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Relationship of Race to Sudden Cardiac Death in Competitive Athletes With Hypertrophic Cardiomyopathy

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OBJECTIVES The goal of this study was to determine the impact of race on identification of hypertrophic cardiomyopathy (HCM).

BACKGROUND Sudden death in young competitive athletes is due to a variety of cardiovascular diseases (CVDs) and, most commonly, HCM. These catastrophes have become an important issue for African Americans, although HCM has been previously regarded as rare in this segment of the U.S. population.

METHODS We studied the relationship of race to the prevalence of CVDs causing sudden death in our national athlete registry, and compared these findings with a representative multicenter hospital-based cohort of patients with HCM.

RESULTS Of 584 athlete deaths, 286 were documented to be due to CVD at ages 17 ± 3 years; 156 (55%) were white, and 120 (42%) were African American. Most were male (90%), and 67% participated in basketball and football. Among the 286 cardiovascular deaths, most were due to HCM (n = 102; 36%) or anomalous coronary artery of wrong sinus origin (n = 37; 13%). Of the athletes who died of HCM, 42 (41%) were white, but 56 (55%) were African American. In contrast, of 1,986 clinically identified HCM patients, only 158 (8%) were African American ($p < 0.001$).

CONCLUSIONS In this autopsy series, HCM represented a common cause of sudden death in young and previously undiagnosed African American male athletes, in sharp contrast with the infrequent clinical identification of HCM in a hospital-based population (i.e., by seven-fold). This discrepancy suggests that many HCM cases go unrecognized in the African American community, underscoring the need for enhanced clinical recognition of HCM to create the opportunity for preventive measures to be employed in high-risk patients with this complex disease. (J Am Coll Cardiol 2003;41:974–80) © 2003 by the American College of Cardiology Foundation

Sudden death in the young athlete is always an unexpected and tragic event with great impact on families, the community, and medical establishment (1–7). Previously, we and others have documented that the cause of such athletic field deaths is predominantly structural cardiovascular disease (CVD) (1,3–6). However, the demographic profile of these devastating events is incompletely resolved, including the impact of race. Premature sudden cardiovascular deaths, including those in competitive athletes, has become a major issue for African Americans, and identification of the causes of these catastrophes is a priority (2,8,9). In the present report, we utilized our unique registry (5) to analyze the causes of sudden cardiac death in competitive athletes with respect to race, and assess the relevance of these findings to preventive and community medicine.

METHODS

Selection of subjects. The Minneapolis Heart Institute Foundation registry was established in 1992 to systematically assemble, prospectively and retrospectively (from 1985), cases of young athletes who died suddenly. These cases were primarily identified from news media accounts, but also from a variety of other sources including informal communications and reports from high schools and colleges (5). A total of 584 young athletes who died suddenly were consecutively enrolled in the registry to April 2000. Subjects were initially considered for inclusion if they were: 1) truly a competitive athlete, that is, a participant in an organized team or individual sport requiring regular training and competition while placing a high premium on excellence and achievement (10), and 2) <35 years old while actively engaged in competitive sports. The number of cases reported to the registry have increased with time: 117 (20%) before 1990; 105 (18%) from 1990 to 1994; and 362 (62%) from 1995 to 1999. The largest number of reported cases in a single year occurred in 1996 (n = 70). Forty-two states were represented, most commonly: California (n = 31 cases), Florida (n = 17 cases), Michigan and New York (n = 16 each cases), Ohio (n = 15 cases), and Texas (n = 13 cases).

