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Original article

Comparative Research Between Sportsman's Heart and Hypertrophic Cardiomyopathy

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Summary

Physiological left ventricular hypertrophy is the result of the left ventricle having to function harder due to intense physical exercise. After exercise is stopped, this modest and reversible hypertrophy persists.
Studying these structural alterations is now feasible because to cardiac echodoppler.
Distinguishing this adaptive hypertrophy from the pathogenic hypertrophic cardiomyopathy might be challenging at times.
We examined 212 athletes who competed and a group of hypertrophic cardiomyopathy patients who had asymmetric septal hypertrophy that was confirmed.
The findings demonstrated that there is a boundary between pathological and normal hypertrophy.
This zone contained four athletes, one of whom had hypertrophic cardiomyopathy.
Numerous variables led to the diagnosis, including the patient's history, electrical anomalies, septal thickness, and a diastolic diameter of less than 45 mm. Deconditioning further supported the diagnosis.

Keywords: ***Physiological hypertrophy; Pathological hypertrophy; Sports heart; Hypertrophic cardiomyopathy; Cardiac Doppler.***

Results

Functionally, the same symptoms were observed in both groups: dyspnea, chest discomfort, and palpitations; however, the athletes had these symptoms more frequently and during activity. A clinical examination of both groups likewise showed systolic murmur and bradycardia. Systolic murmur predominated in patients: 80% of instances; bradycardia predominated in athletes: 70% of cases. In both populations, the cardiothoracic index at telethorax is superimposable. One of the athletes had experienced the unexpected death of a brother, according to an analysis of first-degree family histories.

An electrical examination revealed that 30% of the athletes, or 64 participants, had electrical anomalies. Thirteen athletes had repolarization disorders, thirteen had conduction issues (incomplete right), and 38 athletes had left ventricular hypertrophy. , conduction problems (incomplete right bundle branch block 6, Wenckeback-type atrioventricular block 7) in 13 athletes and repolarization disorders in 13 others. Eighty percent of the patients had these electrical anomalies, or 23 out of 28. Of them, 22 had LVH, 14 had repolarization disorders, and 4 had conduction disorders (many patients had two or three anomalies at the same time). Particularly for LVH and repolarization disorders (negative T waves), which are prevalent in both populations, these abnormalities were identical in both groups.

Stress tests were normal for all athletes. Of the athletes with repolarization disorders, 13 had negative her T waves. In 12 of these cases, the motor test was normalized and the abnormality was attributed to movement. The 13th athlete maintained her T wave negative. Echocardiography revealed normal mean values for septal and posterior wall thickness and left ventricular diameter, with measurements categorized according to the sport practiced by the athlete (**Table 3**). The mean patient measurements show significant septal hypertrophy and a significant decrease in diastolic diameter. Therefore, there is no possible correlation between the two populations (**Table 4**). However, when looking at the upper and lower limits of the various measurements, Table 4 clearly shows that an athlete's minimum diastolic diameter and maximum septal thickness are You can see that they overlap. patient. Therefore, between these two populations there is an overlap zone, the Anglo-Saxon "gray zone", the border zone between physiological hypertrophy and pathological hypertrophy, in which no conclusions can be drawn. It is difficult. Examination of diastolic mitral blood flow in two populations identified abnormal Appleton type I mitral blood flow in 70% of patients and normal blood flow in 100% of cases.

Stress echocardiography using the method described above did not reveal early (before the E wave merges with the A wave) mitral blood flow abnormalities in any of the athletes. On the other hand, this study was conducted in 6 patients with hypertrophic cardiomyopathy who had no signs of obstruction and had normal mitral Doppler. Early reversal of mitral blood flow was observed. This is a distinguishing feature in favor of pathological hypertrophy. Our study highlighted the existence of a borderline zone where hypertrophy remains suspect. Four athletes fall into this zone (**Table 5**). they were all men.

