REVIEW ARTICLE

Atrial Fibrillation in Athletes: Is Adaptability a Synonym of Risk?

Fibrilación auricular en el atleta: ¿Adaptabilidad es sinónimo de riesgo?

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ABSTRACT

Background: Atrial fibrillation (AF) is the most common cardiac arrhythmia and the most interesting due to its high morbidity and mortality. It is associated with cardiovascular risk factors and structural heart disease, and its prevalence increases with age. The incidence is higher in high-intensity athletes, with a pathophysiology different from conventional AF, from the basis of an adaptive response to an excess of sustained exercise over time. There is a clear difference between sexes.
New diagnostic tools are on the horizon, such as smart watch monitors and quantification of cardiac fibrosis with nuclear magnetic resonance imaging, which will be very useful. Treatment of AF is a great challenge due to the scarce evidence and the psychosocial impact caused by stopping training. Pulmonary vein ablation is currently the definitive treatment of choice. Calcium channel blockers seem to be the best option for HR control; nevertheless, more studies are still necessary. The initiation or not of anticoagulation also remains a question mark.

Key words: Atrial Fibrillation - Physical Endurance - Athletes - Fibrosis

RESUMEN

Introducción: La fibrilación auricular es la arritmia cardíaca más frecuente y de mayor interés debido a su alta morbimortalidad. Se encuentra asociada a los factores de riesgo cardiovascular, a la enfermedad cardíaca estructural y su prevalencia aumenta conforme a la edad. Su incidencia es mayor en deportistas de alta intensidad, y se plantea una fisiopatología diferente a la convencional, desde la base de una respuesta adaptativa hasta un exceso de ejercicio sostenido en el tiempo. Se evidencia además una clara diferencia entre sexos.
Nuevas formas de diagnóstico se avizoran en el horizonte como el monitoreo con relojes inteligentes y la cuantificación de la fibrosis cardíaca con la resonancia magnética nuclear, que serán de gran utilidad. Su tratamiento es un gran desafío debido a la escasa evidencia y al impacto psicosocial que produce el cese del entrenamiento. El tratamiento definitivo y de elección hoy en día es la ablación de las venas pulmonares. Los bloqueantes cálcicos parecieran ser la mejor opción para el control de la frecuencia cardíaca, aunque aún son necesarios más estudios. Permanece además como interrogante el inicio o no de la anticoagulación.

Palabras clave: Fibrilación Atrial - Resistencia Física - Atletas - Fibrosis

INTRODUCTION

Atrial fibrillation (AF) is the most common cause of hospitalization due to cardiac arrhythmias in clinical practice. (1) It can be described or classify according to the duration of the episode (Table 1). (2) In general, there is an association between AF and structural heart disease. (2,3)

Morbidity and mortality are high due to its complications, as stroke and heart failure. The prevalence of AF varies according to different factors as the population analyzed, the geographic distribution, age and sex, and is 1.5 times more common in men than in women. (2,3)

Historically athletes are symbols of health, so the association of exercise with the arrhythmia represents a contradiction in relation to the cardiovascular benefits of physical activity. (4)

Atrial fibrillation in high-intensity endurance athletes makes us think about the concept that even healthy behaviors may have detrimental effects when they are performed in excess.

The evidence

The practice of high-intensity endurance exercise over long periods of time, defined as at least 1500 hours of lifetime training, has been associated with an in-

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Table 1. Classification of atrial fibrillation

PAROXYSMAL	Atrial fibrillation that terminates spontaneously or within intervention within 7 days from onset; the episodes may recur with variable frequency
PERSISTENT	Atrial fibrillation that lasts more than 7 days
LONG-STANDING PERSISTENT	Atrial fibrillation that lasts more than 12 months
PERMANENT	Applies when a joint physician/patient decision has been made to accept the presence of atrial fibrillation and stop further attempts to restore and/or maintain sinus rhythm
NON-VALVULAR	Atrial fibrillation in the absence of rheumatic mitral valve disease, a prosthetic heart valve, or mitral valve repair

