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Racial Differences in the Incidence of and Risk Factors for Atrial Fibrillation in Older Adults: The Cardiovascular Health Study

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Abstract

The authors examined whether differential associations of risk factors with atrial fibrillation (AF) by race could explain the lower AF incidence in blacks. Baseline risk factor information was obtained from interview, clinic examination, and echocardiography in 4774 white and 911 black Cardiovascular Health Study participants ≥65 years old without history of AF at baseline in 1989–90 or 1992–93. Incident AF was determined by hospital discharge diagnosis or annual study electrocardiogram. Cox regression was used to assess associations of risk factors and race with incident AF. During a mean 11.2 years of follow-up, 1403 whites and 182 blacks had incident AF. Associations of all examined risk factors were similar in both races, except left ventricular posterior wall thickness, which was more strongly associated with AF in blacks (per 0.2 cm, HR for blacks: 1.72, 95%CI: 1.44, 2.06; whites: 1.30, 95%CI: 1.18, 1.43). Overall, the relative risk of AF was 25% lower in blacks than whites after adjustment only for age and sex (HR: 0.75, 95%CI: 0.64, 0.87), and 45% lower after adjustment for all considered risk factors (HR: 0.55, 95%CI: 0.35, 0.88). Differential associations of the risk factors considered with incident AF by race do not explain the lower AF incidence in blacks.

Keywords

Atrial fibrillation; race; incidence; risk factors

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INTRODUCTION

Atrial fibrillation (AF) is the most common cardiac arrhythmia, currently affecting over 2 million people in the US and expected to affect 12 million by 2050.[1–3] The prevalence of AF increases with age, and AF is associated with a 5-fold increase in the relative risk of ischemic stroke and a 1.5 to 2.0 fold increase in the relative risk of death.[1, 4, 5]

Most studies examining AF have been performed on predominantly white populations, and relatively less is known about AF incidence and risk factors among non-white populations. Available studies suggest that despite a higher level of traditional cardiovascular risk factors and a higher incidence of stroke, blacks have a lower incidence of AF than whites.[6–8] A 1997 analysis in the Cardiovascular Health Study (CHS) reported a lower incidence rate of AF among blacks than among other participants (12.0 vs 19.5 per 1000 person-years, respectively). However, that study was limited to the relatively small number of black participants in the original CHS cohort and was based on an average follow-up of only 3 years; it did not include participants in the large black cohort added to CHS in 1992–93. The goals of this analysis were to determine the age and sex-specific incidence of AF in white and black participants in both CHS cohorts with up to 19 years of follow-up, and to determine whether AF risk factors differed between blacks and whites. If so, such a finding might explain the reported lower AF incidence in blacks.

METHODS

Study design and setting

The Cardiovascular Health Study is a prospective cohort study of risk factors for cardiovascular disease in community-dwelling adults 65 years or older. In 1989–90, 5201 participants, including 246 blacks, were recruited from four field centers, located in Forsyth County, NC; Sacramento County, CA; Washington County, MD; and Pittsburgh, PA. In 1992–93, an additional 687 black participants were recruited. The CHS design and recruitment are described in detail elsewhere.[9, 10]

Baseline examination

Participants underwent a baseline study examination that included height and weight measurement, lung function examination, blood pressure measurement by a random-zero sphygmomanometer, and 12-lead resting electrocardiogram (ECG). Race was determined by self-report. Information was collected on medication use, and blood was collected with the participant in the fasting state, from which glucose, N-terminal proBNP (N-terminal pro-B-type natriuretic peptide), and cystatin C levels were measured.[11, 12] Hypertension was determined by systolic blood pressure ≥140mmHg, diastolic blood pressure ≥90mmHg, or use of antihypertensive medications by a participant who reported a physician diagnosis of hypertension. Diabetes was determined by either a fasting glucose level of ≥126mg/dl or use of an oral hypoglycemic agent or insulin. Forced expiratory volume in 1 second was measured upon entry to the study in the original cohort, and after one year of follow-up in the black cohort. Left ventricular (LV) posterior wall thickness, percent fractional shortening, and left atrial dimension were determined by echocardiography, which was performed upon entry to the study in the original cohort and after two years of follow-up in the black cohort.[13]

Identification of incident atrial fibrillation

The outcome of incident AF was defined as including both incident AF and incident atrial flutter. Incident AF was ascertained during follow-up from two sources. First, participants underwent annual study ECGs between 1989 and 1999 that were read by the CHS

Electrocardiography Reading Center. Second, hospital discharge International Classification of Disease, revision 9 (ICD-9) codes were reviewed for a diagnosis code for AF or atrial flutter (427.3, 427.31, or 427.32). Previous validation of hospital discharge ICD-9 codes among CHS participants revealed a positive predictive value of 98% and a sensitivity of 71% for AF.[14] AF that occurred during a hospitalization for open-heart surgery was excluded; however, if subsequent hospitalizations or study examinations revealed AF unrelated to heart surgery, the date of the subsequent AF occurrence was used as the date of incident AF.

