Androgenic alopecia, premature graying, and hair thinning as independent predictors of coronary artery disease in young Asian males

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Introduction We herewith aimed to explore the association of premature graying, androgenic alopecia (AGA), and hair thinning with coronary artery disease (CAD) in young (≤ 40 years) male individuals from Western India.

Patients and methods In this prospective, case-control study, 1380 male individuals from a super speciality cardiac care center were enrolled, of which 468 were established cases of CAD and 912 were age-matched healthy male individuals not having history of any major illness including CAD. Details of demographics, cardiovascular risk factors, and cutaneous markers were collected for both the groups.

Results Prevalence of hypertension (30.3 vs. 13.6%), obesity (28.8 vs. 12.2%), hair thinning (36.3 vs. 14.6%), premature graying (49.6 vs. 29.9%), AGA (49.1 vs. 27.4%), and lipid abnormalities (total cholesterol – 16.7 vs. 8.8%; low-density lipoprotein – 7.3 vs. 2.2%; and high-density lipoprotein – 92.5 vs. 88.7%) were higher in cases as compared with control. Multiple logistic regression analysis showed that AGA [5.619, 95% confidence interval (CI): 4.025–7.845, $P < 0.0001$] is the strongest predictor of CAD among young Asian male individuals, closely followed by premature graying (5.267, 95% CI: 3.716–7.466, $P < 0.0001$), obesity (4.133, 95% CI: 2.839–6.018, $P < 0.0001$), and hair

thinning (3.36, 95% CI: 2.452–4.621, $P < 0.0001$). SYNTAX score, left ventricle ejection fraction, and degree of disease severity were also found to be independent associates of premature graying and AGA.

Conclusion Our findings support the hypothesis that cutaneous markers are independently associated with underlying CAD irrespective of other classical cardiovascular risk factors. This, in combination with classical markers, could be effectively used for early identification and risk stratification of young patients with occult or established CAD. *Cardiovasc Endocrinol* 6:152–158 Copyright © 2017 Wolters Kluwer Health, Inc. All rights reserved.

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Introduction

Atherosclerosis is a chronic inflammatory disease, triggered in response to endothelial injury occurring to the vessel wall. Although endothelium has regenerative characteristics, in presence of various cardiovascular risk factors (CVRFs), this natural healing process is hampered [1]. It is known that conjoint effects of CVRFs lead to the development of premature atherosclerosis, and these factors may also contribute to premature graying and alopecia by interacting with follicular epithelium and resident stem cells similar to their effects on endothelium and circulating progenitor stem cells [2,3].

Biological age is an established risk factor of coronary artery disease (CAD) and is often associated with various dermatological changes. Earlier published reports have indicated that atherosclerosis and cutaneous markers such as alopecia and premature graying have common

mechanism of actions such as impaired DNA repair, oxidative stress, inflammation, hormonal changes, and senescence of functional cells. Similar to vascular system, mammalian hair follicle system possesses distinct regenerative characteristics, where involved stem cells produce melanocyte, a coloring pigment at the base of follicle. Hence, premature graying of hair indicates initiation of the early degenerative changes in the system and is reported to show a relationship with CAD [4]. Male pattern baldness, often known as androgenic alopecia (AGA), is also a proposed cutaneous marker of CAD risk by many [5]. AGA is a typical pattern of gradual, progressive hair loss, beginning along the frontal hairline and on the vertex. For association of AGA with CAD, several mechanisms such as increased peripheral sensitivity to androgens, metabolic syndrome, insulin resistance, chronic inflammation, dyslipidemia, and hypertension have been proposed [6].

We herewith aim to study the overall prevalence and association of premature graying, AGA, and hair thinning with CAD in young Asian male patients and their predictability for CAD.

