REVIEW

Baldness and Coronary Artery Disease

The Dermatologic Point of View of a Controversial Issue

Alfredo Rebora, MD

Objective: Several articles, most of them written by nondermatologists, have stressed that bald men have a higher risk for coronary artery disease than men who are not bald. This study was performed to evaluate the validity of such conclusions from a dermatologic point of view.

Design: A review of the 24 articles in literature from 1954 to 1999 as provided by MEDLINE and a previous review.

Results: Five articles contained simple comments; 1 was a review of the previous literature; and 3 dealt only with the lipid profile. The remaining 15 articles dealt with coronary artery disease and baldness, and 9 of these concluded that there is a relationship between the 2 conditions, especially in younger subjects with severe early-onset androgenetic alopecia.

Conclusions: Baldness did not coincide with androgenetic alopecia in some of the articles examined, which makes it difficult to settle the issue. Subjects who develop baldness before their 30s may have a higher risk for coronary artery disease than other men, and they may be individuals with early-onset androgenetic alopecia who also present with particularly elevated dihydrotestosterone-testosterone ratios. The baldness theory should be included as a secondary hypothesis in large epidemiological studies of coronary artery disease. Such studies should include dermatologic expertise for accurate, cost-effective evaluation of baldness.

Arch Dermatol. 2001;137:943-947

From the Department of Dermatology, University of Genoa, Genoa, Italy. Dr Rebora is now with the Department of Endocrinologic and Metabolic Diseases, Section of Dermatology, University of Genoa.

In the last decade, several articles have indicated that bald men have a higher-than-normal risk for coronary artery disease (CAD) and that early-onset androgenetic alopecia (AGA), in particular, is somehow related to CAD. Such conclusions cannot be underestimated by dermatologists who treat with patients with AGA and who may prescribe systemic drugs. On the other hand, most of these articles have been written by nondermatologists, without confirmation by specialists in dermatology. Since AGA affects about 90% of the general population, epidemiological studies may rely only on disease severity. The diagnosis of AGA is only apparently easy, and other forms of nonscarring alopecias may be incorrectly included in studies conducted by nondermatologists. Therefore, I screened the literature to evaluate the validity of the conclusions from a dermatologic point of view.

RESULTS

Twenty-four articles met the criteria. Five articles[2-6] contained simple comments; 1 was a survey of the previous literature[1]; and 3 dealt only with the lipid profile.[7-9] The remaining 15 articles were therefore analyzed.

Gertler and White[10] studied men who had a myocardial infarction before 40 years of age. The subjects entered the study up to 10 years after the infarction occurred. Baldness was defined according to the Hamilton scale. Other risk factors for CAD were not studied. There was no difference between bald patients and controls. Only patients who survived myocardial infarction entered the study, and there was no information about those who died.

Buechner et al[11] compared 40 "heart patients" with 153 controls. No better definition of heart patients was provided. The subjects were defined as bald when they presented with extensive frontal and coronal hair loss. The number of persons in each group was not given. Only smoking was considered among CAD risk factors. Statistical analysis was done with the rank test. There was no statistical significance, possibly because of the small size of the sample.

Cotton et al[12] compared 91 men with myocardial infarction or angina with 98

MATERIALS AND METHODS

Medical literature published in any language since 1954 on baldness and CAD was identified through a MEDLINE search using the key words *coronary* and *baldness*. Additional references were identified from the reference lists of a published review.[1]

The articles reviewed involved studies of patients with baldness who were recruited to evaluate other risk factors for CAD, and observational studies (case-control and cohort studies) concerning the possible association of baldness with CAD. Each study was evaluated according to the following criteria: how it was conducted; presence of possible flaws; number of patients examined; accuracy of the diagnosis of baldness; measure of the severity of baldness; cardiac outcome of the patients, adjustment for other risk factors (eg, smoking, history of hypertension, diabetes, high cholesterol level, alcohol intake, and physical activity), if any; and statistical analysis to assess the significance of the association. Additional statistical tests (χ^2 test) were performed whenever necessary.

blood donors. The subjects were classified into 4 groups: no baldness, receding frontal hairline, a critical bald area, and total or subtotal hair loss. The cardiac group had higher scores for baldness, as well as for blood pressure and smoking habit. Nevertheless, multivariate analysis showed that baldness was highly significantly associated with CAD ($P \leq .001$).

