ORIGINAL RESEARCH

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Atrial Fibrillation in Hypertrophic Cardiomyopathy: Prevalence, Clinical Correlations, and Mortality in a Large High-Risk Population

Konstantinos C. Siontis, MD; Jeffrey B. Geske, MD; Kevin Ong, MD; Rick A. Nishimura, MD, MACC; Steve R. Ommen, MD, FACC; Bernard J. Gersh, MBChB, DPhil, MACC

***Background*—**Atrial fibrillation (AF) is a common sequela of hypertrophic cardiomyopathy (HCM), but evidence on its prevalence, risk factors, and effect on mortality is sparse. We sought to evaluate the prevalence of AF, identify clinical and echocardiographic correlates, and assess its effect on mortality in a large high-risk HCM population.

***Methods and Results*—**We identified HCM patients who underwent evaluation at our institution from 1975 to 2012. AF was defined by known history (either chronic or paroxysmal), electrocardiogram, or Holter monitoring at index visit. We examined clinical and echocardiographic variables in association with AF. The effect of AF on overall and cause-specific mortality was evaluated with multivariate Cox proportional hazards models. Of 3673 patients with HCM, 650 (18%) had AF. Patients with AF were older and more symptomatic ($P<0.001$). AF was less common among patients with obstructive HCM phenotype and was associated with larger left atria, higher E/e' ratios, and worse cardiopulmonary exercise tolerance (all P values<0.001). During median (interquartile range) follow-up of 4.1 (0.2 to 10) years, 1069 (29%) patients died. Patients with AF had worse survival compared to those without AF ($P<0.001$). In multivariate analysis adjusted for established risk factors of mortality in HCM, the hazard ratio (95% confidence interval) for the effect of AF on overall mortality was 1.48 (1.27 to 1.71). AF did not have an effect on sudden or nonsudden cardiac death.

***Conclusions*—**In this large referral HCM population, approximately 1 in 5 patients had AF. AF was a strong predictor of mortality, even after adjustment for established risk factors. **(*J Am Heart Assoc*. 2014;3:e001002 doi: 10.1161/JAHA.114.001002)**

Key Words: atrial fibrillation • hypertrophic cardiomyopathy • mortality

Atrial fibrillation (AF) is a common sequela of hypertrophic cardiomyopathy (HCM). Previous studies have estimated a 20% lifetime risk for development of AF in HCM, with prevalence as high as 40% in those older than 70 years.[1–5] Left ventricular outflow tract (LVOT) obstruction portends adverse prognosis and has been associated with increased risk of AF in some series,[6,7] whereas no association has been identified in others.[1,4] Echocardiographic markers of left atrial (LA) dysfunction, such as LA diameter, volume index (LAVI), fractional shortening, and electrocardiographic markers, such as P-wave dispersion, have also been correlated with AF in HCM, but evidence stems mostly from small cohorts.[8,9]

AF is an outcome-modifying factor in HCM that affects quality of life and increases morbidity and mortality.[1,10] Patients with AF are at increased risk of heart failure (HF) exacerbations and hospitalizations.[1,4] Therefore, in this retrospective analysis, we sought to better characterize the prevalence, clinical and echocardiographic correlates, and effect of AF on survival in a large, single-center referral cohort of HCM.

Methods

Patient Population

Adult patients with a diagnosis of HCM evaluated at the Mayo Clinic (Rochester, MN) between January 1975 and September 2012 who provided informed research consent were included in this retrospective study. The diagnosis of HCM was based on the presence of left ventricular (LV) hypertrophy in the absence of other primary cardiac or systemic etiologies.[11,12] Patient demographics, comorbidi-

From the Division of Cardiovascular Diseases (J.B.G., K.O., R.A.N., S.R.O., B.J.G.) and Department of Medicine (K.C.S.), Mayo Clinic, Rochester, MN.

Correspondence to: Bernard J. Gersh, MBChB, DPhil, MACC, Division of Cardiovascular Diseases, Department of Medicine, Mayo Clinic, 200 1st St SW, Rochester, MN 55905. E-mail: gersh.bernard@mayo.edu

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ties, echocardiographic data, laboratory studies, exercise testing, and medications were collected at the time of index visit. The study protocol was approved by the Mayo Clinic Institutional Review Board.

