American Journal of Epidemiology

Vol. 167, No. 6
DOI: 10.1093/aje/kwm365
Advance Access publication January 21, 2008

Original Contribution

Baldness and Myocardial Infarction in Men

The Atherosclerosis Risk in Communities Study

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Received for publication August 10, 2007; accepted for publication November 14, 2007.

Because hair loss may be a surrogate measure of androgenic activity—possibly a determinant of coronary atherosclerosis—several studies have explored the presence and magnitude of an association between male pattern baldness and myocardial infarction (MI). In particular, vertex baldness, but not frontal baldness alone, was strongly associated with incident MI in a large, hospital-based, case-control study. The authors examined these associations in a cross-sectional sample of 5,056 men aged 52–75 years, of whom 767 had a history of MI. The sample was derived from the Atherosclerosis Risk in Communities (ARIC) Study (1987–1998). As compared with a baldness-free reference group, the estimated odds ratios for prevalent MI from a multivariable model were 1.28 (frontal baldness), 1.02 (mild vertex baldness), 1.40 (moderate vertex baldness), and 1.18 (severe vertex baldness). Other regression models have yielded similar results, including the absence of a monotonic "dose-response relation" between the extent of vertex baldness and prevalent MI. The authors also examined the relation of baldness pattern to carotid intimal-medial thickness, a measure of atherosclerosis, among those who were free of clinical cardiovascular disease. The estimated mean differences in carotid intimal-medial thickness between groups of men with various types of baldness and their baldness-free counterparts were all close to zero. The results of this study suggest that male pattern baldness is not a surrogate measure of an important risk factor for myocardial infarction or asymptomatic atherosclerosis.

alopecia; carotid artery diseases; myocardial infarction

Abbreviations: ARIC, Atherosclerosis Risk in Communities; IMT, intimal-medial thickness; MI, myocardial infarction.

The possible relation between balding and coronary heart disease has attracted interest since the 1960s (1), if not earlier. At least 10 studies have addressed the topic, some of which were pioneering, preliminary research, whereas others have met contemporary standards for epidemiologic studies (1–10). Unfortunately, that small body of literature has generated inconsistent findings, and the titles of recent reviews reflect the controversy (11, 12). The following questions, in particular, await empirical answers: 1) Is any baldness, regardless of pattern, meaningfully associated with myocardial infarction (MI) or atherosclerotic burden? 2) Are frontal baldness and vertex baldness equivalent in that respect? 3) Does the extent of vertex baldness matter?

In 1993, Lesko et al. (5) published results from a large, hospital-based, case-control study of male pattern baldness and myocardial infraction. Vertex baldness, but not frontal

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baldness alone, was strongly associated with MI in a monotonic ''dose-response'' fashion: the greater the extent of vertex baldness, the greater the risk of MI. We attempted to replicate those findings in a cross-sectional sample, derived from a population-based cohort. We also extended the question to address the relation between baldness pattern and carotid intimal-medial thickness, a measure of the systemic burden of atherosclerosis (13, 14).

MATERIALS AND METHODS

Sample

The sample was derived from the National Heart, Lung, and Blood Institute-supported Atherosclerosis Risk in Communities (ARIC) Study cohort, a study of atherosclerosis and cardiovascular disease in four US communities: Forsyth County, North Carolina; the city of Jackson, Mississippi; eight northwestern suburbs of Minneapolis, Minnesota; and Washington County, Maryland. Details of the study design were described (15). In brief, over a 3-year period (1987–1989), each field center recruited and examined about 4,000 community residents, men and women between the ages of 45 and 64 years. African-American residents were recruited exclusively in Jackson and were oversampled in Forsyth County, whereas the samples from Minneapolis and Washington County comprised mainly Whites. The cohort was reexamined three times and was followed for cardiovascular disease endpoints (16, 17). Motivated by the article of Lesko et al. (5), we added their classification method of male pattern baldness to the fourth examination of the ARIC cohort (1996–1998). The institutional review boards of participating institutions approved the study, and each participant signed a consent form.