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creased risk of AF, particularly paroxysmal, both in professional athletes and in non-professional subjects with high-intensity training. The risk in athletes with lifetime training > 4500 hours may reach 82%. This has been observed particularly in runners, cyclists and skiers. (5,6) Elite athletes can also be defined as those performing at a competitive level with a maximal oxygen uptake (VO_2max) > 60 mL/kg/min (normal value) with at least 10 years of systematic training and a large metabolic turnover > 25 metabolic equivalents (METs) hours/week. (7)

Athletic training is now recognized as a component of what has traditionally been considered "lone" or "idiopathic" AF and may be present in up to 40% of this group. (8)

Common electrocardiographic patterns in athletes include sinus bradycardia or sinus arrhythmia, J-point elevation with ascending ST segments, first degree atrioventricular (AV) block, voltage criteria for left (LVH) and right ventricular hypertrophy, and incomplete right bundle branch block. Some athletes demonstrate a nodal rhythm or Mobitz type 1 second degree AV block at rest. (9)

The frequency of AF is 2 to 10 times greater in athletes than in sedentary individuals. (4) The prevalence of AF in veteran runners has been reported to be 5.3% and is greater in cross-country skiers. By contrast, the prevalence is very low in young elite endurance athletes (mean age 24 ± 6 years). The diagnostic efforts should mostly focus on middle-aged long-term high intensity male athletes (45–65 years). (5)

There is evidence supporting these findings. The Physicians' Health Study showed that vigorous exercise was associated with an increased risk of developing AF. The Norwegian Longitudinal Study reported a prevalence of AF in the general population of 0.4% in men and 0.2% in women, and the risk was greater in those performing physical activity. In a cohort of cross-country skiers, the investigators noted a 26% increase in the risk of AF for every decade of training. (4) One meta-analysis indicates that the risk of AF development in athletes might be significantly higher than in not athletes or the general population. There was a consistent finding of high OR in most of the studies, which were conducted in different countries and in various types of heavy endurance practicing athletes, with an overall OR of 5.29 (95% CI, 3.57-7.85; $p < 0.001$). (10) Another meta-analysis showed that the risk was 5 times greater in athletes compared with controls. The evidence demonstrates that both vigorous physical training and lack of physical activity are associated with higher risk of AF. In contrast, habitual moderate physical activity is associated with reduced risk. These observations are based on the concept of a "J-shaped" pattern describing the relationship between exercise and AF. (4)

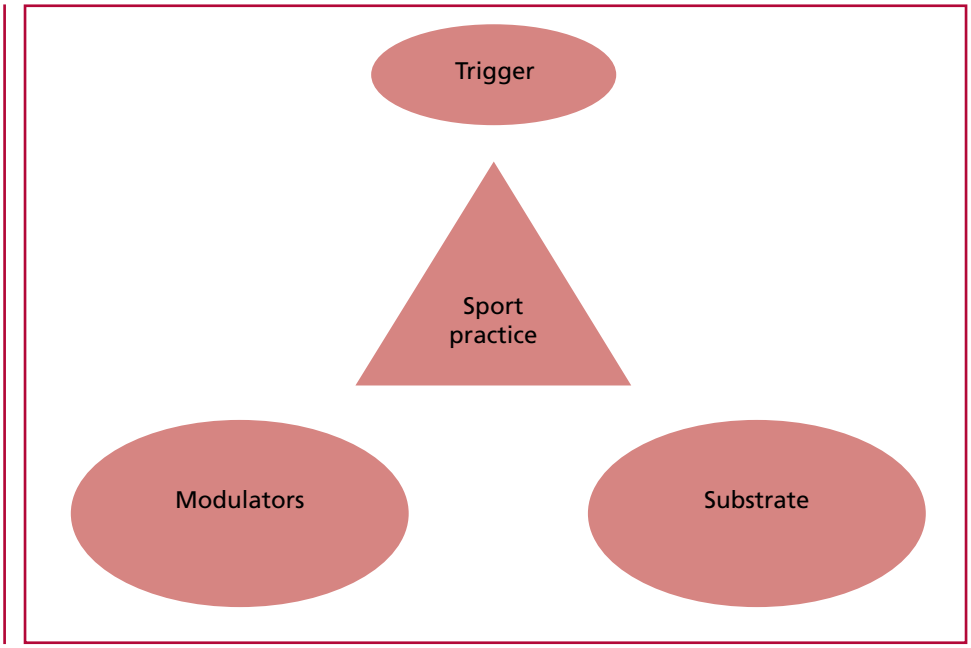
In a cohort of 16 621 participants there was a significant association between vigorous physical exercise and higher risk of AF development in male patients > 50 years; yet the association was less clear in women. (11) A meta-analysis that included 656 720 subjects demonstrated a sex-specific effect. The results of the pooled analysis of such studies clearly demonstrated that low or minimal physical activity increases the incidence of AF in men and women > 40 years from different race, ethnicities and geographic locations. Moderate-intensity exercise was observed to be protective in both genders. The pooled analysis revealed the association of intense physical activity with reduced risk of AF in women and significantly high risk in men. (12)