Definitions

The diagnosis of AF was based on an electrocardiogram (ECG) or Holter monitoring at the index visit, or by an established history of paroxysmal or chronic AF. All patients underwent comprehensive transthoracic echocardiographic evaluation.[13] LAVI was based on 2 orthogonal views using standard methodology.[14,15] We defined HCM as obstructive in patients who satisfied one of the following criteria based on echocardiography or hemodynamic cardiac catheterization: (1) rest LVOT peak gradient >30 mm Hg or (2) provoked (Valsalva maneuver, amyl nitrite, isuprel, or exercise) LVOT peak gradient >50 mm Hg. Labile LVOT obstruction was defined as the presence of provoked LVOT obstruction (peak gradient >50 mm Hg) in the absence of rest LVOT obstruction (peak gradient ≤30 mm Hg). A subset of patients underwent clinically indicated symptom-limited graded exercise testing using a motor-driven treadmill (Quinton, Seattle, WA) with an accelerated Naughton protocol. Survival status was determined by review of electronic medical records, patient correspondence, and Social Security Death Index, and cause of death (sudden cardiac death [SCD], nonsudden cardiac death, or noncardiac death) was adjudicated by review of medical examiner record when possible.

Statistical Analysis

We report patient demographics, clinical, laboratory, echocardiographic, and cardiopulmonary exercise testing data collected during the index evaluation at our institution. Categorical variables are reported as frequencies and percentages. Continuous variables are reported as means and SDs for normally distributed values or medians and interquartile ranges (IQRs) for non-normally distributed values. Normality was determined by visual inspection of distribution histograms. Two-sample Student *t* test and chi-square (χ^2) test were utilized as needed to evaluate associations of the aforementioned variables with AF.

Differences in survival in patients with and without AF were assessed with Kaplan-Meier's analyses, and *P* values were derived by log-rank testing. Survival in the AF group was also compared with the expected survival of an age- and sex-matched population derived from U.S. Census data. A priori power calculations for the survival analyses were not performed. We quantified the effect of AF on overall and cause-specific mortality with Cox's regression analyses with the use of hazard ratios (HRs) and 95% confidence intervals (CIs). Uni- and multivariate analyses, including adjustments for established demographic and clinical risk factors, are reported (age, sex, family history of SCD, New York Heart Association [NYHA] class, and obstructive phenotype). Further adjusting variables included septal thickness and percentage of predicted VO_2 on cardiopulmonary exercise testing. The use of either aspirin or warfarin and use of rhythm-controlling medications at the time of index evaluation were also serially added in the multivariate model. Information on other known risk factors of adverse outcome in HCM, such as history of ventricular tachycardia, blood pressure response to exercise, and B-type natriuretic peptide (BNP) levels, was available in small subsets of our population and therefore these were not included in the multivariate analysis.

In subgroup analysis, we excluded patients from the multivariate model who underwent septal myectomy and/or alcohol septal ablation before or after the index evaluation in order to evaluate whether septal reduction therapy modifies the effect of AF on outcomes. In addition, because our cohort spans several decades and it is possible that contemporary anticoagulation practices may alter the effect of AF on mortality, we performed subgroup mortality analysis focusing only on patients who underwent index evaluation in our institution during or after 2000. Statistical significance was set a priori at $P<0.05$. Patients with missing data were omitted from relevant analyses. All analyses were performed using JMP 9.0.1 software (SAS Institute Inc., Cary, NC).

