

Association of Androgenetic Alopecia With Smoking and Its Prevalence Among Asian Men

A Community-Based Survey

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Objectives: To evaluate the association of androgenetic alopecia (AGA) with smoking and to estimate its prevalence among Asian men.

Design: Population-based cross-sectional survey.

Setting: Tainan County, Taiwan.

Participants: The eligible population consisted of all male residents 40 years or older in Tainan County. A total of 740 subjects aged 40 to 91 years participated in the survey between April 10, 2005, and June 12, 2005.

Main Outcome Measures: Norwood and Ludwig classifications were used to assess the degree of hair loss. Information on smoking, together with other possible risk factors and age at onset of AGA, was collected using a questionnaire interview.

Results: After controlling for age and family history, statistically significant positive associations were noted be-

tween moderate or severe AGA (Norwood types \geq IV) and smoking status (odds ratio [OR], 1.77; 95% confidence interval [CI], 1.14-2.76), current cigarette smoking of 20 cigarettes or more per day (OR, 2.34; 95% CI, 1.19-4.59), and smoking intensity (OR, 1.78; 95% CI, 1.03-3.07). The OR of early-onset history for AGA grades increased in a dose-response pattern. Risk for moderate or severe AGA increased for family history of first-degree and second-degree relatives, as well as for paternal relatives.

Conclusions: The age-specific prevalence of AGA in Taiwan was compatible to that among Korean men but was lower than that among persons of white race/ethnicity. Smoking status, current amount of cigarette smoking, and smoking intensity were statistically significant factors responsible for AGA after controlling for age and family history. Patients with early-onset AGA should receive advice early to prevent more advanced progression.

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ANDROGENETIC ALOPECIA (AGA), a hereditary androgen-dependent disorder, is characterized by progressive thinning of the scalp hair defined by various patterns.¹ It is the most common type of hair loss in men.² Lower prevalence has been seen among Asian, Native American, and African American men, whereas high prevalence has been found among men of white race/ethnicity.³

The changes of AGA are androgen dependent and follow an inheritance mode with gene polymorphisms.³ To our knowledge, only 1 gene that encodes the androgen receptor has been identified.⁴ While prerequisites are androgens and a genetic predisposition, clinical practice has shown that simply blocking androgens does not result in the conversion of miniaturized follicles to terminal ones in advanced alopecia.¹ Some environmental factors such as smoking may play a role in the pathogenesis of AGA. The association between

smoking and AGA has been addressed in 3 studies⁵⁻⁷ with inconsistent results. One study⁵ showed a positive association, one study⁶ failed to demonstrate a statistically significant positive association, and another study⁷ showed the opposite findings, albeit statistically nonsignificant.

The first objective of this study was to estimate the prevalence and types of AGA among Taiwanese men and to compare our findings with those in other countries. The second objective was to investigate the association between smoking, family history, and other potential risk factors and AGA.

METHODS

STUDY SUBJECTS AND DESIGN

Eligible subjects in this study consisted of male residents 40 years or older in Tainan County, Taiwan. Our study was part of a community-based integrated screening program that invited residents 30 years or older. The details of a screening program similar to ours in Tainan

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Table 1. Age-Specific Types of Androgenetic Alopecia

Type	Age Group, y, No. (%)				Total
	40-49	50-59	60-69	≥70	
Norwood					
I	65 (65.7)	55 (57.9)	106 (42.7)	131 (44.0)	357
II	14 (14.1)	18 (18.9)	43 (17.3)	37 (12.4)	112
IIa	3 (3.0)	1 (1.1)	12 (4.8)	10 (3.4)	26
III	5 (5.1)	6 (6.3)	12 (4.8)	19 (6.4)	42
IIIv	2 (2.0)	1 (1.1)	12 (4.8)	12 (4.0)	27
IIIa	1 (1.0)	0	5 (2.0)	6 (2.0)	12
IV	6 (6.1)	5 (5.3)	21 (8.5)	20 (6.7)	52
IVa	1 (1.0)	0	1 (0.4)	3 (1.0)	5
V	1 (1.0)	3 (3.2)	12 (4.8)	14 (4.7)	30
Va	0	0	2 (0.8)	3 (1.0)	5
VI	1 (1.0)	2 (2.1)	10 (4.0)	23 (7.7)	36
VII	0	2 (2.1)	8 (3.2)	13 (4.4)	23
Ludwig					
L-I	0	0	4 (1.6)	6 (2.0)	10
L-II	0	2 (2.1)	0	1 (0.3)	3
L-III	0	0	0	0	0
Total	99	95	248	298	740

County have been published elsewhere.⁸ A community-based survey on prevalence of AGA was added to this integrated community-based screening program between April 10, 2005, and June 12, 2005, by inviting 929 residents who were selected from a population household registry of Tainan County in light of different screening criteria based on evidence-based medicine. Most participants were 40 years or older; this age group is subsidized by the local government. Only 5 subjects were aged 30 to 39 years, and they were excluded from the study. Of the remaining 924 subjects invited, the overall response rate to participate in our survey on AGA was 80.1% (740 of 924). The age-specific response rates were 83.9% for those aged 40 to 49 years (99 of 118), 81.9% for those aged 50 to 59 years (95 of 116), 84.9% for those aged 60 to 69 years (248 of 292), and 74.9% for those aged 70 years and older (298 of 398). There was a similar distribution with respect to age between respondents and nonrespondents. Therefore, the study population for age-specific prevalence in the AGA survey is representative of the underlying population invited to attend the screening. The study protocol was approved by the ethics committee of the Bureau of Tainan County, and informed consent was obtained from all participants.

