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Case report

Detraining-related changes in left ventricular wall thickness and longitudinal strain in a young athlete likely to have hypertrophic cardiomyopathy

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Abstract
One of the diagnostic criteria in order to differentiate between physiological and pathological left ventricular hypertrophy is the wall thickness reduction after at least 3-month detraining period, which is considered a marker of the athlete's heart. This report describes detraining-related regression of LV hypertrophy and improvement in myocardial deformation in a junior athlete likely to have hypertrophic cardiomyopathy.

Key words: Athlete's heart, detraining, echocardiography, hypertrophic cardiomyopathy, left ventricular hypertrophy, myocardial function, strain echocardiography.

Introduction

Pre-participation screening aims to qualify athletes for competitive sports and must often discriminate between athlete's heart (AH) and hypertrophic cardiomyopathy (HCM). As LV wall hypertrophy is recognized to be 13 to 15mm of thickness in white athletes (de Gregorio, 2007; Maron, 2007; 2009; Pelliccia et al., 2006; Venckunas and Mazutaitiene, 2007), or to 16mm in black males (Papadakis et al., 2011), they may fall into a gray area regarding etiology of hypertrophy, and HCM will need to be ruled out. However, LV wall thickness in young athletes rarely overcomes 13mm (Sharma et al., 2002).

In these cases, various criteria can help to differentiate physiological from pathological LV hypertrophy, and LV wall thickness reduction with deconditioning (detraining) is accepted as a distinctive marker of AH. However, despite a general agreement with this statement, it has not been established how wall thickness reduction would definitely be in order to rule out diagnosis of HCM (Basavarajaiah et al., 2006; Ehsani et al., 1978; Maron, 2009; Maron et al., 1993; Pelliccia et al., 2002).

We report the singular case of a junior athlete likely to have HCM, where we observed such detraining-related changes in LV wall thickness (reverse remodeling), along with mild improvement in myocardial strain-deformation.

Case report

A 16-year-old male Caucasian athlete, soccer player for about 15 hours a week since the age of 11, was referred to our department for pre-participation screening. Family history was negative for cardiomyopathies, syncope or sudden cardiac death, but only scant information about his grand-parents were available. No clinically relevant anomalies were found at physical examination of the heart, lung and vascular system. On admission, heart rate was 65 bpm, breath rate 12 pm, and blood pressure 110/70 mmHg.

Twelve-lead electrocardiogram (ECG) showed sinus rhythm 65 bpm, AV conduction time 155ms, high QRS voltages in anterior leads, deep T wave inversion in leads D1, D2, aVL, aVF, V3-V6 (Figure 1A). Based on these latter findings, high Romilth-Estes point score (=8), Sokolov-Lion voltage (4.6 mV), Cornel voltage (2.6 mV) and Cornel Product (2470), the diagnosis of non physiological LV hypertrophy was made.

Afterward, the athlete underwent transthoracic echocardiography that showed moderately severe LV hypertrophy involving both the ventricular septum and the apex, but without resting or evocate dynamic obstruction. The principal findings are reported in Table 1.

Overall, preserved systolic function and mild diastolic dysfunction were recognized. Higher E/E' ratio and lower S' velocity were found on the septum than the lateral mitral annulus. Compared to historical data on healthy young volunteers (Bussadori et al., 2009), and despite normal LV ejection fraction, impaired longitudinal strain (but normal circumferential and radial deformation) was detected in our athlete. Interestingly, global systolic wall asynchrony was also disclosed at strain analysis (Figure 1D). Wall hypertrophy was confirmed by cardiac magnetic resonance (anterior septum 13.5 mm, apical wall 14 mm, other segments 12.5-13mm), which showed no evidence of delayed gadolinium enhancement.

The athlete also underwent cardiopulmonary treadmill-test that demonstrated peak VO2 consumption of 50 $ml \cdot kg^{-1} \cdot min^{-1}$.

Based on all aforementioned findings, the athlete was diagnosed as having HCM (Table 2) involving both the septum and the apex (HCM type IV, according to Maron et al., 1981), disqualified from competitive sports, and scheduled to further control.

Six months later, ECG findings improved notably with deconditioning (Table 1, Figure 1B), and a decrease in both septal and apical LV wall thickness was disclosed at echo. Longitudinal strain-deformation and systolic synchrony weakly recovered, whereas circumferential and radial strain remained unchanged (Figure 1E).