Parietal hypertrophy is septal and exceeds 13 mm and ranges from 14 to 16 mm. Diastolic diameter is 40-62 mm. All four athletes had electrical abnormalities such as LVH, repolarization defects, and conduction defects. Radiologically, the cardiac silhouette was normal. There is no abnormality in mitral valve diastolic flow rate during exercise (no early reversal). However, of these four of his athletes, only one of his - his brother - suffered a first-degree sudden death. This led us to check on members of his family. We discovered very suspicious crown thickening in one of our sisters and our father. The latter two had mild symptoms, i.e., discrete dyspnea, atypical chest pain, which were ignored. Three of his athletes were rowers and the fourth was a cyclist. We kept her conditioned for 4 to 12 weeks and discontinued her sporting activities. Echocardiographic monitoring was performed weekly. In three of our athletes, the parietal hypertrophy resolved after her 4 weeks and the diagnosis of hypertrophic cardiomyopathy was definitively excluded. In the fourth case, hypertrophy persisted for more than 3 months, confirming the presence of hypertrophic cardiomyopathy. The athlete has a history of the sudden death of a brother, and suffers from electrical abnormalities such as left ventricular hypertrophy and waveforms. The septal thickness is 14 mm, the diastolic diameter is 40 mm, and the thickness of the father and sister are questionable. He was a rower. This athlete has been permanently disqualified from the competition

Table 1: Athlete population

Sport	Male	Female	Total	Average age (years)
Athletics	12	11	23	21
Cycling	25	00	25	22
Boxing	14	00	14	22
Football	18	12	30	21
Tennis	16	16	32	18.5
Swimming	13	26	39	24
Weightlifting	03	00	03	23
Rowing	13	11	24	23
Gymnastics	00	22	22	18
Total	114	98	212	21.4

Table 2: Patient population studied

Patients	Average age (Years)
20 Males	22
08 Females	21
28 patients	21.5

Table 3: Comparaison of average measurements in the two populations

	DD	IVST	PWT
Average of athlete measurements	51.2	10.2	09.6
Average of patients measurements	41	18	10.5

DD: Diastolic diameter
IVST: Interventricular septum thickness
PWT: Posterior wall thicknes

Table 4: Comparaison of upper and lower measurement limits in the two populations

Upper and lower measurement limits	Athletes	Patients
IVST	07.5 – **16**	**16**-26
PWT	01	08-11
DD	**40** – 62	**40**-48

IVST: Interventricular septal thickness
PWT: Posterior wall thickness
DD: Diastolic diameter.

Table 5: Athletes in the border zone

	Age (years)	DD (mm)	IVST (mm)
Athlete n°1	20	55	15
Athlete n°2	25	40	15
Athlete n°3	25	50	16
Athlete n°4	20	50	14

Discussion

The existence of a borderline between adaptive and pathological hypertrophy in elite athletes has always been a concern for sports medicine physicians, cardiologists, and others [4]. This is because, considering the risk of sudden death, it is important to exclude the diagnosis of hypertrophic cardiomyopathy. That represents. Studies have been conducted on a large number of athletes. The first study by Pelicia [5] of her 1,000 athletes found 16 athletes to be within this limit.

Corrado [6], conducted on all athletes in the Veneto region of Italy, found 22 athletes out of a total of 3,000 elite athletes with excess hypertrophy of 15-26 mm, and a third Maron [6,7] examined 158 cases. In a 10-year study of sudden deaths in athletes, 48 cases of HCM were revealed by autopsy. Differential diagnosis of these two entities is difficult. The first factors to consider are:

If sudden death or the idea of a familial cardiac abnormality is detected, an interview to follow up the medical history allowing further investigation (especially echocardiographic Doppler): Sudden death in one of the athletes, not in the Pelicia series; Collard's 03 Athletes Series This series had the idea of involving sudden death. This shows the importance of interrogation, which must be conducted like a real police officer, especially since athletes are more "hidden". ECG abnormalities were found in 30% of the athletes in our series. In the Pelicia series, it was 29%. Another study of 1,005 Italian elite athletes, all of whom underwent electrocardiography, found that 40% of electrical abnormalities and 15% had echocardiographic structural abnormalities. The electrocardiogram is an excellent reference test and should be codified as part of athlete testing. Echocardiographic Doppler is a basic test to identify structural abnormalities [8,9]. Thickness analysis showed an increase in 4 athletes, 16 in the Pellicia series and 22 in the Collard series. We showed that the diastolic diameter size was greater than 45 mm in most athletes, in contrast to HCM patients. In Pellicia's study, everyone had a of the 16 borderline athletes with diameters greater than 55 mm who underwent deconditioning, none showed regression of hypertrophy. On the other hand, in Collard's series, all 22 athletes had diastolic diameters between 39 and 39.

