Clinical outcomes in adult athletes with hypertrophic cardiomyopathy: a 7-year follow-up study

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ABSTRACT

Objective Current guidelines recommend precautionary disqualification from competitive sports in patients with hypertrophic cardiomyopathy (HCM). We assessed the incidence of cardiovascular events in a cohort of patients with HCM engaged in long-term exercise programmes and competitive sport.

Methods We reviewed data on 88 consecutive athletes diagnosed with HCM, from 1997 to 2017; 92% male, 98% Caucasian, median age 31 (IQR: 19–44) years. All participated in regular exercise programmes and competitive sport at study entry.

We performed follow-up evaluation after 7±5 (1–21) years. 61 (69%) of the athletes had substantially reduced or stopped exercise and sport (ie, HCM-detrained), and 27 had continued with regular training and sport competitions (HCM-trained). At baseline evaluation, both groups were similar for age, gender balance, symptoms, ECG abnormalities, extent of left ventricular hypertrophy, arrhythmias and risk profile for sudden cardiac death/arrest.

Results During the follow-up period, two participants suffered sudden cardiac arrest or death (0.3% per year) both outside of sport participation. In addition, 19 (22%) reported symptoms (syncope in 3, palpitations in 10, chest pain in 4 and dyspnoea in 2). The Kaplan-Meier analyses of freedom from combined sudden cardiac arrest/death and symptoms (log-rank test p=0.264) showed no differences between HCM-trained and detrained patients.

Conclusion In this adult cohort of low-risk HCM athletes, voluntary decision to pursue in participation in competitive sport events was not associated with increased risk for major cardiac events or clinical worsening compared with decision to reduce or withdraw from exercise programmes and sport. Similar results may not be seen in younger or racially diverse athlete populations, or in patients with more severe HCM phenotypes.

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Hypertrophic cardiomyopathy (HCM) is a heterogeneous, unpredictable disease[1 2] that can cause sudden cardiac arrest and death even in young athletes.[3–6] It is not rare—occurring at the rate of about 1 in 500 people. Guidelines recommend athletes with HCM be disqualified from competitive sport as a precaution.[7–10] It is assumed that the haemodynamic load of intensive exercise, associated with raised neuroadrenergic output on a pathological substrate of myocardial disarray and fibrosis, may trigger ventricular tachyarrhythmias and sudden cardiac arrest.[7–10]

In the lifetime of individuals with HCM, cardiac events have an incidence of <1% per year.[11–15] Whether intensive exercise programmes and competitive sports affect the clinical course of HCM—specifically increasing the risk of cardiac events—is largely unknown.

Therefore, to clarify this issue, we followed a cohort of adult athletes with HCM for a long-term period to assess incidence of symptoms/events with special regard to whether they continued to participate in, or lessened their exposure to, competitive sport.

METHODS

Study population

This was a retrospective study carried out in two cardiology centres in Rome, Italy. Subjects were referred to the centres for management after HCM was discovered or suspected at preparticipation screening. A database of patients evaluated from 1997 to 2017 at the Institute of Sport Medicine and Science and the Cardiology Department of the Azienda Ospedaliera Sant'Andrea was reviewed, and athletes who fulfilled the criteria of 1) diagnosis of HCM and 2) participation in regular exercise programmes and competitive sport were included in this study. The sports played are reported in 'Results' section.

The diagnosis of HCM was based on the examining cardiologist recognising left ventricular (LV) wall thickness of 15 mm or greater in any myocardial segment on echocardiography or cardiovascular magnetic resonance (CMR) in the absence of another condition capable of producing LV hypertrophy.[7 8] When LV wall thickening was in a range of 13–14 mm, HCM diagnosis was supported by association of positive family history for HCM, marked ECG abnormalities and/or pathogenic gene mutation.[7 8] Differential diagnosis from physiological LV hypertrophy (ie, athlete's heart) was conducted by two experienced cardiologists (SC and AP).[16–18]

Cardiovascular evaluation

Cardiovascular evaluation routinely included physical examination, 12-lead ECG and echocardiography.[19] ECG patterns were analysed according to the accepted interpretation criteria in athletes.[20]