Statistical analyses

Analyses were limited to white and black participants without prevalent AF at baseline. Hispanics were included in the analyses if they self-identified as white or black. Age and sex specific incidence rates of AF in white and black participants were calculated as the number of cases divided by the number of person years in each age-sex-race category. Participants began accruing time at risk upon their baseline CHS examination and were followed to the earliest of: diagnosis of AF, death, or June 30, 2008. Age- and sex-adjusted Cox proportional-hazards models were used to assess the association between potential risk factors and incident AF. Models for forced expiratory volume in one second (FEV1) and left ventricular mass were also adjusted for height. Because in the black cohort pulmonary function tests were administered after one year of follow-up, and echocardiograms after two years, models of FEV1 and echocardiographic variables were left censored at the date of the relevant exam for this cohort. Continuous risk factors were scaled to their standard deviation to facilitate comparisons of hazard ratios across risk factors measured on different scales. NT-proBNP was log-transformed because its distribution was right skewed (median: 260 pg/dL; range: 5 – 23,445 pg/dL). To evaluate racial differences in the associations of risk factors with incident AF, we tested interaction terms for each risk factor with race. A significance threshold of $P < 0.003$ was used to account for multiple comparisons (n=15). Scaled Schoenfeld residuals plotted over time were used to assess the assumption of proportional hazards in each model. Cox regression was used to estimate the relative risk of AF in blacks versus whites after adjustment for all risk factors.

RESULTS

Of the 5888 participants in CHS, 167 had prevalent AF at baseline, and a further 36 were not white or black and were excluded from the analyses. Baseline characteristics of the remaining 5685 participants are shown in Table 1. Blacks had higher prevalence of hypertension and diabetes, a higher average BMI, and lower average educational attainment and family income than whites.

During an average of 11.2 years of follow-up, there were 1585 incident AF cases identified, 1403 among whites and 182 among blacks. During the period when participants underwent annual ECG examinations (1990–99), the proportion of AF cases first identified by study ECG rather than ICD-9 code was similar by race (19% for whites, 17% for blacks). Age and sex specific incidence rates are detailed in Table 2. The incidence of AF increased with age, was higher for men than women, and was lower for blacks than whites.

Table 3 summarizes the results of individual age- and sex-adjusted Cox models for each risk factor, stratified by race, and the hazard ratio for interaction by race. For both whites and blacks, high levels of body mass index (BMI), systolic blood pressure, NT-proBNP, cystatin C, and larger left ventricular posterior wall thickness and left atrial dimension were associated with a higher relative risk of AF. Additionally, higher levels of FEV1 were associated with a lower relative risk of AF for both races. None of the risk factors was more weakly associated with AF in blacks than whites; conversely, left ventricular posterior wall

thickness was more strongly associated with incident AF in blacks than in whites. There was no evidence of non-proportional hazards for any of the covariates, thus the hazard ratios presented approximate relative risks.

After adjustment for age and sex, the relative risk of AF was 25% lower in blacks than in whites (hazard ratio (HR): 0.75, 95% confidence interval (CI): 0.64, 0.87). After additional adjustment for all of the risk factors in Table 3, the relative risk of AF was 45% lower in blacks (HR 0.55, 95%CI: 0.35, 0.88).

DISCUSSION

During an average of 11.2 years of follow-up, the relative risk of AF was 25% lower in blacks than whites after adjustment for only age and sex, and was 45% lower after full risk factor adjustment. The risk factors for AF examined in this study do not explain the lower incidence of AF in blacks than whites. Only the association of left ventricular posterior wall thickness with AF was stronger in blacks than whites; all other risk factors examined were similarly associated with AF in the two race groups.

Strengths of the study include the large population of elderly participants, the large number of black participants, and the detailed and thorough assessment of risk factors. This study also includes a number of limitations. Because AF is often transient, annual study ECGs might not detect it, and because AF may be asymptomatic, participants who experienced AF may not have sought medical care.[15] Thus, because we identified cases from annual ECGs and hospitalization records, we may have failed to identify some cases of incident AF. Additionally, the date of AF onset is difficult to estimate accurately, as participants may have been in AF for some time prior to detection.

