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CASE REPORT: 26-YEAR CARDIOLOGICAL FOLLOW-UP OF A HIGH-LEVEL ATHLETE

RELATO DE CASO: ACOMPANHAMENTO CARDIOLÓGICO DURANTE 26 ANOS DE ATLETA DE ALTO NÍVEL

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ABSTRACT

The athlete's heart performs a set of adaptive changes resulting from the practice of intense and prolonged physical exercise, resulting in clinical, electrocardiographic and echocardiographic alterations. The most relevant changes are myocardial hypertrophy, enlargement of the cardiac cavities, and an increase in cardiac mass. In some cases, these changes can be considered extreme, requiring the elimination of pathological conditions. Through a case report of a high-level basketball player, whose only change in the physical examination was the presence of sinus bradycardia, persistent over 26 years of clinical follow-up, which was investigated through echocardiogram, Holter test and stress test, demonstrating benign and physiological characteristics, we present and discuss the importance of cardiovascular evaluation by a physician with experience in analyzing athletes, capable of distinguishing pathological conditions from physiological adaptations to the high-intensity exercise to which the athlete is subjected, avoiding invasive examinations or treatment of the athlete's heart.

Keywords: Athletes; Bradycardia; Sports medicine.

RESUMO

O coração do atleta engloba um conjunto de alterações adaptativas resultantes da prática de exercício físico intenso e prolongado, promovendo alterações clínicas, eletrocardiográficas e ecocardiográficas. As alterações mais relevantes são a hipertrofia miocárdica, o alargamento das cavidades cardíacas e o aumento da massa cardíaca. Em alguns casos, essas alterações podem ser consideradas extremas, exigindo que as condições patológicas sejam eliminadas. Por meio de um relato de caso de um atleta de basquetebol de alto nível cuja única alteração no exame físico era a presença de bradicardia sinusal, persistente ao longo de 26 anos de acompanhamento clínico, investigada através de ecocardiograma, Holter e teste de esforço, demonstrando caráter benigno e fisiológico, apresentamos e discutimos a importância da avaliação cardiovascular por médico com experiência na análise de atletas, capaz de distinguir as condições patológicas das adaptações fisiológicas frente ao exercício de alta intensidade ao qual o atleta é submetido, evitando exames invasivos ou tratamento do coração do atleta.

Descritores: *Atletas; Bradicardia; Medicina esportiva.*

INTRODUCTION

The athlete's heart involves a set of adaptive changes, resulting from intense and prolonged physical exercise that causes clinical, electrocardiographic, and echocardiographic alterations. The most relevant changes are myocardial hypertrophy, enlargement of cardiac cavities, and increased cardiac mass. In some cases, these changes can be considered extreme and require ruling out possible pathological conditions.[1,2] It is important to highlight that the adaptive changes due to physical exercise are reversible with physical deconditioning.[3]

The practice of isotonic exercise leads to an increase in cardiac and maximal oxygen consumption and mean systolic blood pressure, and a decrease in peripheral vascular resistance and diastolic blood pressure.[4-6] The heart is capable of adapting to higher oxygen demand by increasing the systolic volume and, subsequently, the cardiac output via two mechanisms: increasing the end-diastolic volume (by the Frank–Starling's law) and decreasing the end-systolic volume (due to increased contractility).[4,7,8] In the long-term, the volume overload, imposed on the heart, increases the internal diameter and wall thickness of the

left ventricular cavity, which influences the development of eccentric ventricular hypertrophy.[7]

In contrast, isometric exercise significantly increases the systolic, mean, and diastolic blood pressures, without substantially changing peripheral vascular resistance. A higher pressure aims to compensate for higher intramuscular pressure caused by a significant increase in muscle tone (which is typical of this type of exercise), which could compromise the blood supply to the skeletal muscles.[9,10] To compensate for this post-load increase, a significant increase of the intraventricular pressure occurs, which is responsible for the development of concentric hypertrophy.[11,12]