Of these 584 subjects, 298 were ultimately excluded from the present study based on: 1) identification of primary noncardiovascular causes of death (e.g., drug abuse, complications of asthma, heat stroke, drowning, head trauma, or

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Abbreviations and Acronyms

ARVC = arrhythmogenic right ventricular cardiomyopathy
CVD = cardiovascular disease
HCM = hypertrophic cardiomyopathy
LV = left ventricle/ventricular

ruptured cerebral artery); 2) sudden death due to a blunt chest blow in the absence of structural cardiac disease (commotio cordis) (7); 3) incomplete postmortem or toxicology examination, or one insufficient to establish the probable cause of death based on the available clinical and autopsy data; and 4) inability to track and obtain the necessary diagnostic data due to confidentiality considerations restricting access to autopsy information. The remaining 286 athletes constitute the principal study group. Selected data from 134 athletes are included in a prior report (5).

Data assembly. Upon initial identification, a systematic tracking process was established to assemble information on each case, including the autopsy report (with complete gross anatomic, histologic, and toxicologic data), as well as pertinent clinical information. In selected instances, primary pathologic materials were requested and analyzed, and, when necessary, the findings were verified by direct communication with medical examiners. Clinical information (e.g., circumstances of collapse and preparticipation screening) was derived from written accounts and from telephone interviews with family members, witnesses, or coaches. The final diagnoses were based almost solely on the autopsy findings in the vast majority of athletes. However, 46 (16%) had cardiovascular evaluations during life, and, when appropriate, those data were considered in formulating the diagnosis and cause of death.

Diagnosis of hypertrophic cardiomyopathy (HCM). Our primary diagnostic criterion for probable or definite evidence of HCM was a hypertrophied nondilated left ventricle (LV) in the absence of another cardiac or systemic disease capable of producing the degree of hypertrophy present (11–13). Required criteria were, as previously described (5), heart weight of ≥500 g and ≥1 supporting clinical or morphologic feature in any of the following categories: 1) family history of HCM with or without premature sudden death; 2) asymmetric pattern of LV hypertrophy such as prominent bulging of the ventricular septum into the outflow tract, or marked wall thickening (ventricular septum ≥20 mm), and/or enlarged left atrium with small ventricular chambers, fibrous outflow tract contact plaque (on the septum), or markedly elongated mitral valve leaflets; 3) histologic abnormalities of LV, including marked disorganization of cardiac muscle cells (14), abnormal intramural coronary arteries (15,16), and/or replacement scarring (15,16). Alternatively, hearts with weight <500 g but with maximal LV wall thickness ≥20 mm were also regarded as diagnostic of HCM.

Hearts that showed a modest, but otherwise unexplained, increase in cardiac mass (heart weight ≥400 g in males and ≥350 g in females but <500 g) and mild LV wall thickening (15 to 19 mm), but with one or no other supporting diagnostic feature of HCM, were judged as suggestive of HCM, although insufficient to warrant a definitive diagnosis (5).

Clinical HCM cohort. For comparative purposes, a multicenter population of 1,986 patients with HCM who were diagnosed clinically (12) was assembled from the outpatient records of the University of Florida Health Sciences Center (Gainesville) (n = 75), St. Luke's-Roosevelt Hospital Center (New York) (n = 107), Cleveland Clinic Foundation (n = 1,314), and Minneapolis Heart Institute (n = 490) specifically to ascertain the distribution of HCM patients with respect to race. Data from these four centers included all HCM patients referred to that institution, both locally and from other regions.

These institutions were selected because of their recognition as HCM centers, as well as location in large metropolitan areas and/or regions generally accessible to large numbers of African American patients. Therefore, the cohort included a prominent component of largely unselected patients, as well as patients who were part of tertiary center referral patterns.

Ages at initial institutional evaluation were 56 ± 19 years; 1,060 of 1,986 (53%) were male. Maximal LV wall thicknesses obtained with two-dimensional echocardiography were 20 ± 5 mm (range, to 55 mm). Outflow obstruction under basal conditions (Doppler-estimated gradient ≥30 mm Hg) was present in 693 of 1,986 (35%).

Statistical methods. Data are expressed as mean ± SD. Proportions were compared with the chi-square test, where appropriate.