Pathophysiology

It is well known that AF depends on triggers, substrates and modulators, and these factors may be present in association with physical activity. This interaction is described in the classical Coumel´s triangle. (Figure 1). (13) Thus, in the specific case of AF associated with endurance sport practice, the trigger (increased pulmonary vein ectopy) acts on a substrate (atrial pressure and volume overload, with myocyte hypertrophy and fibrosis), in the presence of a modulator: increased vagal

Morphological and functional differences between left ventricle (LV), right ventricle (RV) and left atrium (LA) account for the different response to high physical activity load. (14) Biatrial dilation is an adaptation within the athlete's heart. (15) It is unknown whether atrial dilation geometry differs in athletes and in patients with structural heart disease. At a similar degree of atrial dilation, atrial function seems to be preserved in athletes, but not in patients with structural heart disease. The atrial morphology makes it particu-

Fig. 1. Coumel´s triangle



larly vulnerable to hemodynamic abnormalities; exercise induced volume overload promotes atrial dilation and increases wall stress, contrary to what happens in the LV. This adds to differences in the molecular characterization of atria and ventricles. Indeed, atrial fibroblasts show an enhanced reactivity to pathological stimuli compared with ventricular fibroblasts, resulting in a remarkably larger atrial than ventricular fibrosis burden upon the instauration of non-ischemic heart failure in animal models. All these data suggest that the atria have greater sensitivity to the hemodynamic overload than the left ventricle, potentially justifying that the atria are primarily affected by strenuous physical activity. (14)

Micro-RNAs (miRNAs) are important mediators of pro-arrhythmogenic remodeling and have hence been proposed as potential biomarkers of AF. Blood levels of miRNAs after a marathon showed more pronounced changes in trained 'elite' runners compared with baseline values in less trained 'non-elite' runners. This could explain, at least in part, the discrepancy between beneficial moderate exercise and 'potentially harmful strenuous endurance exercise. Such miRNAs could serve as biomarkers of pro-arrhythmogenic signaling leading to atrial enlargement after long-term strenuous endurance exercise. (5)

In studies using cardiac magnetic resonance (CMR) imaging , several authors analyzed the presence and distribution of late gadolinium enhancement and found that the rate of myocardial damage was 3 times higher in runners than in control subjects, and a high prevalence (50%) of myocardial fibrosis in healthy, asymptomatic, veteran, male lifelong athletes, compared to zero cases in age-matched veteran controls and young athletes. (16)