Results

Demographics and AF Prevalence

Overall, 3673 patients (45% women) were included in this analysis (Table 1). Mean age at index evaluation was 55±16 years. Forty percent of the patients were NYHA class III or IV. The majority (71%) were on beta-blockade at the time of index evaluation. Median resting LVOT gradient was 29 (IQR, 8 to 70) mm Hg. One thousand three hundred and ten (36%) patients demonstrated resting obstruction and 1420 (39%) had labile obstruction. Therefore, 2730 (74%) patients were considered to have the obstructive HCM phenotype. Mean end-diastolic septal thickness was 18±6 mm Hg, and median LAVI was 44 (IQR, 34 to 58) cm^3/m^3 (Table 2). One thousand three hundred and forty patients underwent cardiopulmonary exercise testing; peak VO_2 was 20±7 mL/kg per minute (Table 3).

Table 1. Demographics, Clinical Characteristics, and Pharmacologic Therapy

Characteristic	All (n=3673)	AF (n=650)	No AF (n=3023)	*P* Value
Male	2012 (55)	367 (56)	1645 (54)	0.36
Age, y	55 (16)	60 (14)	54 (16)	<0.001
Family history of HCM	814 (22)	157 (24)	657 (22)	0.30
Family history of SCD	544 (16)	111 (18)	433 (15)	0.11
Systemic hypertension	1690 (46)	298 (46)	1392 (46)	0.96
Known CAD	607 (17)	127 (20)	480 (16)	0.02
Prior stroke	182 (5)	68 (10)	114 (4)	<0.001
Angina	1835 (56)	315 (54)	1520 (57)	0.17
Dyspnea	2662 (76)	519 (82)	2143 (75)	<0.001
Syncope	545 (16)	103 (17)	442 (16)	0.54
NYHA III/IV	1458 (40)	296 (46)	1162 (39)	0.002
Prior pacemaker	336 (9)	117 (18)	219 (7)	<0.001
Prior ICD	248 (7)	70 (11)	178 (6)	0.07
Prior myectomy	135 (4)	43 (7)	92 (3)	<0.001
Prior septal ablation	60 (2)	16 (2)	44 (1)	0.07
Beta-blocker	2458 (71)	502 (81)	1956 (69)	<0.001
ACEi/ARB	420 (32)	98 (40)	322 (30)	0.005
CCB	1370 (44)	309 (55)	1061 (41)	<0.001
Diuretic	489 (37)	130 (50)	359 (34)	<0.001
Amiodarone	194 (5)	154 (24)	40 (1)	<0.001
Disopyramide	302 (8)	104 (16)	198 (7)	<0.001
Sotalol	63 (2)	47 (7)	16 (1)	<0.001
Digoxin	35 (1)	27 (4)	8 (1)	<0.001
Aspirin	723 (20)	151 (23)	572 (19)	0.01
Warfarin	344 (9)	265 (41)	79 (3)	<0.001
BNP, median (IQR), pg/mL*	173 (71 to 383)	318 (131 to 558)	146 (63 to 314)	<0.001

Categorical variables are shown as n (%) and continuous variables as mean (SD), unless otherwise specified. ACEi indicates angiotensin-converting enzyme inhibitor; AF, atrial fibrillation; ARB, angiotensin receptor blocker; BNP, B-type natriuretic peptide; CAD, coronary artery disease; CCB, calcium-channel blocker; HCM, hypertrophic cardiomyopathy; ICD, implantable-cardioverter defibrillator; IQR, interquartile range; NYHA, New York Heart Association; SCD, sudden cardiac death.
*Data available for n=763 patients.

AF was diagnosed in 650 (18%) patients based on available information at the time of index visit (101 AF diagnoses by ECG or Holter monitoring).

Associations of AF With Clinical, Echocardiographic, and Laboratory Variables

Patients with AF were older (60±14 versus 54±16 years; *P*<0.001), more symptomatic (46 versus 39% NYHA class III or IV; *P*=0.002), and had higher BNP levels (median, 318 [IQR, 131 to 558] versus 146 [IQR, 63 to 314] pg/mL). History of coronary artery disease (CAD) and previous stroke were more prevalent among patients with AF. Use of beta-blockers, angiotensin-converting enzyme inhibitors, calcium-channel blockers, antiarrhythmics, and diuretics were higher in patients with AF (all *P*<0.01). Patients with AF were also more frequently taking warfarin and aspirin. At index evaluation, 54% of AF patients were on warfarin or aspirin, but this differed significantly between patients with index evaluation before and after January 1, 2000 (24% and 77%, respectively). As shown in Table 2, patients with AF had significantly thicker posterior LV wall, greater LAVI, higher right ventricular systolic pressure, higher E/e' ratio, and shorter mitral E-wave deceleration time. Moderate or severe mitral regurgitation was more frequently observed in patients with AF (*P*=0.01). AF was more common among patients with nonobstructive HCM, and this association remained significant even after adjustment for hypertension,