Of the 5,148 men who attended the fourth cohort examination, we excluded 92 observations: 55 men who were treated for baldness, 30 who received chemotherapy recently, and seven for whom myocardial infarction status remained uncertain. The maximal sample size was therefore 5,056 observations.

Baldness scale

Baldness pattern was classified according to the Hamilton baldness scale, as modified by Norwood (18, 19). A trained technician in each clinic observed the participant's head from two angles (side and top), compared the natural hair pattern with a series of 12 figures, and chose the best matching figure.

Like Lesko et al. (5), we collapsed the 12 categories of the Hamilton scale into five: no baldness (I, II); frontal baldness alone (IIa, III, IIIa, IVa); mild vertex baldness (III vertex, IV); moderate vertex baldness (V, Va); and severe vertex baldness (VI, VII). In a sample of original and repeated classifications ($n = 164$ pairs), overall agreement on these categories was good (85 percent; kappa statistic: 0.81). Agreement on just three baldness categories (none, frontal, vertex) was very good (93 percent; kappa: 0.87).

Prevalent myocardial infarction

Prevalent MI at the time of the fourth examination of the cohort was defined as self-reported history of physician-diagnosed MI, silent MI by electrocardiography, or hospitalized MI since the baseline examination, ascertained by the study surveillance system (16).

Intimal-medial thickness of the carotid arteries

The intimal-medial thickness (IMT) of the extracranial carotid arteries was measured according to published methods (20, 21). In brief, trained technicians scanned the arteries on each side of the neck by high-resolution B-mode ultrasound and videotaped, from fixed angles, three 1-cm segments: the common carotid artery (proximal to the dilation of the carotid bulb), the bifurcation (proximal to the flow divider), and the internal carotid artery (distal to the flow divider). Using magnified images, trained readers later identified two arterial boundaries (intima-blood; media-adventitia) and measured the intimal-medial thickness at each segment. Missing values from nonvisualized sites were replaced by imputed values (22). We modeled the average thickness of the far wall of six arterial segments (three segments on each side $\times$ 2), as measured during the fourth cohort examination.

Other variables

To replicate the analysis of Lesko et al., we tried to select similarly defined covariates, when available from interviews or physical measurements. With few exceptions, specified below, covariates were measured during the fourth examination (at which the baldness pattern was classified). Smoking status was classified as current smoker, former smoker, or never smoker. Family history of MI was defined as self-reported maternal or paternal history of MI (baseline examination). Use of low density lipoprotein cholesterol-lowering and antihypertension medications was determined from self-reported information or medication bottles. Educational level was classified into three categories (baseline examination): less than high school, high school graduate, and education beyond high school.

Body mass index was calculated from weight and height: (weight (pounds)/2.20)/(height (cm)/100)^2. (One pound = 0.45 kg). Prevalent diabetes mellitus was defined as nonfasting glucose of $>$200 mg/dl, fasting glucose of $>$126 mg/dl, a history of diabetes, or pharmacologic treatment of diabetes. The concentration of high density lipoprotein cholesterol was measured by the method of Warnick et al. (23). Cardiovascular disease-free men should have had neither coronary heart disease nor stroke.

Analysis

To the extent possible, we replicated key tables in the article of Lesko et al. (5). First, we examined the distribution of baldness pattern by prevalent MI status and computed prevalence odds ratios for any baldness and for vertex baldness of any severity (table 1). Second, we examined the relation of baldness (in three categories: none, frontal, vertex)

TABLE 1. Distribution of baldness pattern according to myocardial infarction status, the Atherosclerosis Risk in Communities Study, 1987–1998