Patients and methods

In this prospective case-control study, we recruited 1380 individuals, of which 468 were patients with CAD (<40 years of age) who were admitted for various cardiac surgeries or procedures between the months of January 2014 and January 2016 in a super specialty hospital of Ahmedabad, Gujarat, India. The diagnosis of CAD was performed using angiography, and stenosis of at least 70% diameter was considered as significant. Age-matched young healthy male individuals ($n=912$), not having any major history of illness with normal coronary angiograms, were included as healthy control. Voluntary participation and written informed consent was obtained for all the participants. The study was reviewed and approved by the Institutional Ethics Committee (UNMICRC/CARDIO/2015/72). Basic demographic information, details of comorbidities, past medical history, individual income, smoking habit, and BMI were collected for all participants.

Diagnosis of androgenic alopecia, premature graying, and hair thinning

The diagnosis of the cutaneous markers in the study population was based on detailed history and clinical examinations. Hamilton-Norwood scale was used as a tool for grading of AGA (I–VII) in young male individuals [7]. Patients were classified into various grades of premature graying as follows: 1 = pure black hair, 2 = black more than white, 3 = black equals white, 4 = white more than black, and 5 = pure white. The grades of AGA and premature graying for each patient were determined by two independent observers. Hair thinning was noted based on self-perceived thinning of hair reported by an individual in response to a questionnaire [8].

Cardiovascular risk factor assessment

The patients were evaluated for age, demographical properties, and the CVRFs. Hypertension was defined as the active use of antihypertensive drugs or documentation of blood pressure more than 140/90 mmHg. Diabetes mellitus was defined as fasting plasma glucose levels of more than 126 mg/dl or glucose level more than 200 mg/dl at any measurement or active use of antidiabetic treatment. Patients who were using tobacco products on admission to our hospital and those who quit smoking within the past year were considered as smokers. The family history for CAD was defined as a history of CAD or sudden death in a first-degree relative before the age of 55 years for men and 65 years for women. C-reactive protein level and routine biochemistry including glucose and lipid levels were measured. Serum C-reactive protein was analyzed using a nephelometric technique.

Statistical analysis

The statistical calculations were performed using SPSS software, version 20.0 (SPSS Inc., Chicago, Illinois, USA). Quantitative data were expressed as mean $\pm$ SD, whereas qualitative data were expressed as percentage. Univariate analysis of the continuous data was performed using Student's t -test (paired or unpaired whichever is applicable), whereas χ^2 -test was used for the categorical data. Logistic regression was used for estimation of independent risk factors for the factors with significant P value (<0.05) on univariate analysis.

Results

The demographic and risk factor profile of CAD cases and control was compared and is presented in Table 1. Cases were more hypertensive (30.3 vs. 13.6%), obese (26.7 vs. 12%), and had a greater prevalence of lipid abnormalities (higher total cholesterol – 16.7 vs. 8.8%, $P<0.0001$; higher low-density lipoprotein – 7.3 vs. 2.2%, $P<0.0001$; and low high-density lipoprotein – 92.5 vs. 88.7%, $P=0.0212$) as compared with controls. Similarly, overall prevalence of hair thinning, premature graying, and AGA in CAD cases were 36.3, 49 and 50%, respectively, which was significantly higher as compared with control population (hair thinning – 14.6%, premature graying – 25.8%, and AGA – 27.4%).

Table 1

Variables	Cases ($n=468$)	Control ($n=912$)	Significance (P value)
Diabetes	58 (12.4)	86 (9.4)	0.107
Hypertension	142 (30.3)	124 (13.6)	<0.0001
Premature CAD	41 (8.8)	50 (5.5)	0.0272
BMI			
18.5–21.9	175 (37.4)	130 (14.3)	<0.0001
22–24.9	158 (33.8)	671 (73.6)	<0.0001
25–29.9	125 (26.7)	109 (12.0)	<0.0001
> 30	10 (2.1)	2 (0.2)	0.0009
Dyslipidemia	63 (13.5)	115 (12.6)	0.7172
Hair thinning	170 (36.3)	133 (14.6)	<0.0001
Premature graying			
No premature graying	232 (49.6)	639 (70.1)	<0.0001
1–20%	167 (35.7)	174 (19.1)	<0.0001
20–40%	65 (13.9)	98 (10.7)	0.1042
40–60%	4 (0.9)	1 (0.1)	0.0877
Androgenic alopecia			
No alopecia	238 (50.9)	662 (72.6)	<0.0001
Mild	165 (35.3)	159 (17.4)	<0.0001
Moderate	61 (13.0)	90 (9.9)	0.0906
Severe	4 (0.9)	1 (0.1)	0.0877
Lipoprotein A	82 (17.5)	122 (13.4)	0.0485
Lipoprotein B	71 (15.2)	156 (17.1)	0.4004
High total cholesterol	78 (16.7)	80 (8.8)	<0.0001
High triglycerides	23 (4.9)	53 (5.8)	0.5709
High low-density lipoprotein cholesterol	34 (7.3)	20 (2.2)	<0.0001
Low high-density lipoprotein cholesterol	844 (92.5)	415 (88.7)	0.0212
High total lipids	68 (14.5)	99 (10.9)	0.0582