Echocardiographic assessment was performed according to established criteria.[21 22] Extent and distribution of LV hypertrophy were assessed from the two-dimensional echocardiographic views and confronted with CMR dimensions. LV ejection fraction was calculated by biplane Simpson's

rule. LV diastolic function, early (E) and late (A) pulsed-wave Doppler diastolic peak-flow velocities were measured[22] and tissue Doppler imaging (TDI) assessment of early (e') and late (a') diastolic peak velocities and their ratio were assessed.[22]

To confirm diagnosis (where needed) and to be able to stratify the participants' risk, we selectively performed additional tests. These included 24 hours ECG monitoring (n=72), cardiac MR imaging (n=71) and genetic testing (n=27).

Athletes included in the present analysis underwent a new clinical and diagnostic evaluation and/or structured telephone interview, at a mean of 7±5 years (1–21) from initial evaluation. We evaluated all participants exercise programmes and intensity and duration of sport activity. 'HCM-trained' were those individuals engaged in three (or more) sessions/week, for 2 or more hours per session, including high-intensity exercise bouts, for a global training total of ≥6, and up to 14 hours/week. All other participants—'HCM-detrained'—were either sedentary or engaged in leisure-time and only occasional exercise programmes (ie, <6 hours/week).

The primary cardiovascular outcome was the occurrence of sudden cardiac arrest or death; the secondary outcomes were the occurrence of symptoms (ie, syncope, symptomatic tachyarrhythmias, chest pain or dyspnoea) or hospital admission for cardiac causes.

All clinical data assembled from the study population are maintained in an institutional database.

Statistical analysis

The data, unless otherwise stated, are presented as mean±SD or median with IQR (25th and 75th percentile) for continuous variables and as proportions, simple frequencies (n) and percentages for categorical variables. Statistical significance was set for a p value of <0.05. To assess the distribution, the Kolmogorow-Smirnov test and histograms were used. The same tests with group differences were tested using Student's t-test or Mann-Whitney U test for normally and non-normally distributed variables, respectively. The Fisher's exact test or χ^2 test were used as appropriate to test group differences of proportions.

Unstandardised mean difference (USMD) and their 95% CIs were analysed between the HCM-detrained and the HCM-trained groups.[23] USMD has been computed as the difference between the HCM measured in the detrained and trained group, divided by the whole population variance.

Finally, the two HCM groups (trained vs detrained) were compared in terms of events-free survival for primary or secondary end point (major and minor events) with log-rank test. Events-free survival over the follow-up was graphically represented by Kaplan-Meier plot, by using multivariable Cox regression analysis, including the confounding factors.

Data were analysed using SPSS Statistics V.24 (SPSS, Chicago, Illinois, USA).

RESULTS

Eighty-eight HCM athletes, median age 31 (IQR: 19–44) years at initial evaluation, mostly Caucasian men (n=81 men, 92%; n=86 Caucasians, 98%) met the criteria for inclusion in this study.

Athletes participated in a range of sports, including soccer (n=34), track and field (n=14), cycling (n=8), basketball (n=7), tennis (n=5), swimming (n=4), triathlon and volleyball (each, n=3), rowing and competitive dancing (each, n=2), water polo, handball, boxing, rugby, motocross and diving (each, n=1). At the time of initial evaluation, all were engaged in regular exercise training, with a weekly schedule of 7 (IQR: 6–14) hours, for >10 consecutive months/year and had participated in competitive events for 14 (IQR: 2–19) years. Level of achievement varied, with a few competing at international events including Olympics (n=3, 4%), a subset at national championships (n=10, 11%) and majority at regional/county tournaments (n=75, 85%). No participant was a professional athlete or received financial compensation to play sport.

Clinical characterisation

At study entry, family history was positive in 28 (32%) participants, including in 23 where there was evidence of HCM in a relative. Seven participants reported premature, HCM-related, sudden cardiac death in a first-degree relative. Most athletes were asymptomatic (n=67, 76%). Physical examination was unremarkable in most (n=80, 91%); eight participants (9%) had systolic ejection murmur (table 1).

