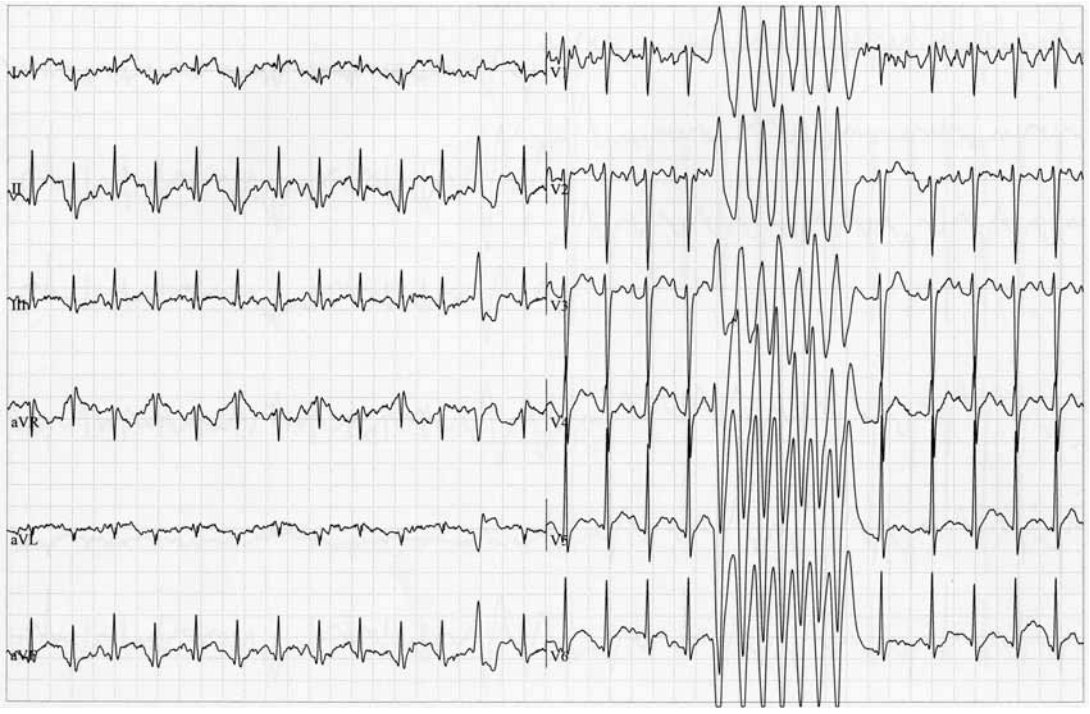

Figure 1. Maximum exercise test was performed soon after the athlete demonstrated his first symptoms of a cardiac abnormality during competition. He had nonsustained ventricular tachycardia (7 consecutive ventricular premature beats) originating from the left ventricle at a workload of 360 W without clinical symptoms. The left ventricle myocardial fibrosis is involved in the generation of ventricular arrhythmia.

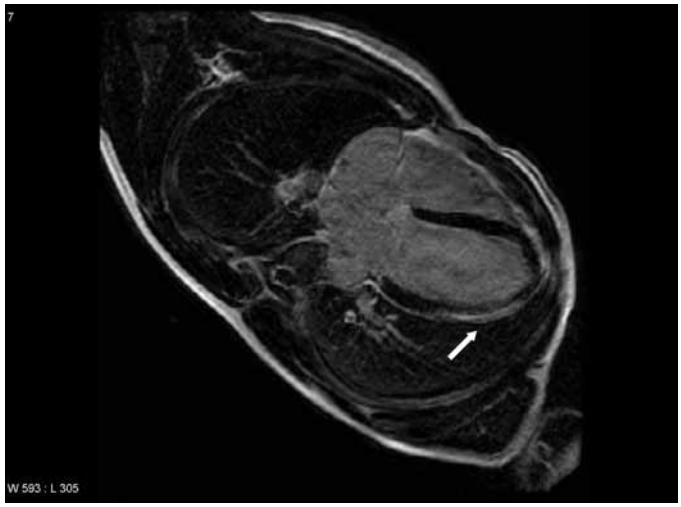

Figure 2. On cardiac magnetic resonance imaging (4-chamber view), note myocardial fibrosis of the left ventricle lateral wall on late gadolinium enhancement (arrow). The fibrosis affected 15% of the left ventricle surface and up to 50% of left ventricle lateral wall thickness and therefore was associated with a dyskinetic segment in systole.