Race specific incidence rates from CHS for both whites and blacks were similar to those reported in ARIC.[8] Incidence rates in CHS among whites were similar to those reported from Olmstead County, Minnesota,[3] but consistently higher than those reported from the Framingham Heart Study.[16] The differences may be due to less frequent study ECGs in the Framingham Study (every two years, as opposed to every year in CHS), increasing AF incidence over time,[12] and greater clinical recognition of AF during the period of the present study compared with the earlier period of time (1948–1986) covered in the Framingham report.

Despite higher prevalence of hypertension and diabetes, and higher values on average for BMI, blacks in CHS were less likely than whites to develop AF. Reasons for this apparent paradox are unclear. Less frequent use of medical care by blacks would decrease the opportunity for AF detection. Indeed, on average, US blacks have poorer access to health care than whites, and among those over age 65 who are Medicare eligible, blacks are more likely than whites to have no supplemental insurance coverage (16% vs 10% in 2007).[17] Differences in access to or use of health care may account in part for lower rates of AF detection in blacks.

Past studies have shown that between 12 and 47% of new-onset AF cases are asymptomatic,[14, 18] and such cases are unlikely to be detected by means other than ECG screening. It has been hypothesized that blacks may suffer a disproportionately high rate of asymptomatic AF and are thus less likely than whites to seek medical care for AF.[19] In this study, the fact that screening ECGs accounted for a similar proportion of incident AF cases in blacks as in whites does not provide support for this explanation. Further study within a large, racially diverse population undergoing longer-term routine ECG screening is needed to determine whether there indeed may be racial differences in the incidence of asymptomatic AF.

An analysis in CHS and ARIC examining the association between European ancestry and incident AF suggests a possible genetic explanation for the lower rate of AF among blacks. Among blacks, each 10% increase in European ancestry was associated with a 17% higher relative risk of incident AF,[20] suggesting that perhaps whites are genetically predisposed to AF; however, further study exploring genetic variants among both races is needed.

Conclusions

This analysis suggests that differences in the associations of a wide variety of risk factors with AF by race do not explain the lower incidence of AF in blacks. Further study, potentially incorporating frequent, routine ECG monitoring and a consideration of genetic variation, is needed to further understand the racial difference in AF incidence.

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Table 1

Baseline Characteristics* of White and Black Cardiovascular Health Study Participants Without a History of Atrial Fibrillation, 1989–90, 1992–93

	Whites (n=4,774)		Blacks (n=911)	
Characteristic	**Mean or %**	**SD**	**Mean or %**	**SD**
Age, y	72.7	5.5	72.9	5.7
Male	43%		37%	
BMI (kg/m^2)	26.3	4.5	28.5	5.5
Height (cm)	165	9.5	165	9.2
Systolic blood pressure (mmHg)	135	21	143	23
Hypertension	63%		77%	
Fasting glucose (mg/dl)	109	32	119	55
Diabetes	14%		26%	
PR interval (ms)	169	31	173	33
≥1 Alcoholic drink/week	53%		34%	
Drinks/week**	4.9	8.1	4.6	9.7
Natural log of NT-proBNP	5	1.1	5	1.3
FEV1 (L)	2.1	0.7	1.8	0.6
Cystatin C (mg/L)	1.1	0.3	1.1	0.5
High school graduate	74%		55%	
LV fractional shortening (%)	42	8.2	42	9.5
LV posterior wall thickness in diastole (cm)	0.8	0.1	0.9	0.2
Left atrial dimension (cm)	3.9	0.7	3.9	0.6

BMI indicates body mass index; FEV1, forced expiratory volume in 1 second; LV, left ventricular; NT-proBNP, N-terminal pro-B-type natriuretic peptide; SD, standard deviation. Data were missing in 2% or fewer of participants for all characteristics except diabetes (missing in 27 whites and 34 blacks), fasting glucose (29 and 47), NT-proBNP (1131 and 195), cystatin C (607 and 67), left ventricular wall thickness (1682 and 342), and left atrial dimension (181 and 199).

* FEV1 was ascertained at baseline in the original cohort, and after one year of follow-up in the black cohort. Echocardiography was performed at baseline in the original cohort and after two years of follow-up in the black cohort.