CAD, coronary artery disease.

Table 2 Association of cardiovascular risk factors with hair thinning

	Unstandardized coefficients		Standardized coefficients		Significance	95.0% Confidence interval for <i>B</i>	
	<i>B</i>	SE	β	<i>t</i>		Lower bound	Upper bound
Constant	−0.078	0.097	–	−0.810	0.418	−0.268	0.111
Diabetes	0.130	0.037	−0.096	3.552	0.000	0.202	0.058
Hypertension	0.011	0.029	0.011	0.385	0.700	−0.046	0.068
Family history of premature CAD	0.194	0.045	0.117	4.264	0.000	0.105	0.283
Central obesity	0.001	0.034	0.001	0.021	0.984	−0.067	0.068
Obesity	0.012	0.004	0.084	2.840	0.005	0.004	0.020
Dyslipidemia	0.038	0.037	0.031	1.031	0.303	0.111	0.035
Tobacco	0.003	0.024	0.003	0.116	0.907	−0.045	0.050
Smoking	0.134	0.023	0.160	5.802	0.000	0.089	0.179
Lipoprotein A	0.062	0.032	0.054	1.974	0.049	0.000	0.124
Lipoprotein B	−0.029	0.031	−0.026	−0.928	0.353	−0.091	0.033
Total cholesterol	0.016	0.035	0.012	0.461	0.645	0.085	0.053
Triglyceride	0.044	0.049	0.024	0.898	0.369	−0.052	0.139
Low-density lipoprotein cholesterol	0.141	0.058	0.065	2.420	0.016	0.027	0.255
High-density lipoprotein cholesterol	0.071	0.040	0.047	1.750	0.080	−0.009	0.150
Total lipids	0.014	0.035	0.011	0.394	0.694	−0.056	0.083
Homocysteine	0.091	0.036	0.067	2.486	0.013	0.019	0.162
<i>R</i> ²				0.072			

CAD, coronary artery disease.

Table 3 Association of cardiovascular risk factors with premature graying

	Unstandardized coefficients		Standardized coefficients		Significance	95.0% Confidence interval for <i>B</i>	
	<i>B</i>	SE	β	<i>t</i>		Lower bound	Upper bound
Constant	0.189	0.160	–	1.179	0.238	−0.125	0.502
Diabetes	0.072	0.060	0.031	1.188	0.235	−0.047	0.190
Hypertension	0.143	0.048	0.079	2.972	0.003	0.049	0.238
Family history of premature CAD	−0.053	0.075	−0.019	−0.705	0.481	−0.200	0.095
Central obesity	0.021	0.057	0.011	0.362	0.717	−0.091	0.132
Obesity	0.009	0.007	0.039	1.362	0.174	−0.004	0.023
Dyslipidemia	0.006	0.062	0.003	0.103	0.918	−0.114	0.127
Tobacco	0.177	0.040	0.116	4.426	0.000	0.099	0.256
Smoking	0.172	0.038	0.120	4.496	0.000	0.097	0.246
Lipoprotein A	0.336	0.052	0.168	6.437	0.000	0.234	0.439
Lipoprotein B	−0.237	0.052	−0.121	−4.550	0.000	−0.339	−0.135
Total cholesterol	−0.018	0.058	−0.008	−0.315	0.753	−0.132	0.095
Triglyceride	0.387	0.080	0.124	4.818	0.000	0.230	0.545
Low-density lipoprotein cholesterol	0.455	0.096	0.122	4.733	0.000	0.266	0.644
High-density lipoprotein cholesterol	−0.276	0.067	−0.107	−4.121	0.000	−0.407	−0.144
Total lipids	−0.091	0.059	−0.041	−1.554	0.120	−0.206	0.024
Homocysteine	0.046	0.060	0.020	0.769	0.442	−0.072	0.164
<i>R</i> ²				0.14			

CAD, coronary artery disease.