Hamby et al[13] prospectively evaluated 710 men. Baldness was merely defined as frontoparietal hair loss. A significantly higher proportion of bald men had CAD than did nonbald men. Eighty-one percent of the bald men with CAD were bald before the clinical onset of CAD, and 65% were bald before 35 years of age.

Ben Halim et al[14] interviewed 48 men who were in the hospital for myocardial infarction and compared them with 48 men with benign conditions. Hypertension and diabetes were exclusion criteria. Baldness was classified according to the Hamilton scale. No statistical differences were found between the 2 groups.

Cooke[15] examined 478 hospitalized men in London, England. Baldness was defined as Hamilton class III or worse. Men with diabetes were excluded. Cooke concluded that there was little relationship between baldness and CAD. When Cooke's data were reanalyzed, however,[1] a possible stronger relationship was revealed. The 50- to 59-year-old group had an odds ratio (OR) of 2.77 (95% confidence interval [CI], 1.19-6.47). The data were insufficient to adjust baldness for other risk factors. My reanalysis of Cooke's data revealed that his bald subjects had smoked significantly more than his nonbald subjects (52% vs 35%; OR, 1.57; 95% CI, 1.05-2.35); therefore, smoking may account for the differences observed in CAD. Actually, smokers did not have CAD more often than nonsmokers (OR, 0.69; 95% CI, 0.45-1.07); however, only individuals who smoked 10 or more cigarettes daily were defined as smokers, a factor that could underscore the variable.

Persson and Johansson[16] studied 464 men for 22 years for CAD. Baldness was defined as "baldness or tonsure," and no statistical analysis was performed. Nevertheless, baldness was suggested to be "a new risk factor" for CAD. In their review, Herrera and Lynch[1] reanalyzed the data and suggested that the percentage of bald men with CAD was not significantly higher than that of nonbald men (25% vs 19%; $P = .16$).

Emidy et al[17] evaluated 1594 men who were 40 to 59 years old at entry for 25-year mortality from CAD. Baldness was not defined. Only 40- to 49-year-old bald men were found to be more susceptible to CAD. Other CAD risk factors were considered in the statistical analysis. The original data were not available to me.

The case-control study of Lesko et al[18] involved 665 men younger than 55 years who had survived a first myocardial infarction. The controls were 772 men admitted for noncardiac diagnoses. Men with history of rheumatic heart disease, cardiomyopathy, or prior cardiac surgery were excluded from both groups. Baldness was scored using the 12-point modified Hamilton scale. Other risk variables, such as blood pressure level, lipid levels, glucose intolerance, and cigarette smoking, were evaluated in interviews and self-reports. After adjusting for age, the OR estimate for baldness involving the vertex area of the scalp was 1.4 (95% CI, 1.20-1-90). The risk of myocardial infarction increased with the severity of baldness ($P < .01$), and the OR was 3.4 (95% CI, 1.70-7.00) for severe vertex baldness.

Herrera et al[19] assessed the relationship between the extent and progression of baldness and CAD in a cohort study of 2017 men from the Framingham Study who were 35 to 74 years old. Baldness was assessed twice 6 years apart on the front, sides, or back areas of the scalp. The subjects were classified as having no bald areas (n = 153), 1 bald area (n = 420), 2 bald areas (n = 587), and 3 bald areas (n = 857). A cohort of 403 men was divided into 3 groups: rapid progression (34 men whose condition progressed from no hair loss to hair loss in all areas); moderate progression (145 men who had no hair loss initially and 2 bald areas 6 years later or 1 initial bald area that progressed to all bald areas); and mild or no progression (224 men who had no change in their baldness, whose baldness progressed from 1 to 2 bald areas, or whose baldness had decreased). The cohort was followed up for up to 24 years for new occurrences of CAD. The results were analyzed with the Cox proportional hazards regression model, and all regressions were adjusted for age and CAD risk factors. The extent of baldness was not associated with any of the outcomes, but progression demonstrated a 2.4 OR for CAD (95% CI, 1.30-4.40), a 3.8 OR for CAD mortality (95% CI, 1.90-7.70), and a 2.4 OR for all-cause mortality (95% CI, 1.50-3.80).