** Among those with ≥1 drink/week.

Table 2

a. Incidence Rates of Atrial Fibrillation Among White and Black Women in the Cardiovascular Health Study, 1989–2008

	White Women				Black Women			
Age Group	AF Cases	Person -Years	Incidence (per 1,000)	95% CI	AF Cases	Person -Years	Incidence (per 1,000)	95% CI
65–69	18	2705	**6.7**	4.2, 10.6	7	506	**13.8**	6.6, 29.0
70–74	93	7386	**12.6**	10.3, 15.4	14	1407	**9.9**	5.9, 16.8
75–79	182	9317	**19.5**	16.9, 22.6	30	1801	**16.7**	11.6, 23.8
80–84	222	8397	**26.4**	23.2, 30.2	30	1506	**19.9**	13.9, 28.5
85–89	156	4494	**34.7**	29.7, 40.6	25	810	**30.9**	20.9, 45.7

b. Incidence Rates of Atrial Fibrillation Among White and Black Men in the Cardiovascular Health Study, 1989–2008

	White Men				Black Men			
Age Group	AF Cases	Person -Years	Incidence (per 1,000)	95% CI	AF Cases	Person -Years	Incidence (per 1,000)	95% CI
65–69	23	1515	**15.2**	10.1, 22.8	6	299	**20.0**	9.0, 44.6
70–74	89	4643	**19.2**	15.6, 23.6	10	814	**12.3**	6.6, 22.8
75–79	162	5859	**27.7**	23.7, 32.3	14	944	**14.8**	8.8, 25.0
80–84	200	5151	**38.8**	33.8, 44.6	19	731	**26.0**	16.6, 40.7
85–89	136	2758	**49.3**	41.7, 58.3	8	333	**24.0**	12.0, 48.1

AF indicates atrial fibrillation; CI, confidence interval

Table 3

Age and Sex Adjusted Hazard Ratios of Incident Atrial Fibrillation in the Cardiovascular Health Study by Race According to Baseline Characteristics,* 1989–2008 – Each Line Represents a Separate Model

	Whites			Blacks			Interaction		
Variable	**N**	**HR**	**95% CI**	**N**	**HR**	**95% CI**	**HR**	**95% CI**	***P*-value**
BMI (per 5 units)	4760	**1.10**	1.03, 1.17	907	**1.31**	1.15, 1.49	**1.20**	1.04, 1.38	0.011
Height (per 10 cm)	4766	**1.32**	1.21, 1.43	909	**1.15**	0.92, 1.45	**0.89**	0.75, 1.05	0.158
Systolic BP (per 20 mmHg)	4767	**1.15**	1.09, 1.21	909	**1.15**	1.01, 1.30	**0.99**	0.87, 1.13	0.931
Hypertension	4745	**1.40**	1.26, 1.56	864	**1.55**	1.07, 2.23	**1.12**	0.77, 1.63	0.555
Glucose (per 40 mg/dl)	4725	**1.19**	1.12, 1.27	891	**1.06**	0.95, 1.19	**0.90**	0.79, 1.02	0.103
Diabetes	4747	**1.56**	1.35, 1.80	877	**1.26**	0.90, 1.77	**0.81**	0.56, 1.17	0.257
PR Interval (per 30 ms)	4768	**1.02**	1.01, 1.04	909	**0.99**	0.93, 1.07	**0.98**	0.91, 1.05	0.505
Alcohol (per drink per day)	4754	**1.01**	0.95, 1.06	907	**0.78**	0.57, 1.07	**0.77**	0.56, 1.05	0.098
NT-proBNP (per log-pg/dL)	3643	**1.63**	1.53, 1.73	716	**1.64**	1.43, 1.88	**1.02**	0.89, 1.18	0.748
FEV1 (per 0.5 L)**	4688	**0.80**	0.77, 0.84	703	**0.74**	0.62, 0.87	**0.91**	0.78, 1.06	0.249
Cystatin C (per 0.5 mg/L)	4167	**1.39**	1.30, 1.49	844	**1.34**	1.19, 1.50	**0.98**	0.86, 1.12	0.772
Graduated from High School	4763	**0.92**	0.82, 1.04	906	**0.89**	0.66, 1.20	**0.99**	0.72, 1.35	0.926
LV percent fractional shortening (per 10%)	3046	**0.89**	0.82, 0.97	541	**0.90**	0.70, 1.16	**1.01**	0.78, 1.31	0.951
LV posterior wall thickness in diastole (per 0.2 cm)**	3088	**1.30**	1.18, 1.43	555	**1.72**	1.44, 2.06	**1.32**	1.10, 1.58	0.003
Left Atrial Dimension (per 0.5 cm)**	4586	**1.26**	1.21, 1.32	695	**1.29**	1.12, 1.49	**1.01**	0.87, 1.17	0.886

BMI indicates body mass index; BP, blood pressure; FEV1, forced expiratory volume in 1 second; LV, left ventricular; NT-proBNP, N-terminal pro-B-type natriuretic peptide.

* FEV1 was ascertained at baseline in the original cohort, and after one year of follow-up in the black cohort. Echocardiography was performed at baseline in the original cohort and after two years of follow-up in the black cohort.

** Adjusted additionally for height.