The relationship of hair thinning, premature graying, and AGA with various risk factors is assessed and tabulated in Tables 2–4, respectively. Multiple linear regression analysis showed that diabetes, hypertension, habits of smoking and tobacco consumption, and various lipid abnormalities are independently associated with these cutaneous markers. SYNTAX score – an index of CAD complexity – was also found to be an independent associate of premature graying ($P < 0.0001$) and AGA ($P < 0.0001$) but not of hair thinning ($P = 0.737$) (Table 5). Similarly, LVEF as assessed by Simpson's method on echocardiography and degree of severity were also associated with these cutaneous markers. Adjusted odds ratio was calculated to investigate the independent risk of each factor for development of CAD (Table 6). AGA had the highest odds [5.619, 95% confidence

interval (CI): 4.025–7.845, $P < 0.0001$], which was closely followed by premature graying (5.267, 95% CI: 3.716–7.466, $P < 0.0001$), whereas hair thinning showed odds of 3.36 (95% CI: 2.452–4.621, $P < 0.0001$). Risks exerted by various factor according to age are presented in Table 7. Premature graying and AGA had higher odds in young adult patients (30–40 years) as compared with younger population (20–30 years), whereas association of hair thinning was not influenced by age.

Discussion

The current study results indicate that presence of cutaneous markers such as hair thinning, premature graying, and AGA are potential prognostic markers that could be effectively used in designing primary preventive strategies for effective management of CAD in

Table 4 Association of cardiovascular risk factors with androgenic alopecia

	Unstandardized coefficients		Standardized Coefficients		Significance	95.0% Confidence interval for <i>B</i>	
	<i>B</i>	SE	β	<i>t</i>		Lower bound	Upper bound
Constant	0.442	0.165	–	2.676	0.008	0.118	0.766
Diabetes	–0.119	0.062	–0.052	–1.900	0.058	–0.241	0.004
Hypertension	–0.005	0.050	–0.003	–0.109	0.913	–0.103	0.092
Family history of premature CAD	–0.044	0.078	–0.016	–0.572	0.568	–0.197	0.108
Central obesity	–0.081	0.059	–0.042	–1.371	0.171	–0.196	0.035
Obesity	–0.005	0.007	–0.020	–0.673	0.501	–0.019	0.009
Dyslipidemia	–0.057	0.064	–0.027	–0.899	0.369	–0.182	0.068
Tobacco	0.090	0.041	0.060	2.181	0.029	0.009	0.171
Smoking	0.133	0.039	0.095	3.379	0.001	0.056	0.211
Lipoprotein A	–0.021	0.054	–0.011	–0.391	0.696	–0.127	0.085
Lipoprotein B	–0.225	0.054	–0.117	–4.186	0.000	–0.330	–0.120
Total cholesterol	0.152	0.060	0.069	2.533	0.011	0.034	0.269
Triglyceride	–0.130	0.083	–0.042	–1.561	0.119	–0.293	0.033
Low-density lipoprotein cholesterol	–0.159	0.099	–0.043	–1.598	0.110	–0.354	0.036
High-density lipoprotein cholesterol	–0.033	0.069	–0.013	–0.484	0.629	–0.169	0.102
Total lipids	0.101	0.061	0.047	1.674	0.094	–0.017	0.220
Homocysteine	0.185	0.062	0.082	2.984	0.003	0.064	0.307
<i>R</i> ²				0.044			

CAD, coronary artery disease.