Regardless of the type of sports, the athlete develops cardiac changes, including dilation and increased thickness of the left ventricular wall.[13] The intensity of these changes varies between athletes. Non-genetic factors, such as the type of sports, sex, age, and athlete's body surface area, account for 75% of the change. The remaining 25% occur due to unknown factors, although it is believed that these are related to genetic aspects.[8] According to Pelliccia, hereditary factors are involved in cardiovascular alterations by genetically controlling the response to exercise and genetic predisposition to performing a more intense physical exercise, resulting in better performance during competitions. Thus, genetic influence could aid in explaining the significant differences in the intensity of cardiovascular changes and in the performance of athletes with the same anthropometric characteristics, undergoing the same type/intensity of training.[5]

One of the most important features of the athlete's heart is the increase in the parasympathetic tonus, with an associated decrease in the sympathetic tonus. This feature accounts for findings that are often observed in the athlete, such as resting bradycardia, sinus arrhythmia, and delays in atrioventricular conduction.[8,14]

The systolic volume of athletes is higher compared with non-athletes because exercise requires higher cardiac output. However, the cardiac output of both athletes and non-athletes is similar at rest. Thus, the athlete's heart rate decreases.[8,14,15]

Electrocardiographic changes could be found in up to 80% of healthy athletes, being more common in males and practitioners of isotonic exercise.[8,16] Common findings are increase in the QRS complex voltage and early repolarization patterns. Changes can be explained through an increased parasympathetic tonus, structural remodeling, early repolarization of ventricles, and lower layer of adipose tissue present in these individuals.[17,18]

This article aimed to report functional physiological changes in relation to physical conditioning, which should not be considered pathologic during an athlete's evaluation.

CASE REPORT

The case presented involves a male basketball athlete, who started competitive training for 5–6 hours daily, 6 times per week at the age of 15 years. At the age of 19 years, he became a part of the Paulista and Brazilian basketball national team. After selection into the Brazilian basketball team, he won the World Championships of Basketball held in Chile and Sao Paulo, in 1959 and 1963, respectively. He won a bronze medal in the 1960 and 1964 Summer Olympics in Rome and Tokyo, respectively. He also won the bronze medal in the Pan American Games in 1959. Still maintaining a competitive level of physical activity, at the age of 28 years, he reduced the training frequency and intensity to three to four times a week, with a daily load of up to 5 hours.

The patient began to be followed-up at the Sports Cardiology Outpatient Clinic of the Dante Pazzanese Institute of Cardiology when he was 51 years old and still practicing basketball at a competitive level. He was asymptomatic, weighing 86 kg, measuring 186 cm, in height, eupneic, presenting physical examination without alterations, except for a resting heart rate of 36 bpm. With the aim to evaluate his bradycardia, he underwent 12-lead electrocardiography at rest (Figure 1), echocardiography (Table 1 and Figure 2), Holter (maximum HR of 112 bpm, mean of 56 bpm, and minimum of 38 bpm, sinus rhythm, 153 polymorphic and isolated ventricular extrasystoles and one paired episode, 12 polymorphic and isolated supraventricular extrasystoles), and stress tests via Bruce's protocol with electrocardiography at rest. The results revealed sinus bradycardia (HR, 50 bpm) with an early ventricular repolarization pattern, an effort phase showing a satisfactory increase in peak heart rate of 162 bpm (96% of the recommended maximum HR), no suggestive signs of ischemia, and the disappearance of the early ventricular repolarization pattern, which was again manifested at the end of the recovery phase. In addition, laboratory tests showed no changes.

At 78 years of age, the patient showed no cardiovascular complications. He remained asymptomatic, without showing any abnormality during physical examination, except for sinus bradycardia. Routinely, the patient was asked to undergo 12-lead electrocardiography (Figure 3), echocardiography (Table 2 and Figure 4), and stress tests (modified Ellestad's protocol). This started from HR of 48 bpm at rest to 128 bpm. This corresponds to 90% of maximal HR recommended without abnormalities suggestive of myocardial ischemia, with the occurrence of ventricular and supraventricular extrasystoles, which are rare during exercise and frequent during recovery, with two episodes of non-sustained supraventricular paroxysmal tachycardia. The current Holter showed a maximum, mean, and minimum HR of 117, 57, and 36 bpm, respectively, with 235 supraventricular extrasystoles, seven of which were paired, and six episodes of non-sustained paroxysmal supraventricular tachycardia (maximum 11 beats), variable PR interval with periods of first-degree atrioventricular block, and ten rare, isolated and polymorphic extrasystoles.

Figure 1. The 12-lead electrocardiogram of a 51-year-old patient. Sinus rhythm, SÂQRS −30°.