Table 2. Echocardiographic Assessment

Characteristic	All (n=3673)	AF (n=650)	No AF (n=3023)	*P* Value
Rest LVOT gradient, median (IQR), mm Hg	29 (8 to 70)	21 (0 to 59)	31 (9 to 71)	<0.001
Resting LVOT gradient >30 mm Hg	1310 (36)	202 (31)	1108 (37)	<0.001
Obstructive phenotype*	2730 (75)	441 (68)	2289 (76)	<0.001
LVEF, %	70 (9)	67 (11)	70 (8)	<0.001
LVEF<50%	83 (3)	38 (7)	45 (2)	<0.001
RVSP, mm Hg	38 (14)	43 (16)	36 (12)	<0.001
LVEDD, mm	45 (6)	46 (7)	45 (6)	<0.001
Septal thickness, mm	18 (6)	18 (5)	18 (6)	0.66
Posterior wall thickness, mm	13 (3)	13 (3)	12 (3)	0.007
Moderate or severe mitral regurgitation	586 (16)	130 (20)	456 (15)	0.01
LAVI, mL/m^{2}†	48 (23)	62 (38)	45 (16)	<0.001
Deceleration time, ms‡	228 (64)	216 (69)	231 (62)	<0.001
Medial E/e' ratio§	18 (8)	19 (9)	17 (8)	<0.001
Lateral E/e' ratio‖	14 (7)	14 (7)	14 (7)	0.37

Categorical variables are shown as n (%) and continuous variables as mean (SD), unless otherwise specified. AF indicates atrial fibrillation; IQR, interquartile range; LAVI, left atrial volume index; LVEDD, left ventricular end-diastolic dimension; LVEF, left ventricular ejection fraction; LVOT, left ventricular outflow tract; RVSP, right ventricular systolic pressure.
*Includes patients with resting LVOT obstruction (gradient >30 mm Hg) or labile LVOT obstruction (resting gradient <30 mm Hg and provoked gradient >50 mm Hg).
Data available for †n=1706; ‡n=2556; §n=1649; ‖n=824 patients.

CAD, LAVI, wall thickness, and surrogate measures of LV filling pressures. Patients with ejection fraction (EF) <50% (n=83) were more likely to have AF than those with EF ≥50% (*P*<0.001). Patients with AF had significantly worse exercise tolerance on cardiopulmonary exercise testing (Table 3).

Mortality Risk

During a median follow-up of 4.1 (IQR, 0.2 to 10; mean, 6.1; SD, 6.8) years, 1069 (29%) patients died. Annual mortality rates were 4.7%, 6.9%, and 4.4% in the overall HCM population, AF, and non-AF groups, respectively. Survival in both the AF and non-AF groups was worse than the expected survival of an age- and sex-matched U.S. population (*P*<0.001). In unadjusted Cox's proportional hazards analysis, AF conferred an increased risk of overall mortality in HCM patients (HR, 1.76; 95% CI, 1.51 to 2.03; *P*<0.001; Figure). The association remained highly significant after adjustments for age and sex (HR, 1.49; 95% CI, 1.28 to 1.72; *P*<0.001) and after serial additions of family history of SCD, NYHA functional class III to IV, and obstructive physiology in the model (Table 4). When septal thickness or the percentage of predicted VO_2 were added as covariates to the model, including age, sex, family history of SCD, NYHA functional class III to IV, and obstructive physiology, AF remained a significant predictor of all-cause mortality (HR [95% CI], 1.62 [1.36 to 1.92] and 1.93 [1.19 to 3.06], respectively). Use of antiarrhythmics and aspirin or warfarin at the time of index evaluation did not have any effect on the association between AF and all-cause or cause-specific mortality. At the time of last follow-up, 1198 and 202 patients had undergone septal myectomy and alcohol septal ablation, respectively, either before or after the index evaluation at our institution. Exclusion of these patients from the multivariate models did not appreciably alter effects estimates. In another subgroup analysis, we examined the association between AF and mortality in patients with index evaluation during or after 2000 (n=1997) and the effect remained statistically significant (HR. 1.66; 95% CI, 1.11 to 2.44).