RESULTS

Demographic profile of athletes. In the 286 athletes who died of CVD, ages were 9 to 40 years (mean, 17 ± 3); 256 (90%) were male. Distribution according to race was: white (n = 156; 55%); African American (blacks of African descent) (n = 120; 42%); and other races (n = 10; 3%), including Asian (n = 5; 2%); Hispanic (n = 4; 1%); and Native American (n = 1; 0.3%). Most athletes were competing in organized high school (n = 188; 66%) or college sports (n = 53; 19%), and 12 (4%) were professional athletes. The remaining 33 athletes (12%) were ≤14 years and engaged in organized youth or junior high school sports. Nineteen athletes (7%) were regarded as elite, having achieved national or international levels of competition.

A variety of 18 competitive sports were represented, most commonly basketball and football (combined: 192, 67%); only in basketball were African American athletes more common than whites (i.e., 63:35). Of the 286 athletes who died suddenly of heart disease, 204 (71%) collapsed during

Table 1. Frequency and Racial Differences for Cardiovascular Causes of Sudden Death in 286 Young Competitive Athletes

Cause of Death	No. (%) of Athletes	M:F	Age ± SD	White No. (%)	A-A No. (%)	Other No. (%)	p Value*
HCM	102 (36)	99:3	17.0 ± 2.5	42 (41)	56 (55)	4 (4)	0.002
Coronary artery anomalies of wrong sinus origin†	37 (13)	28:9	15.3 ± 2.5	15 (40)	18 (49)	4 (11)	NS
Indeterminant, possibly HCM‡	29 (10)	27:2	17.9 ± 4.0	15 (52)	12 (41)	2 (7)	NS
Myocarditis	20 (7)	16:4	17.1 ± 3.9	12 (60)	8 (40)	0	NS
Ruptured aortic aneurysm	12 (4)	9:3	18.4 ± 5.1	8 (67)	4 (33)	0	NS
ARVC	11 (4)	9:2	17.0 ± 2.4	10 (91)	1 (9)	0	0.033
Tunnelled coronary artery	11 (4)	11:0	16.4 ± 3.0	8 (73)	3 (27)	0	NS
Aortic valve stenosis	10 (3)	10:0	15.4 ± 1.5	10 (100)	0	0	0.017
Atherosclerotic coronary artery disease	10 (3)	9:1	17.5 ± 3.9	7 (70)	3 (30)	0	NS
Idiopathic dilated cardiomyopathy	9 (3)	8:1	17.9 ± 1.8	5 (56)	4 (44)	0	NS
Mitral valve prolapse	9 (3)	8:1	18.4 ± 6.5	5 (56)	4 (44)	0	NS
Coronary artery hypoplasia	8 (2)	8:0	18.8 ± 9.1	7 (88)	1 (12)	0	NS
Other congenital coronary anomalies§	8 (2)	5:3	15.6 ± 2.5	6 (80)	2 (20)	0	NS
Cardiac sarcoidosis	3 (1)	3:0	21.3 ± 4.6	0	3 (100)	0	NS
Long QT syndrome	3 (1)	3:0	16.7 ± 0.6	2 (67)	1 (33)	0	NS
Congenital heart disease‖	3 (1)	2:1	14.0 ± 2.3	3 (100)	0	0	NS
Myocardial infarction (etiology unresolved)	1 (0.3)	1:0	15.0 ± 0.0	1 (100)	0	0	NS

*Comparison of the proportion of athletes with each individual cardiovascular abnormality calculated relative to the total number of African American (n = 120) or white athletes (n = 156) who died of heart disease; †includes 32 with anomalous left main coronary artery from right (anterior) sinus of Valsalva, and 5 with anomalous right coronary artery from left sinus; ‡hearts with autopsy findings regarded as consistent with HCM, but insufficient to be diagnostic of the disease; §includes origin of left anterior descending coronary artery from pulmonary trunk (n = 1), intussusception associated with hypoplastic left circumflex coronary artery (n = 1), single coronary artery (n = 2), acute angulation of left coronary artery (n = 3), and coronary artery aneurysm (n = 1); ‖includes secundum atrial septal defect (n = 1), coarctation of aorta (n = 1), and double outlet right ventricle (n = 1).