The cohort study of Schnohr et al[20] involved 13 000 men and women, 30 to 79 years of age, who did not have ischemic heart disease at entry. During the 12-year follow-up period, 750 first myocardial infarctions were diagnosed. Baldness was scored in the frontoparietal region as "no bald triangle, bald triangle but >3 cm in front

The Most Recent Studies Reviewed*

Source, y	Type of Study	No. of Patients (Controls)	AGA Scoring	Highest Relative Risk	95% Confidence Interval	Biases
Lesko et al,[18] 1993	Case-control	665 (772)	Hamilton/Norwood scale	3.4	1.70-7.00	None
Herrera et al,[19] 1995	Cohort	2017	Hamilton progression	2.4	1.30-4.40	No family history; correct AGA diagnosis?
Schnohr et al,[20] 1995	Cohort	750	Modified Hamilton	1.7	1.10-2.50	Poor assessment of baldness
Ford et al,[21] 1996	Cohort	3932	Personal scores	2.5	1.01-6.24	No information on the type of baldness; no family history
Mirić et al,[23] 1998	Case-control	842 (712)	Personal scores	1.9	1.42-2.20	Only the survivors were studied
Lotufo et al,[24] 2000	Cohort	22 071	Hamilton simplified	1.4	1.11-1.67	Recall bias

*Other risk factors were adjusted for in all 6 studies. AGA indicates androgenetic alopecia.

of ear, bald triangle but ≤3 cm in front of ear." In the crown-top region, baldness was described as "thick hair, partly thin hair, bald spot or bald spot and front." The Cox proportional hazards model was used to control for various myocardial infarction risk factors. Separate models were used for men and women. In the analysis, the frontoparietal baldness variable was included in the model as no baldness or bald triangle. The "crown-top baldness" variable was included as no baldness or bald spot. Schnohr and colleagues found that "men lose their hair, but women keep it." There was a significant correlation ($P<.05$) between the small or large triangle of frontoparietal baldness and myocardial infarction in men (relative risk [RR], 1.6; 95% CI, 1.10-2.30). The relationship between crown-top baldness and myocardial infarction was borderline ($P<.06$), with a 1.2 RR (95% CI, 1.00-1.50) for men with a bald spot or bald top/front compared with men with thick hair/partly thin hair. The combined variable for baldness was significantly associated with a higher risk (RR, 1.7; $P<.02$; 95% CI, 1.10-2.50) for myocardial infarction in men but not in women.

Ford et al[21] studied 3932 men aged 26 to 76 years at entry. Baldness was scored as none, minimum, moderate, and severe. Scoring details have been published elsewhere, but were unavailable to me. Dermatologic examination was performed by a third-year dermatology resident. A proportional hazards regression model was used that included age and several risk factors for CAD. In a 14-year follow up, 378 men (9.6%) died of myocardial infarction and 939 (23.9%) had incident CAD events. There were 61 deaths (3%) and 239 CAD events (11.8%) in 2019 men younger than 55 years. Baldness was not associated with an increased rate of CAD incidence or mortality. For men younger than 55 years, however, the OR for severe baldness was 2.51 (95% CI,1.01-6.24) for CAD mortality and 1.72 (95% CI, 0.96-3.08) for CAD incidence.

In 1998, Schnohr et al[22] published another report on their series of 13 000 men and women and found no correlation between all-cause mortality and baldness.

Mirić et al[23] conducted a case-control study of 842 men younger than 60 years who were admitted for a first-time nonfatal myocardial infarction. The controls were 712 patients with acute peptic ulcer or traffic accident injuries who had normal electrocardiographic findings at rest and no history of CAD. Baldness was categorized as no baldness, frontal baldness, parietal baldness, and frontoparietal baldness. Traditional CAD risk factors were taken into account. Men with parietal baldness had a 1.90 adjusted OR for myocardial infarction (95% CI, 1.42-2.20), and those with frontoparietal baldness 1.68 (95% CI, 1.20-2.50).

Lotufo et al[24] examined the association of baldness and CAD in a retrospective cohort of 22 071 male physicians. Coronary artery disease was defined as nonfatal myocardial infarction, angina pectoris, and/or coronary revascularization. Baldness was not defined but was measured according to a simplified Hamilton scale. Subjects were asked which of the scale sketches mostly approximated their status at 45 years of age. Compared with men with no hair loss, those with frontal baldness had a relative risk of 1.09 (95% CI, 0.94-1.25), while those with mild, moderate, and severe vertex baldness had a relative risk of 1.23 (95% CI, 1.05-1.43), 1.32 (95% CI 1.10-1.59), and 1.36 (95% CI, 1.11-1.67), respectively. The findings of multivariate analysis, after potential confounders were controlled for, did not alter the results.