Table 5 Association of cutaneous markers with disease severity parameters

	Unstandardized coefficients		Standardized coefficients		Significance	95.0% Confidence interval for <i>B</i>	
	<i>B</i>	SE	β	<i>t</i>		Lower bound	Upper bound
Hair thinning							
Constant	0.387	0.038	–	10.090	0.000	0.312	0.462
LVEF	0.120	0.022	0.163	5.508	0.000	0.078	0.163
Severity of CAD (SVD, DVD, and TVD)	0.175	0.028	0.353	6.300	0.000	0.121	0.230
SNTAX score	0.007	0.019	0.009	0.336	0.737	–0.032	0.045
Premature graying							
Constant	0.285	0.046	–	6.232	0.000	0.195	0.375
LVEF	0.046	0.026	0.054	1.780	0.075	–0.005	0.098
Severity of CAD (SVD, DVD, and TVD)	0.090	0.033	0.155	2.701	0.007	0.025	0.155
syntax score	0.243	0.033	0.205	7.313	0.000	0.178	0.308
Androgenic alopecia							
(Constant)	0.414	0.046	–	9.084	0.000	0.324	0.503
LVEF	0.081	0.026	0.095	3.115	0.002	0.030	0.132
Severity of CAD (SVD, DVD, and TVD)	0.102	0.033	0.179	3.082	0.002	0.037	0.167
SYNTAX score	0.121	0.034	0.101	3.617	0.000	0.056	0.187

CAD, coronary artery disease; DVD, double-vessel disease; LVEF, left ventricle ejection fraction; SVD, single-vessel disease; TVD, triple-vessel disease.

Western Indians. Though these markers are nonmodifiable in nature, it could contribute significantly in risk stratification of the population in whom controlling of modifiable risk factors and periodic screening for CAD may be beneficial.

Several epidemiological evidences have indicated a positive association between CAD and AGA, where androgen levels are suggested to play a pivotal role. It is also known that men with baldness have higher levels of serum androgens, androgen receptors in scalp, and total and free testosterone [9–13]. After adjusting the influence of other confounders, current study results postulate that the patients with AGA and premature graying have approximately five times higher risk of developing CAD at a younger age. This is in concordance with one of our earlier study where we found a significant association between vertex baldness (grades IV–VII) and CAD in

young (<45 years) male patients with CAD, with an overall AGA incidence of 37.73% [5]. It is documented that 50% of white men have some degree of baldness before the age of 50 years, and in this ethnic group, thinning of hair starts as early as age of 12 years [14]. Similar to AGA, premature graying is also an indicator of accelerated ageing process which is associated with CVRFs through various interlinked pathways. Loss of hair pigmentation and atherosclerosis are known to share similar molecular and cellular mechanisms, and scalp ageing is known to predispose an individual to premature CAD. Pomerantz had reported that premature graying is more common in patients with CAD younger than 35 years and is subjected to various intrinsic and extrinsic factors such as smoking, ultraviolet rays, pollution, and lifestyle factors [15,16]. In contrast to our findings, Sari *et al.* [17] reported lack of association between male

Table 6 Overall odds (adjusted for other risk factors)

	B	SE	Wald	Significance	Exp(B)	95% CI for EXP(B)	
						Lower	Upper
Age	0.050	0.017	8.291	0.004	1.051	1.016	1.088
Diabetes	0.202	0.221	0.840	0.359	1.224	0.794	1.888
Hypertension	1.086	0.169	41.391	0.000	2.964	2.129	4.126
Family history of premature CAD	-0.041	0.265	0.024	0.877	0.960	0.571	1.613
Central obesity	-0.729	0.238	9.374	0.002	0.482	0.302	0.769
Obesity	1.484	0.195	57.825	0.000	4.412	3.009	6.467
Dyslipidemia	-0.458	0.239	3.685	0.055	0.632	0.396	1.010
Tobacco	0.070	0.149	0.217	0.641	1.072	0.800	1.436
Smoking	-0.032	0.145	0.049	0.825	0.968	0.729	1.286
Hair thinning	1.214	0.162	56.357	0.000	3.366	2.452	4.621
Premature graying	1.662	0.178	87.155	0.000	5.267	3.716	7.466
Alopecia	1.726	0.170	102.857	0.000	5.619	4.025	7.845
Lipoprotein A	-0.185	0.195	0.895	0.344	0.831	0.567	1.219
Lipoprotein B	0.294	0.209	1.972	0.160	1.341	0.890	2.021
Total cholesterol	0.556	0.205	7.315	0.007	1.743	1.165	2.607
Triglyceride	-0.597	0.308	3.765	0.052	0.551	0.301	1.006
Low-density lipoprotein cholesterol	0.784	0.345	5.158	0.023	2.191	1.113	4.311
High-density lipoprotein cholesterol	0.755	0.243	9.685	0.002	2.128	1.323	3.425
Total lipids	0.725	0.218	11.057	0.001	2.065	1.347	3.166
Homocysteine	1.013	0.216	22.086	0.000	2.754	1.805	4.203
Constant	-4.808	0.648	55.084	0.000	0.008	-	-