Table 1. Results of echocardiography performed on May 21, 1991 (51-year-old patient).

Aortic diameter	30mm
Left atrium diameter	34.4mm
Left ventricular systolic diameter	35.7mm
Left ventricular diastolic diameter	52.9mm
Interventricular septum	13.9mm
Left posterior ventricular wall	13.9mm
Right ventricular systolic diameter	12.8mm
Right ventricular diastolic diameter	15.2mm
Left ventricular ejection fraction	60%
Others	- Concentric hypertrophy of the left ventricle.
	- Absence of myocardial contractility abnormalities.

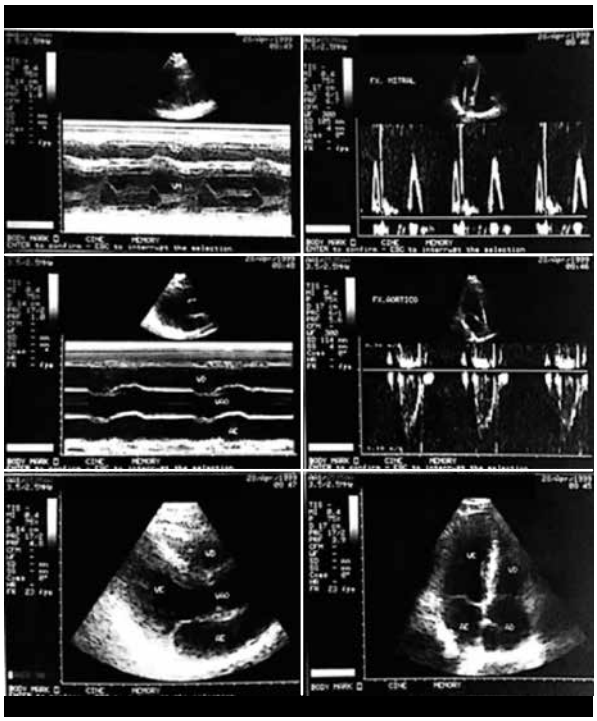

Figure 2. The results of echocardiography performed on May 21, 1991 (51-year-old patient). Information see Table 1.

Figure 3. The 12-lead electrocardiogram of a 78-year-old patient. Sinus rhythm, SÂQRS −30°.

METHODS

All data contained in this study were extracted from the patient's medical record. This contained data regarding all outpatient visits to the hospital, as well as the complementary tests (electrocardiogram, stress test, echocardiogram, and Holter), as previously mentioned. The medical record included 55 years of patient's follow-up.

DISCUSSION

Prolonged and high-intensity physical activity can lead to a series of anatomical and functional changes, which aimed to result in physiological adaptation to the effort of the cardiovascular system. These changes can be objectively reported through the visualization of electrocardiographic and/or echocardiographic alterations, indicating a condition known as the athlete's heart. This can be considered a pathological condition, without careful anamnesis. The present report highlights the importance cardiovascular evaluation of an athlete being performed by an experienced physician, who is capable of distinguishing pathological conditions from physiological adaptations to high intensity-exercise. This allows avoiding invasive examinations or unnecessary treatment for the athlete's heart.[19] Such treatments are of utmost importance because silent cardiovascular diseases predispose athletes to sudden death and have been documented in several published records.[1] The frequent finding of benign abnormalities in the electrocardiograms of athletes contrasts with important rare cardiovascular diseases and the even more uncommon incidence of sudden death in this population.[2,9,19]

The correct interpretation of the clinical history and complementary examinations prevents a wrong pathological diagnosis.[15,16] Indeed, when an athlete is diagnosed with a potential cardiovascular disease, which reflects only the presence of benign adaptations related to the practice of physical activity, a cascade of psychological, social, and financial implications appear, as well as possible unnecessary disqualification.[19]

Electrocardiographic changes, as those shown by the patient, are present in most athletes, although their frequency and intensity vary according to the sport modality. Manifestations of increased vagal tonus, such as bradycardia/sinus arrhythmia, first-degree atrioventricular block, and early repolarization, are the most obvious in the physiological spectrum. In addition, anatomical adaptations regarding

Table 2. Results of echocardiography performed on December 21, 2016 (78-year-old patient).