COMMENT

Some of the 15 studies were performed with a sample size that was too small to detect a difference; others had substantial biases, possibly yielding results in favor of a nonexistent association. The studies of Gertler and White[10] and Mirić et al[23] examined only patients who survived myocardial infarction. No information was collected about those who did not survive. In the study of Buechner et al,[11] cases were not defined and only percentages were provided. The original data for the studies of Hamby et al[13] and Emidy et al[17] are not available. In the studies of Ben Halim et al[14] and Cooke,[15] diabetes was an exclusion criterion, but persons with diabetes can also be bald. Risk factors for CAD were considered, but data were insufficient for adjusting, and the definition of "smoker" penalized the study. No statistical analysis was performed in Persson and Johansson's[16] study, but when it was done later, the findings proved that the authors' conclusion was unwarranted. Schnohr and colleagues' 1998 study dealt only with all-cause mortality. The remaining 6 articles[18-21,23,24] involved many subjects and provided ample methodological information and result data (**Table**).

DEFINITION OF BALDNESS

Baldness and *hair loss* are popular terms. *Androgenetic alopecia* is the correct medical designation for the disease that seems to be the topic of the studies that were reviewed. But was this the case?

Lesko et al[18] and Lotufo et al[24] mention male pattern baldness and, by referring to the Hamilton baldness scale as modified by Norwood, clearly refer to AGA. Also, Herrera et al[19] mention baldness and refer to the Hamilton baldness scale. Schnohr et al[20] and Mirić et al[23] merely use the term *baldness*, without referring to any known AGA scale. Ford et al[21] use *alopecia* and *baldness* indifferently. None of the authors of the 6 articles refers to AGA as the disease they were dealing with.

DIAGNOSIS OF AGA

If it is conceded that by *baldness*, the authors meant AGA, was AGA the real diagnosis? Androgenetic alopecia is only apparently easy to diagnose. Ford et al[21] (they were the only epidemiologists to rely at least on a dermatology resident) defined baldness as the condition that "corresponded to observable baldness upon the first encounter with the participant." Actually, AGA deserves a little more attention. In the 1995 study by Herrera et al,[19] for example, one wonders if such attention was totally lacking. Thirty-seven of their 420 patients who had 1 bald area in 1956 had no bald spots 6 years later. Since no active medication was available for AGA at the time, AGA would have spontaneously regressed in 12% of the patients. Herrera and colleagues confirmed this finding when they included men "who had decreased baldness" in their "no progression group." To the best of my knowledge, there is no evidence that AGA may spontaneously revert to normality. Thus, one would legitimately suspect that some other conditions (alopecia areata, trichotillomania, acute telogen effluvium?) could have been included in the study.

SEVERITY OF AGA

The severity of AGA was assessed in various ways. Lesko et al[18] relied on the telephone assessment of the subjects and the interviewing nurses using the Hamilton/Norwood scale and a continuous 5-point scale. It has been noted that the nurses were aware of the study hypothesis,[5] but, more important, the subjects' own scoring could have been biased in many ways. Dermatologists are well aware of the peculiarities inherent in patients' attitude toward the hair loss problem. Some patients dramatize their negligible alopecia. Lotufo et al[24] relied on the patients' memory of a condition that had developed 40 years before. This typical *recall bias* jeopardizes their conclusions, which were otherwise based on very low relative risks.