Low rest heart rate (HR) in athletes is due to increased vagal tone. The intrinsic change that can occur in the sinus node of endurance athletes might be due to the remodeling of the ion channels that govern pacing. Athletes seem to be more affected by autonomic influence than sedentary individuals, owing on the one hand to a dominant vagal tone at rest or during low-intensity physical work, and on the other hand to bursts of high sympathetic activity during acute strenuous exercise endurance, high-intensity training sessions and competitions. Together with oxidative and metabolic stress, inflammation is indeed a main contributor to atrial electro-anatomical remodeling and extracellular tissue formation, promoting the development of AF. (5) Several studies have demonstrated an increase in inflammatory markers such as cytokines interleukin 1and 6 and C-reactive protein in response to intensive or prolonged endurance exercise. (17) Although regular physical activity has been demonstrated to yield a systemic chronic anti-inflammatory effect, each exercise bout disturbs the inflammatory balance and prompts a pro-inflammatory status with transient increase in neutrophil count and release of pro-inflammatory cytokines that affect the myocardium. (14) Tumor necrosis factor alpha (TNFα) is a key factor in the genesis of AF induced by high-intensity endurance exercise. (6) Atrial fibrosis might play a key role in the genesis of AF. (5)

In 1994 Coumel suggested that the duration of the atrial refractory period, its dispersion and adaptation to HR are the parameters that correlate with the spontaneous occurrence of AF in a condition called "atrial vulnerability". Even normal atria can fibrillate if the disturbance produced by the autonomous nervous system is strong enough. (18) He demonstrated in experimental studies that shortening of the action potential and refractory period creates heterogeneity across the

tissue and a substrate for re-entrant arrhythmogenesis. Vagal stimulation causes release of acetylcholine, which acts predominantly on M2 receptors, activating the G protein-activated inwardly rectifying potassium channels and producing shortening of action potential duration and effective refractory period. The non-uniform distribution of nerve endings might generate AF. Vagal stimuli such as eating, relaxation following exercise, sleeping, and alcohol consumption have been implicated. (19)

Athletes who are involved in vigorous exercise can present dehydration, and acid-base and electrolyte disorders which may also contribute to AF. (6) Atrial fibrillation is commonly triggered by focal ectopic discharges near the four pulmonary veins (PVs). It has been postulated that increased sympathetic tone during physical activity can trigger atrial ectopy. (6) In patients with focal ectopy at those levels, sustained episodes of arrhythmias are mainly dependent on variations of the autonomic tone, with vagal predominance. (20)

Sports supplements are commonly consumed and professional athletes occasionally use illicit drugs to improve performance. Isolated cases of arrhythmias including atrial fibrillation have been reported with most of them. The suggested mechanism is genetic susceptibility exaggerated by autonomic modulation of this population. (6)

Diagnosis

As up to one third of the patients are asymptomatic, the diagnosis may be hampered. (7) The diagnostic efforts for identifying AF associated with physical activity should focus on middle-aged long-term endurance male athletes (45–65 years), especially those who have been engaged at the 'elite' or competitive level for years. The typical clinical profile is a male young athlete usually in his forties or fifties with a history of long-term regular physical activity who is still regularly involved in this type of exercise (generally runners, cyclists or cross-country skiers) on almost all days of the week and with an energy expenditure of at least 8 METs per day. Atrial fibrillation is usually paroxysmal, in the form of self-limited acute crises that progressively increase in duration. Episodes usually occur at night or after meals but can sometimes occur during exercise. The population presents with what is known as "athlete's heart". All these characteristics have derived in the creation of a new syndrome, paroxysmal atrial fibrillation in young and middle-aged athletes (PAFIYAMA), with defined diagnostic criteria. (5)

Major criteria:

- Onset as paroxysmal atrial fibrillation
- Age ≤ 60 years, male sex
- Prolonged practice of strenuous endurance exercise ≥6 to 8 hours/week with intensity 60% of maximum HR, for > 6 months
- Left ventricular ejection fraction ≥ 55%

Minor criteria:

- J-point elevation at ≥ 0.1 mm in 2 leads
- T-wave inversion in 2 leads
- Increased vagal tone (sinus bradycardia, prolonged PR interval, first degree AV block)
- LA enlargement
- Left ventricular hypertrophy
- Normal, or even supranormal, diastolic function

The diagnosis is made in the absence of common AF risk factors: overweight or obesity, hypertension, diabetes and smoking habits. The following conditions should also be excluded: metabolic or hormonal diseases (hyperthyroidism, pheochromocytoma), cardiomyopathies, pericarditis, coronary artery disease, Wolff-Parkinson-White syndrome, Brugada syndrome, long QT syndrome, arrhythmogenic cardiomyopathy or catecholaminergic ventricular tachycardia, use of performance-enhancing agents or illicit drugs, obstructive sleep apnea and electrolyte abnormalities.

Vagal activation accounts for 70% of AF cases (mostly asymptomatic episodes) even immediately after exercise. Adrenergic AF occurs in younger athletes with palpitations limiting functional capacity. (8)

The diagnosis of AF demands documentation of the arrhythmia, which may require prolonged monitoring in this population. For this reason, ''patch" devices, or smartphone-enabled rhythm recorders currently in clinical use in Europe and USA may play a major role. (8)

The athlete's heart is characterized by global remodeling of the four chambers with preserved biventricular systolic function, or at the lower limit of normal. Ventricular relaxation is normal or even better than in healthy people who do not exercise regularly. (21) Left atrial enlargement and atrial wall stretching could be directly related with lifetime training hours. The association between LA size and the incidence of AF is controversial but is closely related with the up-regulation of miRNAs. (5) Despite left atrial size and volume are increased in athletes, the mechanical function measured by speckle-tracking echocardiography does not seem to be impaired. (5)

Left ventricular and RV volumes and diameters as well as global and regional contractile function can be accurately assessed with steady state free precession CMR imaging, which allows not only the evaluation of induced changes in the athlete's heart by training, but also the structural changes upon its cessation. Late gadolinium enhancement can also differentiate between different patterns of myocardial involvement (ischemic vs. non-ischemic, subendocardial involvement, etc.). A more recent and promising methodology, T1 mapping, provides the assessment of diffuse myocardial fibrosis using quantification of the extracellular volume from pre- and post-contrast T1 mapping. (22)

Treatment

There are no clinical trials evaluating the different strategies for this entity; consequently, the clinical

management is based on extrapolation from "non-athletic" populations, observational data, and expert opinion. (4) A common recommendation is to reduce or discontinue physical activity for 3 months to evaluate the association between AF and exercise. (1) In a small study of 30 athletes followed-up for 9 years, 30% remained without symptoms after stopping exercise activity. (23) Therefore, this is indicated by the Study Group on Sports Cardiology of the European Society of Cardiology for Cardiovascular Prevention and Rehabilitation. (24) The second therapeutic step is HR control or rhythm control. Heart rate control is preferred in patients with few symptoms or in those who are asymptomatic. This strategy has the disadvantage that the drugs used, as beta-blockers (BB) or calcium channel blockers, are poorly tolerated due to their negative chronotropic and inotropic effects and, consequently, decrease sports performance. (4) As bradycardia with nocturnal AV block may contraindicate beta blockers, calcium antagonists may be a preferred option. (15) Furthermore, BB use in professional athletes is banned by the World Anti-Doping Agency for certain sports (8,25) which suggests calcium channel blockers for HR control.