A-A = African American; ARVC = arrhythmogenic right ventricular cardiomyopathy; F = female; HCM = hypertrophic cardiomyopathy; M = male.

or immediately after a training session (n = 129) or a formal athletic contest (n = 75). The remaining 82 athletes (29%) died either during mild recreational physical activities (n = 46), or while sedentary (n = 36; including 11 during sleep). Only 55 athletes (19%) were known to have experienced symptoms that could have been cardiovascular in origin (e.g., chest pain, exertional dyspnea, syncope, or recurring dizziness).

Of the 286 athletes, information regarding preparticipation medical evaluations were available in 252. Customary personal and family history and physical examination, or other clinical evaluations, were performed as part of medical clearance for high school or college sports in 247 of the 252 athletes (98%). Preparticipation examinations were documented in 98.6% of whites and 97.3% of African Americans (p = NS).

Cardiovascular causes of sudden death in athletes. A variety of CVDs were identified as the cause of sudden death in the 286 athletes (Table 1). In three of these, standard autopsy examination did not identify a cause of death, but a clinical evaluation had previously documented or strongly suggested long QT syndrome (17). Three other athletes with Marfan's syndrome did not have autopsies, but the clinical circumstances of their death was judged most consistent with aortic dissection and rupture (18).

Hypertrophic cardiomyopathy was the most common cause of death and occurred in 102 athletes (36%). Maximum LV wall thickness was 23 ± 5 mm, ranging to 40 mm, and ≥30 mm in 12 athletes (11–13,19). Of the 102 athletes with HCM, 9 had associated abnormalities that may have contributed to death, including tunneled (bridged) left anterior descending coronary artery (n = 7) (20) and coronary artery hypoplasia (n = 2) (21).

The second most frequent cardiovascular cause of sudden death was coronary artery anomalies of wrong sinus origin (22,23) that were present in 37 athletes (13%); these were left main coronary artery originating from the right (anterior) sinus of Valsalva (n = 32) and right coronary artery from the left sinus of Valsalva (n = 5) (Table 1). The remaining 118 athletes died suddenly of a variety of cardiac diseases or malformations, each of which comprised ≤7% of the overall study group. These included most commonly, myocarditis (n = 20), ruptured dissecting ascending aortic aneurysm with or without evidence of Marfan's syndrome (n = 12), and arrhythmogenic right ventricular cardiomyopathy (ARVC) (n = 11) (Table 1) (24).

Relation of race to HCM and other CVDs. ATHLETES WITH SUDDEN DEATH. Of the 102 competitive athletes who died suddenly of HCM, most (56; 55%) were African American and 42 (41%) were white (p = 0.002); the remaining 4 (4%) were of other races (Table 1, Fig. 1). African American and white athletes with HCM did not differ significantly with respect to age (17.1 ± 2 years vs. 16.7 ± 2 years), gender (98% vs. 95% male), maximum LV wall thickness (23.0 ± 5 mm vs. 22.2 ± 5 mm), nor competitive sport at the time of death (most commonly basketball; 57% vs. 33%; p = NS). Arrhythmogenic right ventricular cardiomyopathy and aortic valve stenosis were both significantly more common in white than African American athletes (Table 1, Fig. 2). Of 102 athletes who died of HCM, the correct diagnosis was ultimately made during life in only 3 (3%), of whom 2 were white and 1 was African American.