Herrera et al[19] relied on a simple classification based on the Hamilton scale. Observing their Figure 1, however, one wonders if in fact there is a difference between "two areas" and "all areas." Also, the finding that 42.5% of all-age individuals are "completely bald," ie, "comparable to Hamilton scale class VIII," is an exceedingly high percentage, and, in any case, is in contrast with prevalences found by other authors. Irrespective of myocardial condition, Lesko et al[18] found only 37 subjects with "severe vertex" baldness, corresponding to a mere 2.6%. None of their subjects belonged to Hamilton scale class VIII. Similarly, only 9.2% of the patients of Mirić et al[23] had "fronto-parietal" baldness, and only 11.6% of the patients of Schnohr et al[20] had a "bald top and front." Only 6.9% of the subjects of Ford et al[21] had "severe" baldness. This latter figure is also the general average obtained by summing all data from the extreme degrees of baldness in the studies of Lesko et al,[18] Mirić et al,[23] Schnohr et al,[20] and Ford et al.[21] Conversely, Mirić and colleagues, Schnohr and colleagues, Ford and colleagues, and Lesko and colleagues found no baldness in 35.5%, 58.9%, 64.0%, and 38.1%, respectively, of men younger than 55 years. Therefore, the finding of a 6.4% prevalence of "normal" men in Herrera and colleagues' study is too low to be reliable, at least when simple clinical observation is used as a diagnostic tool. Furthermore, this wide range of figures shows that *normality* of scalp hairiness is a vague concept.

The observations of Herrera et al[19] differ from those of the other authors, because Herrera and colleagues also examined the progression of AGA in their subjects over a 24-year follow-up period. Individuals whose condition progressed rapidly to complete baldness (7.8%) were reported to have a high risk for developing CAD. The doubts regarding Herrera and colleagues' diagnosis of AGA remain and may also have biased this conclusion. The concept of the early onset and/or rapid progression of AGA is probably important, however. In the study of Hamby et al,[13] 65% of the subjects with CAD were bald before 35 years of age. In the study of Lesko et al,[18] men at all stages of baldness with onset before the age of 25 years had an OR that was significantly higher than that of men with no hair loss (2.1; 95%CI, 1.20-3.50). Partial reanalysis reveals that subjects younger than 25 years with Hamilton class VI-VII had a nonadjusted OR of 3.26 (95% CI, 1.50-7.25). In the study of Schnohr et al,[20] the incidence of myocardial infarction in the 30- to 39-year-old group with the most severe type of AGA (11.1%) was not only definitely higher than in the same age group with less severe AGA but was almost comparable to the incidence in the 60- to 69-year-old group, irrespective of AGA severity.

CONCLUSIONS

Some questions regarding the possibly incorrect diagnosis of AGA and the lack of a satisfactory definition of the condition in the articles that were reviewed do not permit a definitive conclusion of the baldness/CAD issue. In fact, the real problem lies in the striking difference between the accuracy of the diagnosis of myocardial infarction based on clinical, laboratory, and instrumental approaches and the approximation of the diagnosis of AGA that, instead, relies on simple clinical observation "upon the first encounter with the participant." In recent years, major advances have been made in our knowledge of the main pathogenetic factors at work in AGA, but there is still no accurate method with which to es-

tablish the diagnosis and especially the severity of the condition.

Actually, 10 articles concluded that a relationship existed, especially in younger subjects with severe early-onset AGA. Therefore, the baldness/CAD issue cannot be discarded. The existence of a particular group of subjects who develop baldness before their 30s was recently recognized.[24] Such subjects present with an unusually elevated dihydrotestosterone-testosterone ratio compared with men with later-onset AGA, which may account for other clinical features, including thoracic hairiness[25] and, perhaps, a higher susceptibility for developing CAD. The baldness theory should be included as a secondary hypothesis in large epidemiological studies on CAD risk factors. Such studies should include dermatologic expertise for accurate, cost-effective evaluation of baldness.

Worry about the issue is currently increasing and should be primarily addressed by specialists. An epidemiological study with baldness as the primary hypothesis could also be undertaken. Progressing baldness can be considered as a manifest indicator of possible major risk for CAD, and balding young men may benefit from early aggressive screening of other better-known risk factors for CAD. Practicing dermatologists should be cautious, however, in addressing the problem with younger patients who are already concerned with their appearance so as not to aggravate their psychological distress.

Accepted for publication October 25, 2000.

Thanks are due to Cosetta Minelli, MD, for helping me to comply with the reviewers' requirements and to Diana Sears Panconesi, who kindly corrected my English.

Corresponding author: Alfredo Rebora, MD, Department of Dermatology, Clinica Dermatologica, Viale Benedetto XV, 7, 16132 Genova, Italy (e-mail: rebdermo @unige.it).