Antiarrhythmic drugs such as amiodarone and flecainide could provide benefit in the short term, but their long-term adverse effects and pro-arrhythmic risk discourage their use. (5) The pill-in-the-pocket approach with flecainide or propafenone has been used in symptomatic episodes. (15) Direct-current cardioversion may be an option, although considering the bursts of hyperadrenergic activation, it may not be a definitive treatment and there may be a high risk for recurrence. (26)

Circumferential pulmonary vein ablation (CPVA) as a therapeutic option is increasing in middle-aged athletes with paroxysmal AF without structural heart disease; the procedure is safe and efficient. (5)

The impact of radiofrequency pulmonary vein ablation (RPVA) was evaluated in a 2-year study involving 22 athletes who remained in active competition. At the end of the follow-up period, all the individuals improved their maximal exercise capacity, could reinitiate their competitive sport activities and remained free from treatment and asymptomatic. Other aspects also improved, as mental health, social functioning and role limitation due to emotional problems, among others. (27) More recently, the efficacy of CPVA in 94 athletes was compared with 41 controls with mean age of 51 years. When follow-up was completed, 85% of the athletes were free from the arrhythmia versus 87% of subjects in the control group, demonstrating that CPVA is a safe and efficient option for athletes. (28) Circumferential and segmental pulmonary vein ablations are established treatment strategies for AF. Both ablation techniques require the use of a radiofrequency current to anatomical structures that are close to autonomic ganglia. (29) In this setting, one novel and promising idea is the chemical ablation at the site of the ligament of Marshall, a site of prominent autonomic innervation and potential electrical bridge between the PVs and myocardium. (19)

Initiation of anticoagulation therapy should be evaluated. The risk of ischemic stroke is assessed with the same tools used in the general population. It is better not to use the classical bleeding scores in athletes and rather consider the type of exercise practiced (dynamic versus static component). (2,4,30) For example, golf is a sport with low dynamic and static components; martial arts, weight lifting or sailing are sports with high static component and low dynamic component; soccer, squash and long distance running, are sports with high dynamic component and low static component; finally, boxing, cycling or decathlon have high dynamic and high static components. Other sports show combinations of moderate intensity of both components (for example, rugby and synchronized swimming) or moderate intensity of one component with high or low intensity of the other component.

If anticoagulation is used, athletes should be restricted from participating in high-impact contact sports (as skiing and mountain biking). (5) This is recommended by the American Heart Association scientific statement. (31)

It is necessary to evaluate what the continuity of competitive activity represents for a professional athlete in terms of his or her quality of life, mental health and role in society.

In 2001 a study evaluated the effect of injury severity and training time in elite collegiate athletes and showed that the absence of injury had a positive impact on the mental component (particularly on role emotional) and physical component. (32) A meta-analysis performed in adult athletes in 2014 demonstrated the impact and negative association between injuries and quality of life domains, especially in the physical and social aspects. (33)

The questions still unresolved are if the increased risk of AF is a consequence of the number of hours of lifetime training, if AF has a preference for certain sports or which are the causes for prevailing in a particular sex.

In terms of treatment, further studies are needed to confirm whether calcium channel blockers are the drugs of choice for HR control or if risk assessment is needed to initiate anticoagulation therapy.

The future seems promising for diagnostic tools such as smartphones or smartwatches for continuous heart rate monitoring. CMR imaging opens a line of research regarding fibrosis in athletes who develop AF.

Several centuries later, the statement "the dose makes the poison", expressed by Paracelsus, the Swiss alchemist considered the "father of toxicology", is still valid and shows us that any excess, even in physical activity, can be detrimental to health.

CONCLUSIONS

The scientific evidence supports the concept that high-

intensity endurance athletes are at greater risk of developing AF, especially in sports with a high dynamic component.

The pathophysiological pathways are different from those of the general population; inflammation, fibrosis, increased vagal tone and bursts of sympathetic activation during training play a key role, and are adaptive responses to excessive physical activity.

Nowadays, pulmonary vein ablation is the treatment of choice, with the same success rate as in non-athletes. This allows a return to sport activity with a low recurrence rate. Further studies are needed to recommend calcium channel blockers and oral anticoagulation, which will also depend on the type of sport practiced.

Conflicts of interest

None declared.

(See authors' conflict of interests forms on the web/Additional material.)

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