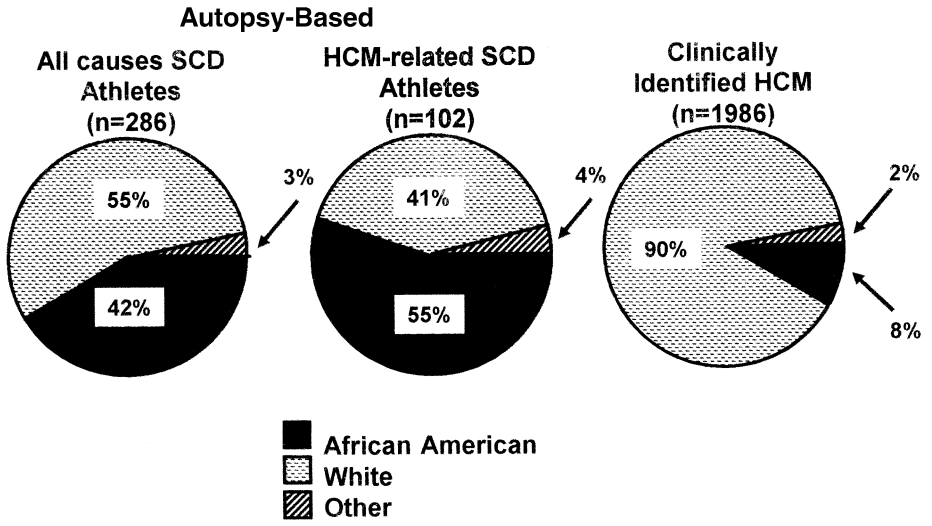

Figure 1. Distribution according to race shown separately for the overall autopsy-based study population of 286 trained competitive athletes who died suddenly from a variety of cardiovascular diseases **(left)**, for those 102 athletes studied at autopsy who died of hypertrophic cardiomyopathy (HCM) **(center)**, and a clinically identified, multicenter hospital-based cohort of 1,986 patients with HCM **(right)**. SCD = sudden cardiac death.

CLINICALLY IDENTIFIED HCM PATIENTS. In the assembled multicenter, hospital-based cohort of 1,986 HCM patients, 1,784 (90%) were white and only 158 (8%) were African American (Fig. 1); the remaining 44 (2%) were of other races. The prevalence of African American patients with HCM was highest at the University of Florida Health Sciences Center (11 of 75; 15%), lowest at the Minneapolis Heart Institute (10 of 490; 2%), and intermediate at the Cleveland Clinic (130 of 1,314; 10%) and St. Luke's-Roosevelt Hospital (7 of 107; 6%).

Therefore, the prevalence of African Americans among athletes who died suddenly of HCM (56 of 102; 55%) was significantly greater than the representation of African Americans in the cohort of HCM patients clinically diagnosed within outpatient and inpatient hospital settings (158 of 1,986; 8%; $p < 0.001$). Also, clinically identified African American patients with HCM were older (59.5 ± 16 years) and more commonly women (64%), and less frequently had outflow obstruction (25%), compared with the HCM patients of other races (54.0 ± 19 years, 45% and 36%, respectively; $p < 0.001$, 0.001, and 0.01. African Americans and those of other races with HCM did not differ with respect to maximum LV wall thickness (20.5 ± 5 mm vs. 20.8 ± 6 mm, respectively).