ECG abnormalities

One or more ECG abnormalities were present in 81 (92%) athletes. T-wave inversion was the most common (87%), either in anterior, lateral and inferior (37%), anterior (10%) or inferior-lateral leads (40%). Only a minority (8%) showed a normal ECG pattern (table 1).

Morphological characterisation

LV hypertrophy showed an asymmetric pattern in 63 (72%), symmetric in 10 (11%) and apical in 15 (17%). Maximum LV wall thickness ranged from 13 to 28 (mean 17.2±3.8) mm. The anterior and posterior ventricular septum were the most thickened segments in athletes with an asymmetric pattern (table 1).

All athletes had a non-dilated LV cavity, that is, end-diastolic diameter ≤54 mm (mean 47.7±3.7), except two with apical HCM, who showed a mildly enlarged cavity (ie, 57 and 58 mm, respectively).

Systolic LV function was normal in all athletes. LV filling pattern, as assessed by transmitral Doppler, was normal in the majority (n=71, 81%); TDI analysis (available in 23 athletes) revealed an abnormal e' velocity (ie, ≤8 cm/s)[22] in 12 (52%).

Left atrial size ranged from 28 to 50 mm, with majority (n=50, 56%) showing an enlarged chamber (transverse diameter ≥40 mm). Aortic root was 23–43 mm (mean 33±4 mm), and exceeded 40 mm in only two athletes.

Cardiac magnetic imaging at initial evaluation revealed the presence of late gadolinium enhancement (LGE) in 30 of 71 athletes (42%); specifically, spotty patterns were observed at the insertion points of left to right ventricles in 16 (22%), transmural scar in 12 (17%) and mid-myocardial elongated striae in the inferior-lateral wall in 2 (3%).

Arrhythmia profile

Nineteen athletes (22%) showed ventricular arrhythmias on 24 hours ECG monitoring. Specifically, 12 (16%) had non-sustained ventricular tachycardia (NSVT), consisting of 4–25 beats, with minimum RR interval of 220 ms. Isolated ventricular ectopic beats (VEBs) (>100/24 hours) were present in six athletes (7%) and polymorphic in one (1%) athlete.

Only three athletes presented short runs of supraventricular tachycardia. None had atrial fibrillation.

DNA analysis

Of the 27 athletes undergoing genetic test, a pathogenic sarcomere mutation was discovered in 13 (MYBPC3 in 6, MYH7 in