Once again, the athlete was discouraged from competitions or intense physical training, and he was scheduled for further control. However, two months later, before coming for 12-month check-up, he started playing

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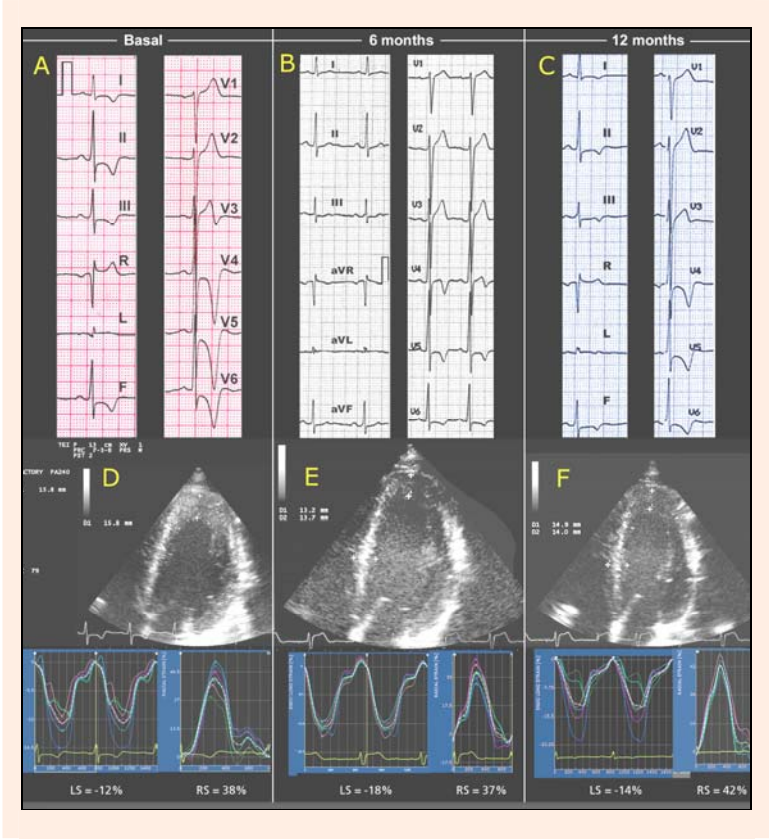

Figure 1. Electrocardiographic and echocardiographic findings in the athlete. Panels A-C: over time differences in T-wave inversion on ECG. Panels D-E: wall thickness changes in apical 4-chamber view. Bottom panels display strain-featured curves of longitudinal and radial deformation. LS, longitudinal strain; RS, radial strain. Pictures 1A and 1D are reproduced by de Gregorio et al., Int J Cardiol 2009, with permission from Elsevier.

recreational tennis. As a result, LV wall thickness mildly increased and ECG picture, longitudinal deformation and wall synchrony worsened again (Table 1, Figure 1C and 1F).

Discussion

Though rare among adolescent athletes, HCM is one of the most common causes of disqualification from competitive sports in Europe and of sudden death all over the world (Corrado et al., 2010; Maron, 2007; 2009; Maron et al., 1981; Pelliccia et al., 2006).

The Italian pre-participation screening program has been demonstrated to be helpful in order to rule out the presence of cardiac diseases like HCM, channelopathies and arrhythmic diseases, simply by adding an ECG to clinical examination. If any, deeply negative T-waves, with or without ST-segment depression, are considered such unusual findings even in elite athletes, being related commonly but not exclusively to apical HCM. The main pathophysiological mechanism has to be researched inside the cell ion channel regulation that can be altered in several conditions (Antzelewitch, 2006). Regrettably, given that T-wave inversion is a challenging expression of either functional or structural anomalies, it should first be regarded as abnormal adaptation to exercise (chiefly LV hypertrophy), especially in adolescents. Cardiac magnetic resonance and/or echocardiography can identify the origin of LV hypertrophy, but overlapping patterns are frequent (Corrado et al., 2010; Maron, 2007; Migliore et al., 2012, Pelliccia et al., 2000; 2002; 2006; Papadakis et al., 2009; Sharma et al., 1999)

Papadakis et al. (2011) described specific T-wave inversion in anterior leads as a normal variant in certain population of Afro-Caribbean athletes. However, studies on large athletic populations disproved the idea that this is a common feature in Caucasian athletes (Corrado et al, 2010; Papadakis et al., 2009; Papadakis and Sharma, 2009). Overall, T-wave inversion was observed in approximately 3% of 1005 highly trained athletes and 2% in young amateur athletes (Pelliccia et al., 2000), whereas no differences between elite athletes and sedentary controls (approximately 4.0% in both groups) were reported by Sharma et al. (1999).

Based on above considerations, ECG findings in our athlete were regarded as expression of pathological LV hypertrophy, and some points deserve attention to confirm our decision. First, LV wall hypertrophy in AH usually involves the septum, whereas apical hypertrophy has never been described in athletes, being an infrequent variant of HCM as well (de Gregorio, 2007; Maron et al., 1981). Moreover, the presence of impaired longitudinal deformation, less than the cut-off value of -19% reported in young healthy populations by Bussadori et al. (2009), likely indicates subclinical systolic dysfunction, which is a distinctive feature of pathological LV hypertrophy.

Furthermore, previous studies have demonstrated that a wall thickness reduction of 2 to 5mm is featuring

Table 1. Main echocardiographic findings at baseline and follow-up.