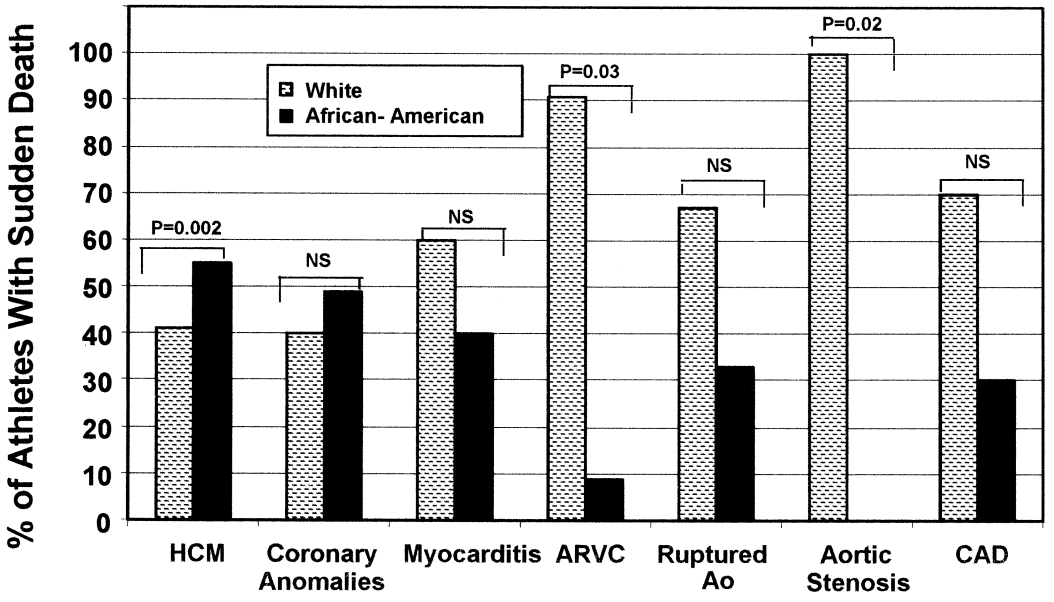

Figure 2. Impact of race on cardiovascular causes of sudden death in the population of 286 competitive athletes, with data shown for those seven structural heart diseases represented by at least 10 deaths. Ao = aortic; ARVC = arrhythmogenic right ventricular cardiomyopathy; CAD = atherosclerotic coronary artery disease; HCM = hypertrophic cardiomyopathy.

DISCUSSION

Demographics and causes of athletic field deaths. Sudden death on the athletic field in young participants has been of considerable interest in the lay and medical communities (1–7,10,25). Indeed, in studies from the U.S. (including the present one), a variety of predominantly congenital CVDs have been documented to be responsible for most of these deaths, with HCM the most common condition occurring in about one-third of cases (1,3–5,25).

Our large prospective registry of sudden deaths in young athletes proved instructive with regard to the epidemiology of these catastrophic events and particularly the significance of race in HCM. We found that the majority of athletes who died of HCM were African Americans (i.e., 55%). While characterization of the racial composition of the entire athlete population in the U.S. is well beyond the scope of the present study, this predominance of African Americans with HCM cannot be solely attributable to relative rates of sports participation among the races. Indeed, there are about 8 million high school and college athletes (of both genders) in the U.S. each year, and the overall U.S. population includes 2.7 million male and 2.7 million female African Americans between the ages of 15 and 24 years. Therefore, for African Americans to comprise 55% of high school and college athletes, fully 80% of all African Americans in the U.S. age 15 to 24 years would have to be members of organized athletic programs. Obviously, because this cannot be the case, it is most reasonable to assume that participants in organized sports nationally are not predominantly black, and that African Americans probably have a representation in the overall athlete population similar to that in the general U.S. population (i.e., about 15%) (26).

HCM in African Americans. Historically, HCM has been regarded as a condition that uncommonly affects and rarely is diagnosed in black patients. The published medical literature reflects this view in that HCM patients reported by race are very uncommonly identified as black (27). This may be explained, in part, by the fact that the tertiary center referral institutions that have traditionally assessed the largest numbers of HCM patients (in the U.S., Canada, and Europe), and contributed substantially to the literature, have generally evaluated relatively few black patients with CVD (12,28,29).

Indeed, the observation that clinically diagnosed HCM patients are uncommonly African American is supported by the large multicenter HCM population assembled for the present analysis in which only 8% of about 2,000 patients with this disease were reported as African American (range, 2% to 15% for each of the four institutions). Therefore, HCM was seven times more common in African Americans when the disease was identified for the first time at autopsy after a sudden death on the athletic field than when recognized within a clinically diagnosed patient cohort. This suggests that many HCM cases in African Americans go undetected in the community. Furthermore, African Americans appear to comprise a smaller proportion of our multicenter clinical HCM population (i.e., 8%) than would be expected from their representation in the general U.S. population (i.e., 16%) (26).