46mm; all were removed from competition due to hypertrophic cardiomyopathy. The diastolic diameter of our athlete with HCM was 40 mm. Suspected hypertrophy when the diastolic diameter is less than 45 mm suggests pathology. All of our athletes

had normal diastolic mitral blood flow. It was destroyed in 64% of HCMs in our series and 80% of HCMs in Marron's study [10]. This is also an important guiding parameter when the mitral flow is abnormal and accompanied by suspicious hypertrophy, although its normality does not exclude him from HCM (as in the case of our athlete).

In our series, we performed stress echo Doppler testing on all athletes, and no early varus was detected even in athletes with HCM. In the case of HCM, such a reversal was observed. This test has received little research. A study by Brion [11] of 14 elite athletes with suspected hypertrophy and 14 HCM patients with normal mitral valve flow found this early reversal in patients, but none in athletes. It wasn't served. Larger series studies are needed to validate this study. This is a relatively simple alternative if appropriate equipment is available, especially a weight-bearing table, to make the examination more comfortable. Diastolic function has also been studied using tissue Doppler techniques, particularly in the study by Dérumeaux [12], which compared 20 normal subjects, 43 athletes, and 20 patients with moderate hypertrophic cardiomyopathy. She found no differences between healthy people and athletes. However, she found a difference in the main prediastolic velocity of the posterior wall and septum, which was significantly lower in subjects with HCM. Myocardial tissue Doppler is a useful technique for detecting moderate hypertrophic cardiomyopathy.

Echocardiogram Doppler remains an important test for elite athletes, but results may not always be conclusive. HCM is a monogenic inherited heart disease [13]. It is true that genetics has made great advances. Although molecular tests now exist, genetic screening is not yet routinely possible. The main problem is that there are patients who do not have LVH. This is more common in children (who often develop LVH as they grow older), but also common in adults. In this case, we are talking about a healthy career. Therefore, ultrasound examinations should be repeated periodically, especially if there are any signs, such as medical history, electrical abnormalities, or borderline thickness. Despite all these tests, there are still many athletes who have a difficult differential diagnosis and need to be conditioned [14]. To get answers you can trust. However, deconditioning is often poorly accepted by athletes and coaches who demand reliable and rapid diagnosis. The study also highlights the fact that all of the athletes found in the border region were male. The same is true for Pelicia's study (all 16 athletes were male) and Collard's study (of the 22 athletes excluded by HCM, only 3 were female). It seems that it is rare for a female athlete to have the strength to reach the limit. In this case, hypertrophic cardiomyopathy is strongly suspected [15].

On the other hand, the influence of sports on the development of adaptive myocardial hypertrophy must be considered. In our study, three of the four athletes were rowers and the fourth was a cyclist. These tend to be sports that combine resistance exercise and large-scale endurance exercise. A meta-analysis [16] conducted on 1,451 athletes divided them into three groups according to the predominance of physical activity: resistance, mixed and endurance, and found the following answers. All sports lead to muscle hypertrophy, but muscle hypertrophy is greater in resistance athletes; by mixed sports and finally by endurance. This hypertrophy can reach up to 16 mm and is physiological.

Conclusion

The morphological adaptation of the heart due to continuous and repetitive physical activity constitutes the "athlete's heart." These include increased myocardial mass due to parietal thickening and cavity enlargement. However, in most cases these changes are only moderate.

In 5% of cases it can exceed 13 mm, but rarely exceeds 16 mm (pathological). Differential diagnosis between adaptive hypertrophy and pathological hypertrophy requires a series of discussions based on history, medical history, and electrocardiography. These are great directional elements.

However, questions arise regarding systematic echocardiographic Doppler. It has been clearly proven that high performance athletes should do this at least once in their sporting life.

In other cases of doubt, it should be used. If that does not help, there is always the possibility that the condition may deteriorate to allow for a definitive diagnosis, while waiting for reliable and rapid genetic diagnosis and, in some cases, magnetic resonance imaging, which is technologically constantly evolving.

List of Abbreviations

DD: Diastolic diameter
ECG: Electrocardiogram
HCM: Hypertrophic cardiomyopathy
IVST: Thickness of the interventricular septum
IRBBB: Incomplete right bundle branch block
LVH: Left ventricular hypertrophy.