Table 1 Characterisation of the HCM athlete population at the study entry

	Overall HCM population (n=88)	HCM-detrained (n=61)	HCM-trained (n=27)	P value*	USMD (95% CI)
Demographics					
Age (years)	31 (19–44)	28 (20–41)	32 (17–49)	0.219	−0.14 (−0.59 to 0.31)
BSA (m^2)	1.90±0.2	1.91±0.2	1.93±0.18	0.711	−0.07 (−0.53 to 0.38)
Male gender (n, %)	81 (92)	56 (92)	25 (93)	0.900	−0.05 (−0.51 to 0.4)
Systolic BP (mm Hg)	123±14	123±14	125±15	0.595	−0.10 (−0.55 to 0.36)
Diastolic BP (mm Hg)	75±9	76±9	73±10	0.211	0.22 (−0.23 to 0.68)
Heart rate (bpm)	63.8±11	63.4±10	64.9±13.3	0.560	−0.09 (−0.54 to 0.36)
Positive family history (n, %)	28 (32)	18 (30)	10 (37)	0.484	−0.15 (−0.6 to 0.3)
Clinical presentation					
Asymptomatic, n (%)	67 (76)	46 (75)	21 (78)	0.806	−0.09 (−0.55 to 0.36)
Palpitation, n (%)	7 (8)	6 (10)	1 (4)	0.327	0.12 (−0.33 to 0.57)
Chest pain, n (%)	3 (3)	2 (3)	1 (4)	0.570	−0.02 (−0.47 to 0.43)
Dyspnoea, n (%)	2 (3)	1 (2)	1 (4)	0.559	−0.04 (−0.49 to 0.41)
Syncope, n (%)	9 (10)	5 (8)	4 (15)	0.345	−0.14 (−0.59 to 0.31)
NYHA I–II, n (%)	88 (100)	61 (100)	27 (100)	1.000	0.00 (−0.45 to 0.45)
12-lead ECG†					
Negative T-wave, n (%)	74 (84)	50 (82)	24 (89)	0.777	−0.27 (−0.73 to 0.18)
ST-segment depression, n (%)	28 (32)	19 (31)	9 (33)	0.839	−0.04 (−0.5 to 0.41)
LVH (by Sokolow), n (%)	47 (53)	34 (56)	13 (48)	0.568	0.19 (−0.27 to 0.64)
Left axis deviation, n (%)	15 (17)	12 (20)	3 (11)	0.310	0.18 (−0.27 to 0.64)
Left atrial enlargement, n (%)	15 (17)	14 (23)	1 (4)	0.025	0.38 (−0.07 to 0.84)
Pathological Q-wave, n (%)	19 (22)	14 (23)	5 (19)	0.641	0.08 (−0.37 to 0.54)
24 hours ECG monitoring‡					
Frequent or polymorphic VEBs, n (%)	7 (8)	6 (12)	1 (5)	0.662	0.14 (−0.31 to 0.59)
NSVT, n (%)	12 (16)	9 (18)	3 (14)	0.749	0.08 (−0.37 to 0.53)
Echocardiography					
Max LV WT (mm)	17.2±3.8	17.3±4.2	17.0±4.4	0.808	0.05 (−0.4 to 0.5)
LV cavity (mm)	47.7±3.7	47.3±3.9	47.5±3.5	0.175	−0.04 (−0.49 to 0.41)
Left atrium (mm)	39.3±4.5	39.5±4.7	39.0±4.3	0.681	0.08 (−0.37 to 0.53)
Doppler E/A ratio	1.40±0.5	1.46±0.5	1.44±0.5	0.901	0.03 (−0.42 to 0.48)
Ejection fraction (%)	65±4.5	65±4.6	65±4.6	0.684	0.00 (−0.45 to 0.45)
Cardiac magnetic resonance§					
LGE, n (%)	30 (42)	24 (39)	6 (22)	0.272	0.36 (−0.1 to 0.81)
Risk profile					
ESC score	2.2 (1.7–3)	2.2 (1.7–3)	2.2 (2–3)	0.438	0.00 (−0.45 to 0.45)

*Comparison between HCM-detrained and HCM-trained patients (p<0.05 was considered significant).
†Recorded in 88.
‡Recorded in 72.
§Performed in 71.
BP, blood pressure; BSA, body surface area; LGE, late gadolinium enhancement; LV, left ventricular; LVH, left ventricular hypertrophy; NSVT, non-sustained ventricular tachycardia; NYHA, New York Heart Association; USMD, unstandardisedmean difference; VEB, ventricular ectopic beat.

3, TNNI3, TNNT2, MYL3 and LMNA in 1 each) and variants of uncertain significance in 4. In the remaining 10 athletes, gene analysis was negative (table 1).

Risk stratification

Methods to assess risk in patients with HCM regularly engaged in sport are lacking. We cautiously used the algorithms currently suggested by the European Society of Cardiology (ESC)[7] and the American Heart Association (AHA).[8] At study entry, the mean risk score according to ESC[7] was 2.2 (IQR: 1.7–3), with vast majority (n=77, 88%) defined as low risk (ie,<4). Eight athletes (9%) were designated as intermediate risk and three (3%) as high risk (one was implanted, two refused implantable cardioverter-defibrillator (ICD)). According to AHA,[8] an ICD was not recommended for the majority (n=67, 76%); was uncertain in 3, useful in 11 and reasonable in 7 athletes. No athlete was in the ICD-recommended group, based on the AHA criteria.