CAD, coronary artery disease; CI, confidence interval.

pattern of baldness and severity of CAD; however, the study had few limitations: (i) smaller sample size and (ii) cross-sectional design of the study. The cumulative evidence from six different (three cohort and three case-control) studies was examined and presented as a meta-analysis by Yamada and colleagues. They found that vertex baldness, but not frontal baldness, is associated with an increased risk of coronary heart disease. They found that severity of the disease is directly related to the severity of vertex baldness [18].

The interplay between atherosclerosis, classical CVRFs, and cutaneous markers has been reported earlier, which is in concordance with our results where diabetes, hypertension, smoking, obesity, and various dyslipidemias were found to be associated with these markers. Sharma *et al.* [19] and El-Esawy and Abd El-Rahman [20] showed that severity of systolic blood pressure is well correlated with severity of alopecia. Park and colleagues evaluated this association in Asians where they aimed to investigate the relationship between Korean men with AGA and various CVRFs, considering life habits, type of hair loss, and sex. They found a direct relationship between AGA severity and increased incidences of CVRFs, namely, hypertension, diabetes, and smoking [21]. Similarly, obesity was also shown to be a causative factor of AGA by Yang *et al.* [22]. Higher circulating lipid levels, namely, total cholesterol, triglycerides, and low-density lipoproteins, are known to be risk factors for atherosclerotic plaque formation, which were found to increase with increased severity of AGA [23]. Apart from this, it is also documented that smokers have greater severity of AGA as compared with nonsmokers, which could be explained by the smoking-induced deleterious effects on microvasculature of dermal hair papilla and

redox and proteases imbalance in the hair follicular system. The toxicants present in the smoke may directly damage the follicle DNA also [24,25]. Herewith we report that 50% of the male CAD population had premature graying of hair and AGA, which significantly correlated with high blood pressure, BMI, smoking, and lipid levels. Apart from these modifiable risk factors, family history also showed a strong association ($P < 0.0001$) with hair thinning indicating a role of genetics in the entire phenomena. A variety of genetic and environmental factors are said to play a role in causing AGA. Androgen receptor gene, which is X-linked recessive, has been mentioned to be the cardinal prerequisite for balding, but other genes are also involved. Genetic sensitivity of hair follicles to dihydrotestosterone causes them to shrink when exposed to it [26]. 5- α -reductase is responsible for converting free testosterone (a major circulating androgen in men) into dihydrotestosterone [27]. Hair growth inhibitory factor- β , released from androgen-stimulated fibroblasts of the follicular dermal papilla, may cause hair growth inhibition, hair follicle miniaturization, and short lifespan, thereby preventing normal hair production [28]. So, our findings suggest that cutaneous markers are common indicators of ageing, which may play a role as a biological age indicator. Similar to various biological age calculators such as Framingham vascular age calculator, a potentially useful scale for biological age having clinical usage may be introduced.