Declarations

Ethical Considerations

Ethical authorization was obtained from the hospital and the scientific council of the Faculty of Medicine; The patients received a written and informed consent form signed after careful explanation objectives, procedure and full involvement of participation in the study. This study was conducted in compliance with ethical rules. standards of our institution on human subjects as well as with the Declaration of Helsinki.

Informed Consent

A signed consent was obtained by the researcher and research assistants before recruitment of the participants into the study after appropriate counselling.

Conflict of Interest

There was no conflict of interest.

Data Availability

Data would be available upon reasonable request.

Funding Statement

The entire financial burdens were burn by the researchers

Author Contributions

M. Abdelbaki: The principal investigator
A. Boureghda; N Hanifi: were involved in the literature search and day to day conduct of the work till conclusion.

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Bibliography

[1] R Amoretti, R Brion: Cardiologie du sport, Edition Masson Paris 2000.

[2] PO Astrand, K Rhodal:Manuel de physiologie de l'exercice musculaire. Edition Masson Paris 1975.

[3] F Carre: L'hypertrophie cardiaque d'adaptation du sportif, les limites physiologiques. Sciences et sport 1994; 9:73-80.

[4] F. Carré: Exercice physique chronique et remodelage cardiovasculaire. Science et Sports; Volume 19, Issue 5, October 2004, Pages 264-266

[5] A Pellicia, BJ Marron, A Sparato, MA Proschan, P Spirito: The upper limit of physiologic cardiac hypertrophy in highly trained elite athletes. N Engl J Med 1991; 324:295-301.

[6] D Corrado, C Basso, M Schiavon, G Thiene: Screening for hypertrophic cardiomyopathy in young athletes. N Engl J 1998; 339:364-368.

[7] BJ Marron, SE Epstein, WC Roberts: Causes of sudden death in the competitive athlete. J Am Coll Cardiol 1986; 7:204-214.

[8] JP Lesbre, B Bernsconi, A Kalisa, Y Demenois, C Shurtz, JL Rey, PH Bor: L'échocardiogramme chez les sportifs. Arch Mal Coeur 1980; 73:976-988.

[9] BJ Maron: structural futurs of the athlete's heart as defined by echocardiography. J Am Coll Cardiol 1986;190-203.

[10] BJ Maron, P Spirito, KJ Green, YE Wesley, RO Bonow, J Arce: Non-invasive assessment of left ventricular diastolic function by pulsed doppler echocardiography in pateients with hypertrophic cardiomyopathy. J Am Coll Cardiol 1987; 10:733-742.

[11] R Brion, MA Slama, G Peres, G Gonnot, JP Olivier, J Droniou: L'échocardiographie d'effort: présentation d'une technique (7eme congrés d'echocardiographie). L'information Cardiologique 1987; 11:583-588.

[12] G Derumeaux, R Douillet, A Troniou, F Jamal, PY Litzler, G Pontier, A Cribier: Différenciation entre l'hypertrophie physiologique du sportif et l'hypertrophie des cardiomyopathies hypertrophiques primitives: intérêt du doppler couleur tissulaire. Arch Mal Cœur 1999; 92:201-210

[13] P Charron, M Komajda: Cardiomyopathie hypertrophique. Encyclopédie cardiologie 1999 ;11-020-A-10.

[14] Bj Marron, A Pellicia, A Sparato, M Granata: Reduction in left ventricular wall thickeness after deconditionning in highly trained olympic athletes. Br Heart J 1993; 69:125-128

[15] A pellicia, BJ Maron, A Sparato, A Biffi, G Caselli, F Culasso: Physiological limits of athlete's heart » in woman. Circulation 1993; 88(Suppl 1):1-57.

[16] MP Babette, L Zwinderman, R Van Der Laarse, A Van Der Wall: The athlete's heart. A meta-analysis of cardiac Structure and Function. Circulation 1999; 100:336-344.

[17] P Berteau, R Amoretti: signes fonctionnels cardiaques du sportif. Médecine du sport pour le praticien. Ed SIMEP, 1993.

[18] H Bjornstad, G Smith, L Storstein, HD Meen, O Hals: Electrocardiographic and echocardiographic findings in top athletes, athletic students and control subjects. Sports Cardiol 1989; 2:425-433.

[19] L Auriacombe, C Mandel, L Fermont, G Simon, JF Piechaud, E Pedroni, J Kachaner: Fonction ventriculaire gauche et adaptation cardiovasculaire à l'effort du jeune sportif. Arch Mal cœur 1987; 80:544-549.