REFERENCES

1. Herrera CR, Lynch C. Is baldness a risk factor for coronary artery disease? a review of the literature. *J Clin Epidemiol.* 1990;43:1255-1260.
2. Predicting coronary artery disease [editorial]. *BMJ.* 1972;831:3.
3. Cheng TO. Baldness and coronary disease [letter]. *J Clin Epidemiol.* 1991;44:865.
4. Wilson PW, Kannel WB. Is baldness bad for the heart? *JAMA.* 1993;269:1035-1036.
5. Kleinmuntz B. Baldness and myocardial infarction [letter]. *JAMA.* 1993;270:48.
6. Sheikh K. Baldness and ischemic heart disease in a national sample of men. *Am J Epidemiol.* 1997;145:670-671.
7. Trevisan M, Farinaro E, Krogh V, et al. Baldness and coronary heart disease risk factors. *J Clin Epidemiol.* 1993;46:1213-1218.
8. Guzzo CA, Margolis DJ, Johnson J. Lipid profiles, alopecia, and coronary disease: any relationship? *Dermatol Surg.* 1996;22:479-486.
9. Sasmaz S, Senol M, Ozcan A, et al. The risk of coronary heart disease in men with androgenetic alopecia. *J Eur Acad Dermatol Venereol.* 1999;12:123-125.
10. Gertler MM, White PD. Findings on masculinity. In: *Coronary Heart Disease in Young Adults.* Cambridge, Mass: Harvard University Press; 1954:72-79.
11. Buechner HA, Brown M, Tretola RJ. Baldness and emphysema. *J La State Med Soc.* 1964;116:329-332.
12. Cotton SG, Nixon JM, Carpenter RG, Evans DW. Factors discriminating men with coronary heart disease from healthy controls. *Br Heart J.* 1972;34:458-464.
13. Hamby RI, Aintablian A, Hoffman I, et al. Is male baldness a coronary risk factor [abstract]? *Clin Res.* 1977;25:226A.
14. Ben Halim MM, Meyrick G. Jeans WD, Murphy D, Burton JL. Myocardial infarction, androgen, and the skin. *Br J Dermatol.* 1978;98:63-68.
15. Cooke NT. Male pattern alopecia and coronary artery disease in men. *Br J Dermatol.* 1979;101:455-458.
16. Persson B, Johansson BW. The Kockum study: twenty-two-year follow-up. *Acta Med Scand.* 1984;216:485-493.
17. Emidy L, Zhang H, Stamler J, et al. Does the state of your pate relate to your fate? baldness, coronary risk factors, and 25-year mortality in the Peoples Gas Company. In: Program and abstracts of the 27th Annual Conference on Cardiovascular Disease Epidemiology; 1987; Charleston, SC. Abstract 76. Cited by: Herrera CR, Lynch C. Is baldness a risk factor for coronary artery disease? a review of the literature. *J Clin Epidemiol.* 1990;43:1255-1260.
18. Lesko SM, Rosenberg L, Shapiro S. A case-control study of baldness in relation to myocardial infarction in men. *JAMA.* 1993;269:998-1003.
19. Herrera CR, D'Agostino RB, Gerstman BB, Bosco LA, Belanger AJ. Baldness and coronary heart disease rates in men from the Framingham Study. *Am J Epidemiol.* 1995;142:828-833.
20. Schnohr P, Lange P, Nyboe J, Appleyard M, Jensen G. Gray hair, baldness, and wrinkles in relation to myocardial infarction: the Copenhagen City Heart Study. *Am Heart J.* 1995;130:1003-1010.
21. Ford ES, Freedman DS, Byers T. Baldness and ischemic heart disease in a national sample of men. *Am J Epidemiol.* 1996;143:651-657.
22. Schnohr P, Nyboe J, Lange P, Jensen G. Longevity and gray hair, baldness, facial wrinkles, and arcus senilis in 13,000 men and women: the Copenhagen City Heart Study. *J Gerontol A Biol Sci Med Sci.* 1998;53:347-350.
23. Mirić D, Fabjianić D, Giunio L, et al. Dermatological indicators of coronary risk: a case-control study. *Int J Cardiol.* 1998;67:251-255.
24. Lotufo PA, Chae CU, Ajani UA, Hennekens CH, Manson JE. Male pattern baldness and coronary heart disease: the Physician's Health Study. *Arch Intern Med.* 2000;160:165-171.
25. Sreekumar GP, Pardinas J, Wong CQ, et al. Serum androgens and genetic linkage analysis in early onset androgenetic alopecia. *J Invest Dermatol.* 1999;113:277-279.