Table 3. Cardiopulmonary Exercise Testing Data

Characteristic	All (n=1340)	AF (n=220)	No AF (n=1120)	*P* Value
VO_2 predicted, %	65 (20)	60 (20)	65 (19)	<0.001
Peak VO_2, mL/kg per minute	20 (7)	17 (6)	21 (7)	<0.001
Peak double product, mm Hg×beats/min	19 635 (7015)	17 205 (6416)	20 129 (7034)	<0.001

Cardiopulmonary exercise data were available for only a subset of the examined population. Variables are shown as mean (SD). AF indicates atrial fibrillation.

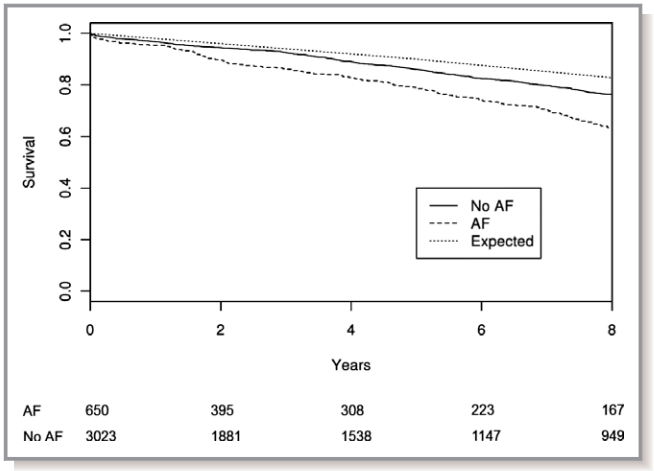

Figure. Kaplan-Meier's survival analysis for HCM patients with and without AF and expected survival of an age- and sex-matched general U.S. population. Survival was worse in HCM patients with AF than those without AF ($P<0.001$), and, even in the absence of AF, it was worse than the expected survival of an age- and sex-matched population ($P<0.001$). AF indicates atrial fibrillation; HCM, hypertrophic cardiomyopathy.

In a sensitivity analysis, when considering only patients with at least 8 years of available follow-up (n=1114), the age- and sex-adjusted effect of AF on survival was similar (HR, 1.50; 95% CI, 1.19 to 1.87; $P<0.001$) and remained significant after serial adjustments for family history of SCD, NYHA class, and obstructive physiology.

The specific cause of death was available in 344 patients. Our analyses did not detect significant associations between AF and SCD or nonsudden cardiac death in uni- or multivariate analyses, but there was a trend for increased risk for these endpoints in patients with AF (Table 4). In univariate analysis, AF was significantly associated with noncardiac mortality (HR, 1.78; 95% CI, 1.25 to 2.49; $P<0.001$), but the association lost nominal statistical significance after adjustment for other variables.

Discussion

This is the largest single-center study of HCM ever reported. In this retrospective analysis of >3500 patients with HCM, approximately 1 in 5 patients had current or established history of AF (permanent or paroxysmal) at the time of index evaluation. AF was associated with worse symptoms, worse exercise capacity and a significantly higher risk of death from any cause compared to patients without AF, even after accounting for known risk factors of mortality in HCM or use of antithrombotic, antiarrhythmic, and septal reduction therapies. Multiple echocardiographic parameters were found to be significantly correlated with the presence of AF, including higher LAVI, posterior wall thickness, medial E/e' ratio, and shorter mitral deceleration time. Interestingly, patients with obstructive physiology were less likely to have AF.