	Basal (heavy training)	6 months (detraining)	12 months (retraining)
Height (m)	1.75	1.75	1.75
Weight (Kg)	75	78	76
Body surface area (m^2)	1.90	1.94	1.91
LV end-diastolic diameter (mm)	46	47	48
LV end-systolic diameter (mm)	25	24.5	26
Posterior ventricular septum thickness (mm)	12.4	12.1	12.5
Posterior wall thickness (mm)	12	9.5	9.5
Relative wall thickness	0.52	0.46	0.46
Apical thickness (mm)	15.8 (*)	13.0 (-2.8)	14.9 (+1.9)
Lateral wall thickness (mm)	12.5	10.4	11.8
LV mass (g)	209.9	182.8	194.0
LV mass index (adult ASE criteria) ($g \cdot m^{-2}$)	108.9	94.5	101.3
LV diastolic volume (ml)	85	83	80
LV systolic volume (ml)	23	25	24
LV ejection fraction	0.73	0.70	0.70
LA systolic area (cm^2)	14	14	14
LA systolic volume (ml)	43	45	44
LA indexed volume ($ml \cdot m^{-2}$)	22.6	23.2	23.0
RA systolic area (cm^2)	13	13	13
RA systolic volume (ml)	36	35	35
RA indexed volume ($ml \cdot cm^{-2}$)	18.9	18.0	18.3
Mitral E/A ratio	1.75	1.72	1.70
Mitral E-wave deceleration time (ms)	175	180	199
E/E' lateral ratio	6.6	8.3	7.5
E/E' septal ratio	13.1 (*)	11.5	13.3
S' lateral velocity ($cm \cdot s^{-1}$)	11	10	10
S' septal velocity ($cm \cdot s^{-1}$)	7 (*)	8	8
IVRT (ms)	57	58	60
TEI index (TDI-derived)	0.26	0.28	0.24
Circumferential strain (%)	-38.2±2.3	-35.3±2.7	38.5±3.4
Radial strain (%)	38.0±2.7	37.0±2.4	42.0±2.9
Longitudinal endocardial strain (%)	-12.0±3.3 *	-18.3±3.2	-14.3±3.1

ASE, American Society of Echocardiography; IVRT, isovolumic relaxation time; LA, left atrial; LV, left ventricular; RA, right atrial. * Variables consistent with HCM and LV functional impairment.

highly-trained olympic athletes after 3-month deconditioning (Maron et al., 1993), whereas almost complete normalization is expected to occur in elite athletes after 5.6 ± 3.8 years (Pelliccia et al., 2002). Accordingly, apical wall thickness reduction of 2.8 mm after 6-month detraining in our athlete would have been regarded as AH, but this is hard to be accepted as accurate among the criteria listed in Table 2. Despite contemporary knowledge of LV hypertrophy in athletes, to make a differential diagnosis in the young still remains challenging, especially outside the

Table 2. Differentiation between physiologic and pathologic LV hypertrophy in the athlete, based on clinical ECG, Echocardiography and Cardiac Magnetic Resonance.

	AH	HCM
No family history of HCM	+	-
Atypical symptoms	-	-
Race, age, type of sports	-	+
ECG criteria for LV hypertrophy	-	+
Deep T wave inversion on ECG	-	+
Septal hypertrophy (>13mm)	Gray area	
Apical hypertrophy (>15mm)	-	+
LV end-diastolic diameter (45-55mm)	Gray area	
No LVOT obstruction	-	-
LV diastolic dysfunction	-	+
Impaired longitudinal strain	-	+
Delay enhancement at CMRI	-	-
Peak VO_2 consumption	Gray area	
SCORE	1 (+3)	6 (+3)

For details see text. CMRI, Cardiac magnetic resonance imaging; HCM, hypertrophic cardiomyopathy; LV, left ventricular; LVOT, LV outflow tract obstruction. (+) Present / confirmed; (-) Absent / excluded.

cardiologists' environment. A number of variables concur to heterogenic features of HCM in adolescents, and heavy training has been established to be such an important trigger of the first clinical expression in genotype-positive individuals (Basavarajaiah et al., 2006; de Gregorio et al., 2009; Maron, 2007; 2007; Papadakis et al., 2009; Sharma et al., 2002).

On the other hand, we may also hypothesize such a novel clinical picture consistent of exaggerate, though reversible, hypertrophic response of the heart to heavy training (e.g. *Hypertrophic Athlete's Heart*) not necessarily expression of primary HCM, but a single case can only hint future working hypotheses.

A pragmatic implication is that continued clinical surveillance and regular follow-up (ECG + echo) are needed in young athletes with LV hypertrophy of any origin (Basavarajaiah et al., 2006; Maron, 2007; Migliore et al., 2012; Pelliccia et al., 2006; Sharma et al., 2002).

Conclusion

The present study indicates that detraining-related reverse remodeling and myocardial functional changes may also be found in adolescent athletes likely to have HCM.

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Key points

- Hypertrophic cardiomyopathy in adolescent athletes can be discovered by 12-lead ECG
- Physical training is an important trigger for the clinical presentation of hypertrophic cardiomyopathy
- Reverse LV remodeling (wall thickness reduction) with detraining is a common echocardiographic finding in athletes with physiological hypertrophy
- This report demonstrates that reverse remodeling can also be found in adolescent athletes likely to have hypertrophic cardiomyopathy

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