	Myocardial infarction ($n = 767$)		No myocardial infarction ($n = 4,289$)		Odds ratio	95% confidence interval
	No.	%*	No.	%*		
Baldness category†						
I (none)	107	14	680	16		
II (little frontal to none)	108	14	741	17		
IIa (only frontal)	22	3	109	3		
III (only frontal)	64	8	273	6		
IIIa (only frontal)	9	1	87	2		
III vertex	94	12	515	12		
IV (vertex)	61	8	372	9		
IVa (only frontal)	17	2	81	2		
V (vertex)	65	8	275	6		
Va (vertex)	38	5	202	5		
VI (vertex)	95	12	563	13		
VII (vertex)	85	11	373	9		
Unknown	2	0	18	0		
Any baldness (categories IIa–VII)	550	72	2,850	66	1.28	1.08, 1.51
No baldness (categories I and II)	215	28	1,421	33	Referent	
Any vertex baldness (categories III vertex, IV, and V–VII)	438	57	2,300	54	1.15	0.98, 1.34
No vertex baldness	327	43	1,971	46	Referent	

* Percentages might not add to 100 because of rounding.
† Hamilton baldness scale.

to several risk factors for coronary heart disease (table 2), which later played the role of covariates in logistic and linear regression models. Third, we regressed the log-odds of prevalent MI on baldness pattern (''none'' serving as the referent) and computed marginal (''crude'') and conditional (''adjusted'') odds ratios (table 3). Fourth, we regressed the log-odds of prevalent MI on age (continuous) and a binary baldness pattern (severe vertex vs. none) and computed odds ratios for severe vertex baldness within strata of risk factors for coronary heart disease (table 4).

To study the relation between baldness pattern and asymptomatic atherosclerosis, we restricted the sample to men who were free of clinical cardiovascular disease ($n = 2,248$) and fit weighted linear regression models: The mean IMT over six arterial sites was regressed on baldness pattern alone and on baldness pattern and covariates, weighting each observation according to the number of nonimputed IMT values (the number of observed sites divided by 6). From these models, we computed marginal and conditional mean differences of IMT between various types of baldness and the referent category of ''none'' (table 5).

RESULTS

Of the 5,056 men who contributed data to this analysis, 1,636 (32 percent) had little or no evidence of baldness (Hamilton categories I and II). A total of 662 (13 percent) were classified as having frontal baldness alone; 2,738 (54 percent) were classified as having vertex baldness; and 20 (<1 percent) could not be classified. Severe vertex baldness was observed in 1,116 participants (22 percent of the sample). A total of 767 participants (15 percent) were classified as having prevalent MI. The mean and median age in the sample were both 63 years (range: 52–75 years).

Table 1 replicates the first table in the article by Lesko et al. (5). The distribution of the Hamilton baldness scale in men who were classified as prevalent MI did not differ much from its distribution in their MI-free counterparts. Likewise, the marginal (''crude'') associations of MI status with baldness status (any vs. none to ''little frontal'') and between MI status and vertex baldness status were weak. For example, the estimated odds ratio for ''any vertex baldness'' was only 1.15, much smaller than the estimate we computed from the case-control study of Lesko et al. (5): $(214/175)/(442/594) = 1.64$.

Like Lesko et al. (5), we did not find strong or consistent associations between baldness pattern and common risk factors for coronary heart disease, except for age (table 2). Because participants in the study of Lesko et al. were younger, we also show the distribution of these risk factors in participants younger than age 60 (table 2). The results were similar.

TABLE 2. Distribution (column percent) of several risk factors for coronary heart disease among myocardial infarction-free participants, according to their baldness pattern, the Atherosclerosis Risk in Communities Study, 1987–1998

Risk factor	Hair loss pattern			Hair loss pattern among participants aged ≤60 years		
	None ($n = 1,421$)	Frontal ($n = 550$)	Vertex ($n = 2,300$)	None ($n = 639$)	Frontal ($n = 219$)	Vertex ($n = 779$)
Age (years)						
≤60	45	40	34	N/A*	N/A	N/A
>60	55	60	66	N/A	N/A	N/A
Family history of MI*	36	34	38	34	36	37
Hypercholesterolemia drug treated	12	12	12	11	10	11
Hypertension drug treated	29	32	33	24	24	30
Diabetes	16	16	16	16	15	16
Cigarette use, current	16	18	15	19	24	19
Body mass index (kg/m^2)						
<25	22	26	21	20	23	17
25–28	40	39	38	39	42	39
>28	38	36	40	41	35	44

* N/A, not applicable; MI, myocardial infarction.