Prevalence of AF

Our study confirms previous smaller studies that have reported AF prevalence of ≈20% in HCM patients.[1–3] We

Table 4. Cox's Proportional Hazards Models for the Association of Atrial Fibrillation With Overall and Cause-Specific Mortality

Model Adjustments	Hazard Ratio (95% Confidence Interval)			
	All-Cause Death (n=1069)	Sudden Cardiac Death (n=79)*	Non-Sudden Cardiac Death (n=65)*	Non-Cardiac Death (n=200)*
None	1.76 (1.51 to 2.03)†	1.73 (0.96 to 2.92)	1.56 (0.77 to 2.89)	1.78 (1.25 to 2.49)†
Age, sex	1.49 (1.28 to 1.72)†	1.42 (0.79 to 2.41)	1.17 (0.58 to 2.17)	1.36 (0.95 to 1.90)
Age, sex, FHx SCD	1.51 (1.30 to 1.74)†	1.44 (0.80 to 2.45)	1.12 (0.55 to 2.09)	1.37 (0.96 to 1.91)
Age, sex, FHx SCD, NYHA class III/IV	1.48 (1.27 to 1.72)†	1.47 (0.81 to 2.50)	1.01 (0.48 to 1.93)	1.36 (0.96 to 1.91)
Age, sex, FHx SCD, NYHA class III/IV, obstructive phenotype‡	1.48 (1.27 to 1.71)†	1.45 (0.80 to 2.48)	1.01 (0.48 to 1.92)	1.34 (0.94 to 1.88)
+ Aspirin or warfarin	1.47 (1.26 to 1.71)†	1.58 (0.86 to 2.74)	1.27 (0.59 to 2.45)	1.59 (1.11 to 2.24)†
+ Antiarrhythmics	1.45 (1.24 to 1.69)†	1.34 (0.71 to 2.36)	0.93 (0.42 to 1.85)	1.56 (1.09 to 2.18)†
Age, sex, FHx SCD, NYHA class III/IV, obstructive phenotype‡, excluding patients with SRT§	1.44 (1.20 to 1.71)†	1.44 (0.75 to 2.60)	1.22 (0.49 to 2.61)	1.36 (0.92 to 1.95)

FHx SCD indicates family history of sudden cardiac death; NYHA, New York Heart Association; SRT, septal reduction therapy.
*Information on the cause of death was not available for n=630 patients.
†Statistically significant effect.
‡Includes patients with resting left ventricular outflow tract (LVOT) obstruction (gradient >30 mm Hg) or labile LVOT obstruction (resting gradient <30 mm Hg and provoked gradient >50 mm Hg).
§Septal myectomy and/or alcohol septal ablation before or after the index evaluation.

did not determine the precise point in time when patients developed AF (before or after the diagnosis of HCM), nor what the subsequent extent of incident AF was among patients at sinus rhythm at index evaluation. However, it has been previously shown that the diagnosis of HCM precedes the development of AF in the majority of patients,[1] which strongly suggests that the anatomic and physiological changes related to HCM, including diastolic dysfunction, myocardial ischemia, and autonomic dysregulation, predispose to development of AF.[16–18]

Echocardiographic Correlates

Left atrial enlargement is an established predictor of the development of AF in HCM, and our study confirms previous findings.[1,4,9,19] We have previously demonstrated the correlation between LAVI and invasive filling pressures.[20] Left atrial enlargement is a multifactorial process in HCM dependent upon obstructive physiology, intrinsic myocardial stiffness, mitral regurgitation, and rhythm disturbances.[21] Previous studies in general patient populations have clearly demonstrated associations between large left atria and risk for incident and recurrent atrial fibrillation,[22,23] but whether left atrial enlargement in HCM is a secondary phenomenon or precipitator of AF, or a combination of both, remains undetermined.