Conclusion

We herewith conclude that screening of young male patients with CAD for the presence of AGA, premature graying of hair, and thinning of hair facilitates

Table 7 Odds according to age (adjusted for other risk factors)

						95% CI for Exp(B)	
Age coding	B	SE	Wald	Significance	Exp(B)	Lower	Upper
20–30 years							
Age	−0.042	0.090	0.223	0.637	0.959	0.804	1.143
Diabetes	−0.056	0.549	0.010	0.919	0.946	0.322	2.774
Hypertension	0.316	0.542	0.340	0.560	1.371	0.475	3.964
Family history of premature CAD	1.032	0.891	1.342	0.247	2.806	0.490	16.082
Central obesity	−1.376	0.720	3.653	0.056	0.253	0.062	1.036
Obesity	1.488	0.501	8.803	0.003	4.426	1.657	11.825
Dyslipidemia	−1.756	0.787	4.975	0.026	0.173	0.037	0.808
Tobacco	0.874	0.471	3.444	0.063	2.396	0.952	6.032
Smoking	0.346	0.446	0.603	0.437	1.414	0.590	3.387
Hair thinning	1.322	0.460	8.245	0.004	3.751	1.521	9.249
Premature graying	0.817	0.525	2.419	0.012	2.264	0.809	6.337
Alopecia	1.656	0.501	10.931	0.001	5.237	1.962	13.975
Lipoprotein A	−0.169	0.528	0.103	0.748	0.844	0.300	2.376
Lipoprotein B	0.660	0.554	1.420	0.233	1.935	0.653	5.732
Total cholesterol	0.958	0.628	2.323	0.127	2.605	0.761	8.926
Triglyceride	−0.490	0.826	0.352	0.553	0.613	0.121	3.091
Low-density lipoprotein cholesterol	−0.591	1.328	0.198	0.657	0.554	0.041	7.485
High-density lipoprotein cholesterol	1.927	0.843	5.227	0.022	6.869	1.317	35.840
Total lipids	0.904	0.580	2.425	0.119	2.469	0.792	7.700
Homocysteine	1.139	0.600	3.606	0.058	3.122	0.964	10.112
Constant	−2.393	2.457	0.948	0.330	0.091	–	–
30–40 years							
Age	0.050	0.027	3.442	0.064	1.052	0.997	1.109
Diabetes	0.266	0.247	1.160	0.281	1.305	0.804	2.120
Hypertension	1.200	0.183	43.044	0.000	3.321	2.320	4.752
Family history of premature CAD	−0.146	0.285	0.262	0.609	0.864	0.495	1.510
Central obesity	−0.689	0.262	6.918	0.009	0.502	0.300	0.839
Obesity	1.462	0.218	44.964	0.000	4.315	2.815	6.617
Dyslipidemia	−0.277	0.261	1.124	0.289	0.758	0.455	1.265
Tobacco	0.007	0.161	0.002	0.966	1.007	0.734	1.381
Smoking	−0.097	0.157	0.382	0.537	0.908	0.667	1.234
Hair thinning	1.236	0.179	47.811	0.000	3.443	2.425	4.889
Premature graying	1.797	0.194	85.522	0.000	6.031	4.121	8.826
Alopecia	1.817	0.187	94.429	0.000	6.154	4.266	8.878
Lipoprotein A	−0.093	0.214	0.189	0.663	0.911	0.599	1.386
Lipoprotein B	0.228	0.231	0.975	0.323	1.256	0.799	1.976
Total cholesterol	0.495	0.223	4.947	0.026	1.641	1.061	2.539
Triglyceride	−0.662	0.342	3.735	0.053	0.516	0.264	1.009
Low-density lipoprotein cholesterol	0.855	0.366	5.465	0.019	2.352	1.148	4.820
High-density lipoprotein cholesterol	0.653	0.263	6.173	0.013	1.921	1.148	3.216
Total lipids	0.725	0.241	9.088	0.003	2.065	1.289	3.309
Homocysteine	1.000	0.234	18.278	0.000	2.720	1.719	4.302
Constant	−4.881	0.994	24.109	0.000	0.008	–	–

CAD, coronary artery disease; CI, confidence interval.

identification of a subset of individuals who are at an increased risk for future acute CAD and who require more rigorous follow-up and treatment. Intensive monitoring of these patients and strict advocacy of exercise and lifestyle modification are necessary to reduce the incidence of premature CAD.

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Conflicts of interest

There are no conflicts of interest.

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