Aortic diameter	36
Left atrium diameter	42
Left ventricular systolic diameter	40
Left ventricular systolic diameter	60
Interventricular septum	11
Left posterior ventricular wall	10
Right ventricular systolic diameter	-
Right ventricular diastolic diameter	26
Left ventricular ejection fraction	61%
Others	- Eccentric hypertrophy of left ventricle.
	- Change in the relaxation of the left ventricle.

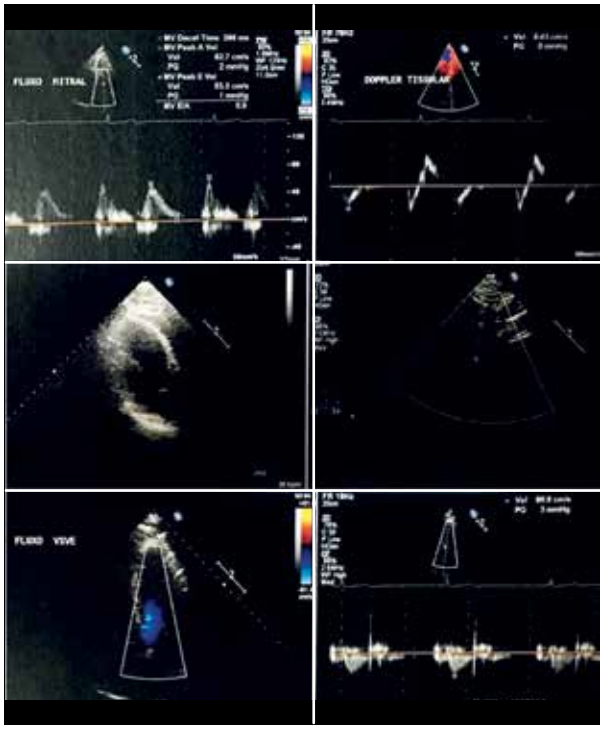

Figure 4. Results of echocardiography performed on December 21, 2016 (78-year-old patient). Information see Table 2.

ventricular remodeling, such as myocardial hypertrophy, can be shown by voltage criteria.[8,15,19]

According to Brazilian guidelines,[19] a stress test can contribute to the diagnosis and prognosis of a cardiovascular disease in an asymptomatic patient and should always be performed in symptomatic athletes or those practicing high-intensity physical activity. For our patient, the indication sought to exclude also coronary artery disease, which is the main cause of death in individuals older than 35 years. The proper chronotropic response presented by the patient justifies that the bradycardia from the vagal tone that was found, which ruled out the possibility of a cardiac conduction system disease.

Although controversial, due to cost-effectiveness evaluation, the indication for an echocardiogram in the pre-participation and during the monitoring evaluation relies on the early identification of changes that may increase the risk of morbidity and mortality while practicing sports. In addition, when suspecting a pathological condition, investigations should persist until all doubts regarding the case are clarified.

There is robust evidence that alterations related to the athlete's heart are benign and self-limited, regressing within a year,[20] when applying physical deconditioning. However, according to the work published by Azevedo,[21] in about one-fifth of high-level athletes, the athlete's heart is still found even after this period, possibly due to having maintained a high rate of recreational sports activity. However, the literature is scarce in describing the long-term follow-up of these patients. This article reinforces that the adaptations to intense physical training are not associated with adverse cardiovascular outcomes even after a long monitoring period.

CONCLUSION

This article describes a case of long-term outpatient follow-up of a patient dedicated to sports. It reports cardiac changes with physical conditioning and relates them to the spectrum of physiological adaptive alterations, which is established in medical literature as a condition known as the athlete's heart. Increased myocardial mass, enlargement of cardiac cavities, and predominance of cholinergic activity on the heart, demonstrated mainly by the presence of bradycardia, were observed. Such changes should be distinguished from pathological conditions, such as systemic arterial hypertension and hypertrophic cardiomyopathy. Thus, our report highlights the importance of evaluation of athletes being performed by experienced medical professionals.

CONFLICTS OF INTEREST

The authors declare that they have no conflicts of interest in conducting this study.

AUTHORS' CONTRIBUTIONS: Each author contributed individually and significantly to the development of the manuscript, participating actively in its preparation, collection of clinical data, bibliographic research, manuscript revision and intellectual concept of the study.

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