Events and symptoms over the follow-up

Over the 7-year (1–22) follow-up period, with 616 athlete-years of follow-up, there were two sudden cardiac arrests (incidence: 0.3% per year). First, a leisure-time tennis player aged 52 years incurred sudden cardiac arrest while walking in a shopping area. An automated external defibrillator was available and it showed ventricular fibrillation—the player was successfully defibrillated. Coronary angiography excluded significant atherosclerotic lesions. Second, a novice male boxer, with obstructive HCM at study entry at age 14 years (left ventricular outflow tract (LVOT) gradient=30 mm Hg) became symptomatic at age 19 for dyspnoea with worsening obstruction (LVOT gradient=65 mm

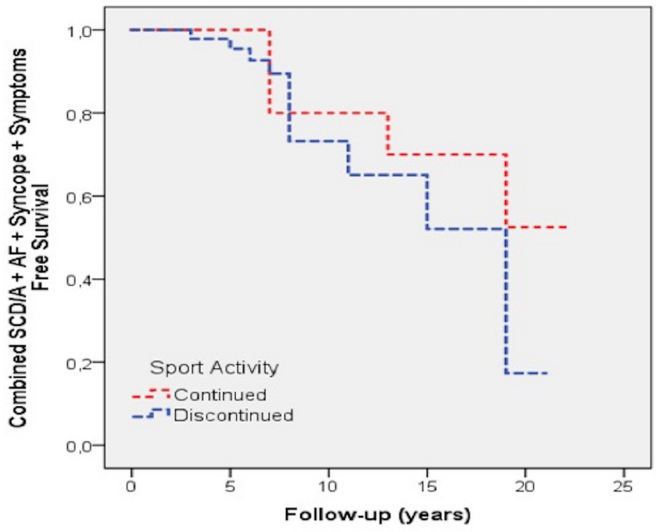

Figure 1 Kaplan-Meier analysis for survival free from event/symptoms over the follow-up in athletes with hypertrophic cardiomyopathy according to their continued participation or withdrawal from exercise programmes and sport competitions (plot constructed from Cox regression analysis data after adjustment for all confounding variables). AF, atrial fibrillation; SCD/A, sudden cardiac death/arrest.

Hg). He stopped sport activities, underwent septal myectomy and his symptoms resolved. One year after septal myectomy—while still asymptomatic—he collapsed and died while out walking in his community (not exercising or playing sport).

Nineteen other study participants reported symptoms during the follow-up—8 of them had been symptomatic at the study entry (ie, incidence of symptoms of 1.3% per year). Symptoms included syncope in 3, palpitations in 10 (with evidence at 24 hours ECG monitoring of bursts of NSVT in 7, frequent VEBs in 1 and paroxysmal atrial fibrillation in 2), chest pain in 4 and dyspnoea in 2 athletes.

Two athletes underwent ICD implantation, due to increased LV wall thickness >30 mm (in both), in association with extensive LGE area (in both) at repeated cardiac magnetic imaging, and NSVT at 24 hours ECG monitoring (in one).

Sport participation and incidence of events/symptoms

At first evaluation, all athletes with HCM were advised to withdraw from competitive sport according to the international and Italian guidelines.[9 10 24] At their most recent evaluation, 61 out of the 88 had dismissed, or had substantially reduced their exercise programmes to occasional and leisure-time activities (in total, <6 hours/week) (defined as 'HCM-detrained'). The remaining 27 patients continued with regular training, with a global load of 6–14 hours/week (defined as 'HCM-trained').

Demographic and clinical characteristics of the two groups were similar as per age, gender and prevalence of symptoms at the time of study entry. Indeed, ECG abnormalities, the presence of arrhythmia, cardiac dimensions and the ESC risk score showed no differences between the two groups. (table 1).

