Outcomes in Atrial Fibrillation Patients With and Without Left Ventricular Hypertrophy When Treated With a Lenient Rate-Control or Rhythm-Control Strategy

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Although left ventricular (LV) hypertrophy has been proposed as a factor predisposing to atrial fibrillation (AF), its relevance to prognosis and selection of therapeutic strategies is unclear. We identified 2,105 patients with echocardiographic data on LV mass enrolled in the Atrial Fibrillation Follow-up Investigation of Rhythm Management (AFFIRM) trial. LV hypertrophy was defined as increased LV mass, stratified by American Society of Echocardiography criteria. The primary end point was all-cause mortality, secondary end point was as per AFFIRM trial definition, and tertiary end point was cardiovascular hospitalizations. We compared "strict" versus "lenient" rate control in patients with increased LV mass, and studied association of heart failure (HF) with preserved and decreased systolic function in patients with increased LV mass. Over 6 years, 332 deaths (15.7%) were reported. Adjusted hazard ratio (HR) of severely increased LV mass for all-cause mortality was 1.34 (95% confidence interval [CI] 1.01 to 1.79, p = 0.045) for the overall population and 1.61 (95% CI 1.09 to 2.37, p = 0.016) for the rhythm-control arm. Increased LV mass was a predictor of cardiovascular hospitalizations in the lenient rate-control group (HR 1.72, 95% CI 1.05 to 2.82, p = 0.03) but not in the strict rate-control group. Severely increased LV mass was predictive of cardiovascular hospitalizations in patients with HF with preserved (HR 1.8, 95% CI 1.0 to 3.2, p = 0.03) and decreased LV systolic function (HR 2.4, 95% CI 1.1 to 5.2, p = 0.02). Thus, LV hypertrophy is a significant independent predictor of mortality in patients with AF, especially those managed with rhythm control. In patients with LV hypertrophy, strict rate control may be associated with better outcomes than lenient rate control. LV hypertrophy portends higher cardiovascular morbidity in patients with AF and HF. **© 2014 Elsevier Inc. All rights reserved.** **(Am J Cardiol 2014;113:1159−1165)**

From a clinical standpoint, a clear case for rhythm control or stricter rate control could be made for patients with atrial fibrillation (AF) and associated systolic or diastolic ventricular dysfunction associated with left ventricular (LV) hypertrophy, but no data are available to support either strategy. There is a need to compare various rate-control approaches in carefully defined subgroups of patients with AF. Large case registries may provide additional opportunities to evaluate therapeutic strategies in AF subgroups. Consequently, we sought to investigate whether the selection of rhythm control versus strict or lenient rate control had an impact on mortality and morbidity in patients with AF with LV hypertrophy and associated diastolic or systolic dysfunction from the Atrial Fibrillation Follow-up Investigation of Rhythm Management (AFFIRM) trial.

Methods

We performed post hoc analysis of data from patients enrolled in the AFFIRM trial. A public use, limited-access data set devoid of patient identifiers was obtained from the National Heart, Lung and Blood Institute. None of the investigators are affiliated with the National Heart, Lung and Blood Institute or participated in the AFFIRM trial. The details of the AFFIRM study have been described previously.[1−8] In brief, this was a prospective trial (n = 4,060) comparing survival in patients with AF and at least 1 risk factor for stroke randomized to a strategy of rate control (n = 2,027) versus a strategy of rhythm

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See page 1165 for disclosure information.

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Table 1
Baseline characteristics of the study population stratified by the left ventricular (LV) mass