Table 3 corresponds to table 5 in the article of Lesko et al. (5). In ARIC data, any association of baldness pattern with prevalent MI was weak, was not limited to vertex baldness, and did not show a monotonic dose-response relation with the extent of vertex baldness. All of these findings stand in sharp contrast to those of Lesko et al. Because the baldness pattern was only weakly associated with risk factors for coronary heart disease (table 2), conditional associations between baldness pattern and prevalent MI (from multivariable regression models) did not differ much from marginal, or age-adjusted, associations.

Lesko et al. found consistent and strong associations between incident MI and the extent of vertex baldness (severe vertex vs. none) across strata of several risk factors for coronary heart disease: Almost all of the odds ratios were greater than 2.0, and several were greater than 3.0 (table 7 in their article) (5). Our results stand in sharp contrast, again (table 4). Most of the estimates did not exceed 1.3 after conditioning on age. Among younger men (i.e., those aged ≤60 years), our estimated odds ratio for severe vertex baldness versus ''none'' was 1.52. The best matching estimate from their case-control study was 2.8—almost twice as large.

Table 5 shows the relation of baldness pattern to carotid IMT among 2,248 men, free of clinical cardiovascular disease, for whom ultrasound data were available. The

TABLE 3. Relation of baldness pattern to myocardial infarction status, the Atherosclerosis Risk in Communities Study, 1987–1998

Baldness pattern	Myocardial infarction ($n = 767$)	No myocardial infarction ($n = 4,289$)	Crude odds ratio	Age adjusted		Multivariable adjusted	
				Odds ratio*	95% confidence interval	Odds ratio†	95% confidence interval
None	215	1,421	Referent	Referent		Referent	
Frontal	112	550	1.35	1.26	0.98, 1.62	1.28	0.97, 1.68
Mild vertex	155	887	1.15	1.08	0.87, 1.36	1.02	0.80, 1.30
Moderate vertex	103	477	1.43	1.31	1.01, 1.70	1.40	1.05, 1.86
Severe vertex	180	936	1.27	1.14	0.91, 1.41	1.18	0.93, 1.49
Unknown	2	18					

* By adding age (continuous) to a logistic regression model.

† By adding the following covariates to a logistic regression model: age (continuous), smoking status (indicator variables), body mass index (continuous), race-center (indicator variables), use of a cholesterol-lowering medication, use of an antihypertensive medication, high density lipoprotein cholesterol (continuous), diabetes status, educational level (indicator variables), and family history of myocardial infarction.

TABLE 4. Relation of baldness pattern (severe vertex vs. none) to myocardial infarction status, in strata of selected risk factors for coronary heart disease, the Atherosclerosis Risk in Communities Study, 1987–1998

Factor and extent of baldness	Myocardial infarction (no.)	No myocardial infarction (no.)	Crude odds ratio	Age adjusted	
				Odds ratio	95% confidence interval
Age					
≤60 years					
None	71	639			
Severe vertex	49	280	1.58	1.52	1.03, 2.25
>60 years					
None	144	782			
Severe vertex	131	656	1.08	1.03	0.80, 1.34
Parental history of myocardial infarction					
No					
None	110	915			
Severe vertex	97	565	1.43	1.31	0.97, 1.76
Yes					
None	105	506			
Severe vertex	83	371	1.08	0.99	0.72, 1.36
Cholesterol medication					
No					
None	140	1,240			
Severe vertex	120	818	1.30	1.20	0.92, 1.56
Yes					
None	73	176			
Severe vertex	60	116	1.25	1.13	0.74, 1.72
Hypertension medication					
No					
None	97	998			
Severe vertex	78	614	1.31	1.21	0.88, 1.66
Yes					
None	111	414			
Severe vertex	100	318	1.17	1.11	0.81, 1.51

Table continues

estimated differences between mean IMT in men with various types of baldness and those with no baldness were all close to zero. For comparison, in these models the estimated mean difference between current smokers and never smokers was 0.09 mm (data not shown).