This finding of a large disparity in the clinical identification of HCM between African Americans and whites is provocative and can possibly be explained in a number of ways. First, in general terms, there is a disproportionate access to subspecialty medical care between the black and white communities, specifically with regard to referral for specialized cardiovascular procedures (30–36). This perception suggests that underdiagnoses of HCM in young African Americans may be attributable, in large measure, to socioeconomic factors that potentially limit access to medical specialty referral (and echocardiography), which is usually a prerequisite for the clinical diagnosis of HCM. Consequently, it may be much less likely for young black males (compared with their white counterparts) to be identified with HCM, particularly if asymptomatic. Of note, our clinically identified African American patients with HCM were not predominantly male (as is consistently the case in white patients with this disease) (12,13,19,28), but, rather unexpectedly, were more commonly female (i.e., 64%). Finally, the well-recognized difficulties in diagnosing HCM in generally healthy asymptomatic populations (1,25) are likely to be accentuated in African Americans for whom HCM-related LV hypertrophy may be ascribed erroneously to mild degrees of systemic hypertension (37).

Preparticipation screening. Our findings may also be relevant to the issues surrounding preparticipation screening for competitive athletes in the U.S. (25). We found no obvious differences in the frequency with which standard screening and cardiovascular evaluations were carried out between African American and white athlete populations. The failure to identify during life a substantial number of athletes (either black or white) who died suddenly with HCM raises the question of the efficacy attributable to customary screening as it is generally practiced in the U.S. with only a history and physical examination (38,39). Certainly, the rigor of preparticipation screening has been of concern for both high school (38) and college-age (39) competitive athlete populations, as well as the implicit limitations of the history and physical examination in identifying or raising the suspicion of HCM (and other CVDs). Of note, in Italy, preparticipation screening employs an obligatory 12-lead electrocardiogram (40), which has been shown to increase the likelihood that HCM will be diagnosed before competitive athletics are undertaken (41).

Significance of the findings. Alternatively, it is possible that HCM in African Americans may represent a more virulent form of the disease, possibly due to a malignant genetic substrate when associated with exercise (42,43), and, thereby, predisposing to sudden death on the athletic field in susceptible individuals. However, regardless of these considerations, it is our aspiration that the present report

will trigger greater awareness that HCM not uncommonly occurs and is an important cause of sudden death in young African American males, thereby creating a higher index of suspicion and ultimately more frequent clinical HCM diagnoses in such athletes. Indeed, the failure to identify HCM in young African American athletes has important and potentially life-threatening consequences. Specifically, there is the possibility that such individuals will not be afforded important options, that is, disqualification from intense competitive sports (in accord with recommendations of Bethesda Conference #26) (10,44) to reduce sudden death risk during physical activity, nor employment of potentially life-saving prophylactic interventions such as the implantable cardioverter-defibrillator in high-risk HCM patients (45).

Study limitations. The present study is a retrospective autopsy-based analysis, comprised largely of athletes without cardiovascular evaluation or diagnosis during life, for whom we had no access to family screening or laboratory-based genetic analysis (12,42,43). These patient selection factors make any comparisons of clinical expression and outcome, between our autopsy series of competitive athletes and the hospital-based patient cohort, exceedingly difficult. Therefore, in this study, our multicenter cohort of clinically identified HCM patients was reserved solely for comparisons of prevalence.

The assembly of this large series of athletic field deaths required substantial reliance on news media accounts for the identification of cases. We recognize that this process could have created certain selection biases relevant to our autopsy-based series. For example, the sudden deaths of non-elite athletes are probably less likely to achieve media visibility, thereby underestimating the frequency with which sudden deaths occur. Due to these selection factors, as well as the absence of a systematic national reporting registry for such deaths, precise estimates of the prevalence of athletic field catastrophes in young athletes of all races are beyond the scope of the present study.

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