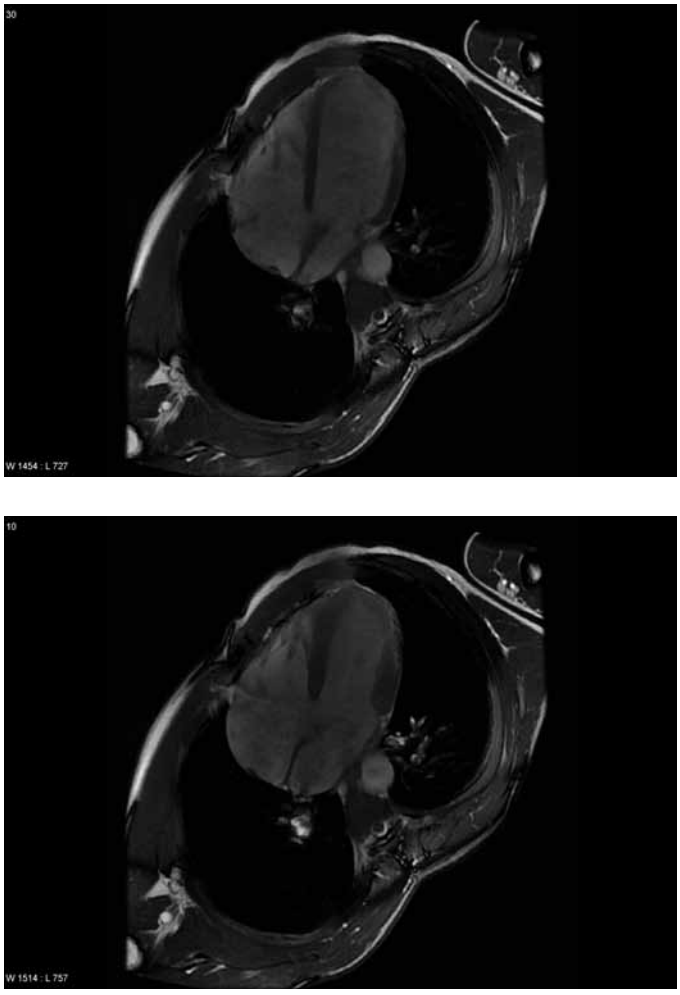

Figure 3. A, End-diastolic, and B, end-systolic images (cardiac magnetic resonance imaging) demonstrated intracardiac dimensions consistent with physiologic remodeling without evidence of hypertrophic cardiomyopathy. Left ventricle wall thickness was 11.46 mm. End-diastolic and end-systolic volume indices were 121 mL/m² and 52 mL/m², respectively.

MRIs clearly showed the development of focal fibrosis of the left ventricle within at least a 2-year period. During this 2-year period, the athlete reported no changes in behavior, especially concerning training and dietary habits. The absence of regression of focal fibrosis despite complete cessation of exercise over 6 months in association with the emergence of nonsustained VT originating from the left ventricle led us to disqualify this young athlete from cycling competition.[10] Despite the desire of the athlete to continue competition, no other solution than withdrawal from sport participation could be proposed. Indeed, EPS did not induce VT, which excluded a catheter ablation procedure and did not represent an indication for use of an implantable cardioverter defibrillator.[10,11] Although some uncertainty remains about the precise diagnosis, any other cause of the fibrosis was excluded. Clinical presentation, multiple blood samples, and exhaustive cardiovascular examinations, including echocardiography, cardiac MRI, and EPS, exhibited no evidence of arrhythmogenic right ventricular cardiomyopathy[18] (according to standardized diagnostic criteria proposed by an international task force[19]), myocarditis (no personal history of previous infectious disease, no viral prodrome, normal white blood cell count and C-reactive protein, and no wall motion abnormalities), or known use of drugs and toxic agents. Therefore, we believe that in this peculiar case a pathologic process of myocardial replacement was involved in the origin of the exercise-induced fibrosis.

The case raised another major point supporting the crucial evaluation of athletes with symptoms that suggest a cardiac abnormality. Sudden cardiac death of a young athlete is always a tragedy, and the preparticipation evaluation should include a careful medical history, physical examination, and sometimes cardiac diagnostic tests to detect underlying potentially lethal cardiovascular conditions, such as hypertrophic cardiomyopathy or coronary artery anomalies.[1,2] Unfortunately, in most cases the first clinical expression of such a disease is sudden death itself. However, careful attention must be paid to symptoms that suggest a cardiac abnormality, especially if they occur during exercise, because they often are linked with life-threatening cardiovascular disease.[20] The patient we described presented with monomorphic VPBs (1998–2006), then 3 consecutive VPBs (2007–2008), and finally brief palpitations followed by presyncope symptoms during a competition. Before symptoms became apparent, the athlete presented with monomorphic VPBs, which are common in the athletic population and usually do not confer an adverse prognosis.[11] However, even an isolated VPB might be an indicator of underlying heart disease, and these apparently benign observations might have been early signs of a pathologic adaptation to exercise. As soon as symptoms became apparent, the athlete was referred promptly to a cardiology department, revealing the nonsustained VT (7 consecutive VPBs) on the exercise stress testing. Clinical evaluation that is carried out as soon as possible after the athlete demonstrates symptoms of a cardiac condition must be approached systematically and thoroughly, permitting the detection of a possible underlying cardiac abnormality that exposes the athlete to a higher risk of SCD.[21] Athletes then might be disqualified from competition, and the responsibility for such a restriction falls to the physician.[22]

CONCLUSIONS

The benefit of exercise in preventing cardiovascular morbidity and mortality is well established. However, circumstantial evidence has suggested that excessive exercise might have harmful effects on cardiac health, sometimes leading to rare but remarkable sudden cardiac events. Our case supports this hypothesis for such a pathologic adaptation; left ventricle exercise-induced fibrosis associated with life-threatening arrhythmia in a young, elite athlete led to complete withdrawal from sport participation. Although some uncertainty remains about the precise cause of the fibrosis, exhaustive cardiovascular examinations did not reveal an underlying cardiovascular disease usually associated with myocardial fibrosis. Furthermore, despite numerous recommendations related to screening for cardiovascular abnormalities in elite athletes, SCD continues to occur most often without previous symptoms. Nevertheless, exercise-related symptoms that suggest a cardiac abnormality sometimes occur and must be investigated immediately to detect a possible heart abnormality that exposes the athlete to SCD. Therefore, health care providers from the athletic trainer to the physician should systematically evaluate symptoms that suggest a cardiac abnormality in the athletic population to prevent SCD.

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