During the follow-up, two major events occurred in the HCM-detrained group outside the context of exercise. The incidence of new symptoms was similar in patients classified as 'HCM-trained' versus 'HCM-detrained'. Specifically, palpitations were reported by six (10%) of detrained versus one (4%) of trained (p=0.346); chest pain by two (3%) versus one (4%) (p=0.809); dyspnoea by two (3%) versus zero (0%) (p=0.462) and syncope by five (8%) versus four (15%) (p=0.319). Also similar was the incidence of arrhythmias (ie, NSVT) at 24 hours ECG monitoring (31% vs 23%; p=0.83), while paroxysmal atrial fibrillation occurred uniquely in two HCM-detrained patients (p=0.341).

The Kaplan-Meier analyses of freedom from combined events and symptoms (log-rank test p=0.264) showed no differences within the HCM population, regardless of the continuation or interruption of competitive sports activities (figure 1).

DISCUSSION

HCM and the risk of sport

The present investigation was prompted by the concern and uncertainty in advising exercise programmes and sport participation in young and adult patients with HCM. While a cautionary approach is recommended in the current guidelines,[9 10] the patient's (and parents) aspiration for continued sport participation and the lure of fame or financial benefits mean that patients commonly ignore medical advice. This behaviour is not uncommon among patients with HCM (not just athletes).[25] Our clinical experience is that about one-third of patients with HCM disregarded our medical advice—breaking Italian guidelines[24]—and continued to engage in competitive sport activities.

The cautious approach of the guidelines was reasonably developed by recognition of the heterogeneous and unpredictable clinical course of HCM, the results of retrospective pathological registries of sudden cardiac death in athletes, and the realistic assumption that strenuous exercise may trigger fatal tachyarrhythmias in patients with HCM.[3–8] However, the lack of a registry with prospective data collected from patients with HCM regularly engaged in sport activities represents an obvious limitation and relevant knowledge gap of the current recommendations.

The risk in athletes with HCM

This investigation provides unique data that shed some light on the outcomes of HCM in adult patients engaged in sport activities. Specifically, we found no differences in clinical outcome in a cohort of adult patients with HCM who remained engaged in sport versus those that withdrew from exercise programmes. The incidence of major symptoms (syncope) or ventricular arrhythmias (ie, NSVT) was also not different in the two subgroups of HCM (trained vs detrained) over an average 7-year follow-up.

We emphasise that the two major events (one sudden cardiac death postmyomectomy and one sudden cardiac arrest that survived) occurred in individuals in the HCM-detrained group who were not exercising. Our data, although limited by individuals' self-selection into continued training or detraining, does not provide evidence for the case sport activities negatively influence the clinical course of HCM in all patients. It is critical to recognise that this cohort comprised an adult athlete population (mean age 31 years at diagnosis) with a predominantly low-risk phenotype (see below), and thus these results should not be applied to younger, adolescent athletes, or patients with a more severe HCM phenotype.

It is important also to state that the athletes we examined do not represent the typical HCM patient population, but are more likely a selected cohort, characterised at initial assessment by the combination of an average mild LV hypertrophy, normal indexes of LV function and low prevalence of limiting symptoms. Indeed, the adult age was presumably an important determinant of the favourable outcome. In addition, the algorithms in place confirmed that most athletes (ie, 88% according to ESC

and 76% according to AHA[7 8]) were at low risk, and without indication for prophylactic ICD implant.

The present data confirm and expand our previous observations in a small group of 35 athletes with HCM.[26] Our results are also consistent with the observation that HCM with sedentary patients, randomly assigned to exercise programmes or usual care for a 16-week period, had no occurrence of adverse events, although the study was not designed or powered to establish safety from sudden cardiac arrest/death in sport.[27] Lampert *et al* reported no differences in the rate of shocks the patients with HCM-ICD received either at exercise or at rest, which was different from what occurred in patients with arrhythmogenic right ventricular cardiomyopathy (ARVC-ICD).[28] A previous case series of athletes with HCM led to the conclusion that within the large spectrum of the disease, there were individuals who were able to engage in intense athletic training and competitions for many years, without incurring symptoms or dying suddenly.[25]