Variable	Overall Population (n = 2,105)	Normal LV Mass (n = 732)	Mildly Increased LV Mass (n = 349)	Moderately Increased LV Mass (n = 299)	Severely Increased LV Mass (n = 725)	p Value
Age	69.4 ± 8.1	70.1 ± 8.1	69.6 ± 8.1	69.3 ± 7.9	68.6 ± 8.2	0.004
Female gender	902 (42.8)	289 (39.4)	163 (46.7)	123 (41.1)	327 (45.1)	0.06
Body mass index (kg/m^2)	28.65 ± 5.91	26.80 ± 4.97	28.42 ± 5.34	29.33 ± 5.80	30.42 ± 6.52	<0.001
Medical history						
History of coronary artery disease	730 (34.6)	201 (27.4)	102 (29.2)	119 (39.8)	308 (42.4)	<0.001
HF	491 (23.3)	94 (12.8)	67 (19.2)	80 (26.7)	250 (34.4)	<0.001
HF with preserved EF	233 (11.0)	62 (8.4)	36 (10.3)	39 (13.0)	96 (13.2)	0.02
Hypertension	1,494 (70.9)	469 (64.0)	234 (67.0)	219 (73.2)	572 (78.9)	<0.001
Stroke	275 (13.0)	99 (13.5)	45 (12.8)	46 (15.3)	85 (11.7)	0.44
Diabetes	414 (19.6)	104 (14.2)	56 (16.0)	69 (23.0)	185 (25.5)	<0.001
Smoker	258 (12.2)	87 (11.8)	44 (12.6)	33 (11.0)	94 (12.9)	0.89
First episode of AF	1,252 (59.4)	434 (59.2)	205 (58.7)	189 (63.2)	424 (58.4)	0.55
Sinus rhythm at time of randomization	1,108 (52.6)	424 (57.9)	189 (54.1)	156 (52.1)	339 (46.7)	<0.001
Duration of AF >1 month	911 (43.28)	317 (43.31)	150 (42.98)	126 (42.14)	318 (43.86)	0.965
Medications before randomization						
β Blockers	897 (42.6)	295 (40.3)	138 (39.5)	136 (45.4)	328 (45.2)	0.11
Calcium channel blockers	823 (39.1)	280 (38.2)	121 (34.6)	129 (43.1)	293 (40.4)	0.13
Digoxin	1,130 (53.6)	364 (49.7)	172 (49.2)	176 (58.8)	418 (57.6)	0.002
Warfarin	1,793 (85.2)	618 (84.4)	293 (84.0)	259 (86.6)	623 (85.9)	0.67
Angiotensin-converting enzyme inhibitor or angiotensin receptor blocker	821 (39)	226 (30.8)	122 (34.9)	111 (37.1)	362 (49.9)	<0.001
Amiodarone	376 (17.8)	139 (18.9)	49 (14.0)	50 (16.7)	138 (19.0)	0.168
Sotalol	331 (15.7)	133 (18.1)	52 (14.9)	44 (14.7)	102 (14.0)	0.157
Class I antiarrhythmics	285 (13.5)	111 (15.1)	37 (10.6)	46 (15.3)	91 (12.5)	0.127
Echocardiographic parameters						
Left atrial size						<0.001
<4 cm	765 (36.3)	357 (48.7)	142 (40.6)	87 (29.1)	179 (24.6)	
4.1–4.5 cm	612 (29.0)	215 (29.3)	97 (27.7)	88 (29.4)	212 (29.2)	
≥4.6 cm	728 (34.5)	160 (21.8)	110 (31.5)	124 (41.4)	334 (46.0)	
LVEF (>50% = referent)						<0.001
>50%	1,587 (75.3)	627 (85.6)	283 (81.0)	219 (73.2)	458 (63.1)	
40%–49%	262 (12.4)	67 (9.1)	34 (9.7)	40 (13.3)	121 (16.6)	
30%–39%	154 (7.3)	27 (3.6)	18 (5.1)	20 (6.6)	89 (12.2)	
<30%	102 (4.8)	11 (1.5)	14 (4.0)	20 (6.6)	57 (7.8)	
Mitral regurgitation	446 (21.1)	131 (17.9)	74 (21.2)	66 (22.0)	175 (24.1)	0.03
Rhythm-control arm	1,061 (50.4)	398 (54.3)	150 (42.9)	148 (49.5)	365 (50.3)	0.006

Data are presented as mean ± SD or n (%).

control (n = 2,033). Eligible patients were either aged ≥65 years or had at least 1 of the following risk factors for stroke or death: hypertension, diabetes, heart failure (HF), previous stroke or transient ischemic attack, systemic embolism, left atrial diameter >50 mm by echocardiography, LV ejection fraction (EF) <0.40, or fractional shortening <25% determined by any technique.[1,2]

We identified 2,105 patients with echocardiographic data on LV mass. Patients with incomplete echocardiographic data were excluded (n = 1,945). LV mass measurements were defined as per the American Society of Echocardiography's guidelines on chamber quantification. Mass was calculated by subtraction of the LV cavity volume from the volume enclosed by the epicardium to obtain LV muscle or shell volume. The shell volume was then converted to muscle mass by multiplying by myocardial density according to the following formula[9,10]:

$$\text{LV mass} = 0.8 \times \{1.04\,[(\text{LVIDd} + \text{PWTd} + \text{SWTd})^3 - (\text{LVIDd})^3]\} + 0.6,$$

where LVIDd denotes left ventricular internal diastolic diameter; PWTd, posterior wall thickness at end-diastole; and SWTd, septal wall thickness at end-diastole. LV mass was categorized as normal, mildly abnormal, moderately abnormal, or severely abnormal according to the American Society of Echocardiography criteria, which varied by gender.[9,10]

To assess the relation between degree of rate control and outcomes in relation to LV mass, we performed a subanalysis including patients with available LV mass data and without pacemaker insertion before randomization, originally enrolled in the rate-control arm with documented AF both at baseline and at 2-month visit, with available data of heart rate at rest at both visits. This cohort (n = 366) was stratified according to the degree of rate control, with adequately rate-controlled patients included in the strict rate-control group (n = 105) and the remainder (n = 261) in the lenient rate-control group. Adequate control at 2 months was defined as heart rate at rest ≤80 and postexercise heart rate ≤110 beats/min after 6 minutes of exercise.[11] To maintain power in this relatively small subgroup, LV mass was classified into just 2 categories: normal or mildly increased LV mass was

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