DISCUSSION

The ARIC data did not corroborate the hypothesis that male pattern baldness or the severity of vertex baldness is a surrogate measure of a strong risk factor for MI, such as androgenic activity. Moreover, there was no evidence of a meaningful association between baldness pattern and carotid wall thickness, a measure of the systemic burden of atherosclerosis (13, 14). If baldness as measured in the ARIC Study represents mainly androgenetic alopecia, androgenic stimulation may not be an important risk factor for myocardial infarction or atherosclerosis in men.

Several explanations could account for our failure to replicate the findings of Lesko et al. (5). First, it is possible that their estimates and ours are both unbiased and happen to disagree simply because two unbiased *estimates* of the same parameter could differ (the so-called ''play of chance''). Second, the estimates from one of the studies might have originated in biased estimators for reasons such as confounding, selection bias, or information bias. Of these, selection and information bias are more likely to operate in

TABLE 4. Continued

Factor and extent of baldness	Myocardial infarction (no.)	No myocardial infarction (no.)	Crude odds ratio	Age adjusted	
				Odds ratio	95% confidence interval
Diabetes					
No					
None	142	1,177			
Severe vertex	126	801	1.30	1.19	0.92, 1.54
Yes					
None	70	221			
Severe vertex	54	130	1.31	1.21	0.79, 1.84
Smoking					
Never					
None	41	392			
Severe vertex	33	278	1.13	1.07	0.66, 1.74
Former					
None	135	785			
Severe vertex	119	520	1.33	1.20	0.91, 1.58
Current					
None	35	229			
Severe vertex	28	132	1.39	1.30	0.75, 2.24
Body mass index					
<25 kg/m^2					
None	34	312			
Severe vertex	38	183	1.91	1.72	1.04, 2.84
25–28 kg/m^2					
None	81	567			
Severe vertex	67	360	1.30	1.20	0.84, 1.70
>28 kg/m^2					
None	99	540			
Severe vertex	75	392	1.04	0.96	0.69, 1.33
Race					
Black					
None	49	304			
Severe vertex	27	152	1.10	0.99	0.59, 1.65
White					
None	166	1,115			
Severe vertex	151	783	1.30	1.19	0.94, 1.52

a case-control sample where controls are a subset of the study base and where knowledge of case-control status may influence one's classification of baldness pattern. In the cross-sectional sample of the ARIC Study, there was no sampling of controls, and the study technicians were unlikely to know the man's prevalent MI status when they classified his baldness pattern. On the other hand, if the underlying risk factor affects survival as well, analysis of a cross-sectional sample can be open to prevalence-incidence bias. Possible confounders did not play an important role in either study. Both studies have adjusted for race, and the ARIC Study results were similar for Blacks and Whites (table 4).

A third explanation is linked to the idea of effect modification. A different distribution of (unknown) effect modifiers in two samples can generate different sizes of the effect measure of interest (24, 25), although it is difficult to imagine how such differences could have accounted for consistent disagreement in so many subsets of the two samples (table 4 in our article as compared with table 7 in the article of Lesko et al.). Finally, despite our attempt to replicate the analysis of Lesko et al., an epidemiologic study is never an

TABLE 5. Relation of baldness pattern to carotid intimal-medial thickness (mean, mean difference, and 95% confidence interval) in participants free of clinical cardiovascular disease, the Atherosclerosis Risk in Communities Study, 1987–1998