The zero risk does not exist

Our results should not provide a false sense of reassurance regarding the clinical course of patients with HCM able to maintain an athletic lifestyle. Our experience shows that, regardless of the participation or interruption of sport activities, a proportion of patients (>20%) incurred symptoms over the 7-year follow-up period, at an average incidence of 1.3% per year. The occurrence of two sudden cardiac arrests (0.3% per year) was notable given the mild phenotypic abnormalities and absence of major risk markers. These considerations confirm the clinical unpredictability of HCM, and highlight that no patient with HCM may be considered at zero-risk.[1 3–6 29 30]

Limitations

Certain limitations apply to our investigation. First, our study was an observational analysis. We assembled our study population as consecutive from two different large databases, in order to minimise the selection bias. However, it is likely that patients included in this study represent a selected cohort of low-risk adults with HCM, and not the typical clinical spectrum of patients with HCM. Our investigation cannot be considered to 'predict' the outcome of all patients with HCM, particularly in younger adults, adolescents and children, engaged in sport activities.

Indeed, we enrolled only Caucasian subjects, mostly males, and the role of race and sex cannot be assessed. This is an important limitation given multiple studies have demonstrated that HCM as a cause of sudden cardiac death has disproportionately affected black athletes.[3 5 31] Types of sport included were those common in Europe, while other disciplines (ie, American football, baseball or cricket) were not included.

Finally, the major limitation of this study was the small sample size leading to lack of adequate power and sparse-data bias,[32] which limits the statistical power of our analysis to compare the two 'groups', and the relatively short period of follow-up. With only two major cardiovascular events and the only sudden cardiac death occurring in a patient after septal myomectomy (which might have increased the risk of arrhythmia), definitive conclusions regarding the safety of continued sports participation versus detraining in adult athletes with HCM cannot be drawn.

Larger investigation is needed, especially among younger competitive athletes (ie, adolescent and college aged) where the majority of eligibility decisions are made, although such a prospective study enrolling a large cohort of young athletes with HCM appears very difficult to be implemented.

Therefore, it is conceivable that other studies including a larger cohort of patients with HCM followed for a prolonged period of observation are needed to confirm our results and to support the apparent absence of an additional independent risk related to sport participation in adult athletes with a low-risk HCM phenotype.

CONCLUSION

In our adult cohort of athletes with HCM, classified as low-risk by ESC score, voluntary decision to pursue in participation in competitive sport events did not convey, over a 7-year follow-up, an increased risk for sudden cardiac arrest or clinical worsening, compared with decision to reduce or withdraw from exercise programmes and sport.

Similar results may not be seen in younger or racially diverse athlete populations, or in patients with more severe HCM phenotypes.

What are the findings?

- Our results suggest that adult patients with mild phenotypic hypertrophic cardiomyopathy (HCM) who remained engaged in training and competition were not exposed, over a 7-year follow-up period, to an increased risk for major cardiac events or clinical worsening, compared with patients who reduced or stopped exercise programmes.

How might it impact on clinical practice in the future?

- Adult patients with HCM and a low-risk profile may participate in regular exercise programmes and competitive sport without increased cardiovascular risk.
- Results may not be applicable to younger or more racially diverse athlete populations, or in patients with more severe HCM phenotypes.
- Therefore, participation in regular exercise programmes and competitive sport can be selectively considered in adult patients with HCM with a low-risk profile.

Contributors Each of the authors had a substantial part in the planning of the study, collecting data, interpreting results, writing and revising the manuscript. Specifically, planning the study project: AP, SC, CA. Collecting data: EL, MP, DR, LL, FMDP. Interpreting data: AP, SC, CA, EL, MBM, VM, CB. Statistical analysis: CB. Manuscript writing and revising: AP, SC, CA, CB.

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Competing interests None declared.

Patient consent for publication Not required.

Ethics approval The plan of the study was evaluated and approved by the internal Review Board of the Institute of Sport Medicine and Science. The research was done without patient involvement. Patients were not invited to comment on the study design and were not consulted to develop patient relevant outcomes or interpret the results. Patients were not invited to contribute to the writing or editing of this document for readability or accuracy.

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Data availability statement All clinical data assembled from athletes included in this study are maintained in the institutional database of the participating institutions.

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