In this cohort, patients with obstructive HCM were less likely to have AF, regardless of the analyses and adjustments performed. A positive correlation between LVOT obstruction and AF has been previously found in some studies,[6,7] whereas there was no association in others.[1,4] It should be noted, however, that assessment of LVOT obstruction in HCM can be complex because of its dynamic nature.[24] In an attempt to control for such variability, in this study we utilized a comprehensive evaluation of obstruction, rather than relying on a single parameter. It is possible that nonobstructive HCM patients had worse diastolic function, in which case the finding of higher prevalence of AF in these patients would not be totally unexpected. Unfortunately, evaluation of diastolic function in HCM is challenging, especially when the underlying rhythm is AF.[25] Therefore, our findings of higher E/e' and shorter deceleration times among patients with AF should be interpreted with caution.

Cardiopulmonary Exercise Testing and AF

Reduced functional capacity, as assessed with metabolic exercise testing, confers important diagnostic and prognostic information in HCM.[26,27] In our cohort, patients with AF had significantly worse exercise capacity. Invariably, patients with HCM have some degree of restrictive ventricular filling, so the loss of coordinated atrial contraction can result in significant increases in LA pressure and the subsequent development of symptoms under exercise conditions. Also, both AF and decreased exercise capacity may be adverse consequences of progressive structural and functional changes in HCM. Our practice is to incorporate cardiopulmonary testing as part of the routine initial and follow-up evaluation of HCM patients to identify changes in exercise capacity (in addition to SCD stratification) and to optimize treatment.[28]

Mortality Risk

Evidence on the effect of AF on survival in patients with HCM has been limited thus far. A small retrospective study nearly 25 years ago showed no survival difference between patients with AF and those in sinus rhythm,[29] but more-contemporary evidence suggests that AF is an independent predictor of morbidity and mortality.[1,10] Our study demonstrates that AF is associated with a nearly 50% increased relative risk for overall mortality and offers compelling evidence on the association between AF and unfavorable disease outcomes.

Although SCD has been shown to be the most common cause of death in HCM,[2,30] AF-related mortality is mainly mediated by increases in HF and stroke-related deaths.[1,2] In this study, there was a consistent trend for increased risk of SCD and nonsudden cardiac death with AF based on the direction of effect estimates, but the associations did not reach nominal statistical significance. However, the effect of AF on mortality appears to be independent of traditional SCD risk factors, such as history of arrhythmic events, family history of SCD, and delivery of septal reduction therapies that have been shown to improve survival in HCM.[31,32] We detected an increased risk of noncardiac death in univariate analysis. This could be attributed to the risk of fatal AF-related embolic events or bleeding events in the setting of anticoagulation therapy. However, the association lost statistical significance in the multivariable model. Overall, the presence of AF might offer incremental prognostic information when incorporated in future risk prediction scores for overall mortality.

Study Limitations

The reported associations should be interpreted with caution, acknowledging that this is a retrospective analysis with inherent risk for different types of bias in such a study design. The analyzed population is derived from a single referral center and may represent a skewed HCM population with regard to severity of disease and comorbidities. Forty percent of the patients had NYHA class III or IV symptoms.[1,33] Also, previous studies in community-based HCM cohorts have reported annual death rates of ≈1%,[5,34–36] which contrasts with the mortality rate in our cohort. Because this is a referral

cohort, many patients did not receive longitudinal care at our institution, which limits follow-up, information on incident AF, and cause-of-death analysis. Cause-specific mortality analyses may therefore be underpowered to detect any associations. Information on stroke-related deaths was not available. Finally, our cohort spans more than 3 decades with evolving treatment practices, as suggested by a relative underuse of anticoagulation in patients with AF, especially those who underwent index evaluation before 2000. However, it should be noted that the subgroup analysis limited to patients evaluated at our institution in or after 2000 did not reveal any difference in the effect of AF on survival.

Conclusions

Paroxysmal or permanent AF was present in 20% of patients in this referral HCM cohort and was associated with higher symptom burden, worse exercise tolerance, and several echocardiographic markers, such as LAVI and LV wall thickness. Patients with nonobstructive HCM were more likely to have AF. Atrial fibrillation significantly increased the risk of death from any cause independently from other mortality risk factors, whereas no associations were detected between AF and sudden or nonsudden cardiac death.

Disclosures

None.

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