Baldness pattern	Mean intimal-medial thickness (mm)	Mean difference (mm)	Adjusted mean difference* (mm)	95% confidence interval
None ($n = 740$)	0.88	Referent	Referent	
Any baldness ($n = 1,508$)	0.89	0.01	0.00	−0.02, 0.02
Frontal ($n = 295$)	0.87	−0.01	−0.02	−0.05, 0.01
Vertex ($N = 1,213$)	0.90	0.02	0.00	−0.02, 0.02
Mild vertex ($n = 465$)	0.90	0.02	0.01	−0.02, 0.03
Moderate vertex ($n = 248$)	0.89	0.01	−0.01	−0.04, 0.03
Severe vertex ($n = 500$)	0.90	0.02	0.00	−0.03, 0.03

* Derived from weighted linear regression of the mean carotid intimal-medial thickness on baldness variables and the following covariates: age (continuous), smoking status (indicator variables), body mass index (continuous), race-center (indicator variables), use of a cholesterol-lowering medication, use of an antihypertensive medication, high density lipoprotein cholesterol (continuous), diabetes status, educational level (indicator variables), and family history of myocardial infarction.

exact replication of a previous study. For instance, ARIC Study participants were older than the participants in the study of Lesko et al., and the ARIC sample contained prevalent MI cases rather than incident MI cases. Of course, the definition of MI was not identical. These differences, however, did not jeopardize our ability to replicate well-known associations between prevalent MI and numerous risk factors for coronary heart disease—by the very same regression models (data not shown).

Several other studies have followed the work of Lesko et al. Using a retrospective cohort design, Lotufo et al. (10) reported risk ratios of 1.2–1.4 for various kinds and severity of baldness, but the location of baldness did not seem to matter, and there was no clear evidence for a graded association with the extent of vertex baldness. For example, the estimated risk ratios for nonfatal MI (from multivariable models) were 1.26 (frontal baldness), 1.46 (mild vertex baldness), 1.32 (moderate vertex baldness), and 1.30 (severe vertex baldness). Similarly, Schnohr et al. (7) reported a risk ratio of 1.2 for vertex baldness and incident MI from a prospective cohort study but, unexpectedly, a stronger association between frontal baldness and incident MI (a risk ratio of 1.6). Herrera et al. (6), who analyzed data from the Framingham cohort, also failed to replicate the findings of Lesko et al. but reported strong associations between various cardiovascular endpoints and the rate at which baldness progressed. On the basis of cohort data from the first National Health and Nutrition Examination Survey, Ford et al. (8) reported no association between severe baldness and incident ischemic heart disease in their sample (the whole sample), with a risk ratio of 1.72 among men younger than 55 years (a subset of their sample) and the lack of a monotonic dose-response relation with the extent of baldness. (The location of baldness was not recorded). The only study that may be regarded as a successful replication of the findings of Lesko et al. was another hospital-based, case-control study of incident, nonfatal MI: Mirić et al. (9) reported an odds ratio of 1.9 for vertex baldness and an odds ratio of 0.9 for frontal baldness. The extent of vertex baldness was not recorded.

The baldness pattern itself, even if found to predict MI status in some samples, is probably not a risk factor for coronary heart disease. According to the prevailing thought, baldness is one of the consequences of androgenic stimulation and therefore serves as a surrogate measure (a marker) for that unmeasured cause of atherosclerosis. Alternatively, the upstream causal variable, if any, may be a genetic trait that is a common cause of baldness and atherosclerosis. Unless the baldness pattern and the MI status are posited to be linked by a unidirectional causal path, any association between the two variables—whether crude or adjusted—must be confounded (by the true causal variable) (26).

In summary, our results and those of several other research groups suggest that male pattern baldness is not a surrogate measure of an important risk factor for myocardial infarction or atherosclerosis, such as androgenic activity.

ACKNOWLEDGMENTS

The Atherosclerosis Risk in Communities Study is carried out as a collaborative study supported by National Heart, Lung, and Blood Institute contracts N01-HC-55015, N01-HC-55016, N01-HC-55018, N01-HC-55019, N01-HC-55020, N01-HC-55021, and N01-HC-55022.

The authors thank the staff of the ARIC Study for their important contributions.

Conflict of interest: none declared.

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