CLASSIFICATION OF AGA

The diagnosis of AGA was based on the pattern of hair loss of the participants. Assessment of the degree of hair loss was obtained by independent public health nurses trained by a dermatologist. To classify the degree of AGA for each subject, the classification by Norwood,² a standard classification scheme with good test-retest reliability, was used. Some men with "female pattern hair loss" (noninvolvement of the frontal hairline) were assessed separately using the classification by Ludwig.⁹

Androgenetic alopecia among men with cosmetically significant male pattern baldness was defined as type III or greater (types III [including IIIv and IIIa], IV [including IVa], V [including Va], VI, and VII) according to the Norwood classification. The prevalence of more advanced degrees of alopecia characterized by only a remaining horseshoe fringe of hair (Norwood types V, Va, VI, and VII) was also estimated. The prevalence of AGA with female pattern hair loss was reported separately.

Norwood type IV of AGA represents the starting grade of severe frontal AGA concurrent with vertex AGA. Therefore, we divided AGA into 2 categories (mild AGA [Norwood types I-III] and moderate or severe AGA [Norwood types IV-VII]) to assess its association with smoking and other potential risk factors.

SMOKING AND OTHER POSSIBLE RISK FACTORS

In addition to classification of AGA, we collected information on age at onset of AGA together with smoking and other possible risk factors using a face-to-face questionnaire interview. Smoking status (never, quit, or current); age at start of smoking; and quantity, duration, and frequency of smoking were collected using a structured questionnaire administered by trained staff members. In addition to smoking status, we categorized cigarette smoking into 4 groups (never, quit, current smoker of <20 cigarettes per day, or current smoker of ≥20 cigarettes per day) for comparison. Regarding anthropometric measures, body weight, body height, waist circumference, and hip circumference were measured. Body mass index was calculated as weight in kilograms divided by height in meters squared. Blood pressure was measured twice with at least a 5-minute interval between measurements. Hypertension was defined as a systolic blood pressure of 140 mm Hg or higher and a diastolic blood pressure of 90 mm Hg or higher or as taking antihypertensive medication.

To collect biochemical markers, a venous blood sample was obtained after 12-hour fasting to check blood glucose, triglycerides, total cholesterol, and high-density lipoprotein cholesterol levels. Low-density lipoprotein cholesterol level was calculated in accord with the following formula: low-density lipoprotein cholesterol level = [(total cholesterol level - high-density lipoprotein cholesterol level - (triglyceride level/5)]. If the triglyceride level was greater than 400 mg/dL (to convert to millimoles per liter, multiply by 0.0113), the low-density lipoprotein cholesterol level was set as a missing value. Abnormal status for these biochemical markers included hypertriglyceridemia (triglyceride level, >160 mg/dL), hypercholesterolemia (total cholesterol level, >220 mg/dL [to convert to millimoles per liter, multiply by 0.0259]), low high-density lipoprotein cholesterol level (<42 mg/dL), high low-density lipoprotein cholesterol level (≥140 mg/dL), and high fasting glucose level (>110 mg/dL [to convert to millimoles per liter, multiply by 0.0555]). Dyslipidemia was defined as abnormal serum triglyceride, total cholesterol, high-density lipoprotein cholesterol, or low-density lipoprotein cholesterol level or as taking lipid-lowering drugs.

In each subject, a questionnaire regarding diagnoses of chronic diseases such as hypertension, diabetes mellitus, cardiovascular disease, antihypertensive and lipid-lowering drug use, timing of growth spurt in puberty, socioeconomic factors, alcoholic beverage consumption, and betel nut chewing history was completed. The socioeconomic factors included age, occupation, and level of education. Age was defined as the age at study enrollment. Information about baldness among the first-degree, second-degree, and third-degree relatives was also collected using a structured questionnaire administered by trained staff members. The degree of hair loss was recalled by the study subjects.

STATISTICAL ANALYSIS

The age-specific prevalence of AGA (Norwood types ≥III) was expressed as a percentage. A multivariate logistic regression model was used to assess the relationships of each possible risk factor such as smoking and degrees of relative relationships for family history of AGA (first-degree, second-degree, and third-

degree relatives) to moderate or severe AGA (Norwood types \geq IV). A cumulative logits model was used to assess the incremental effect of age at onset of AGA (\leq 40 vs $>$ 40 years) on AGA severity (Norwood types I-VII) with ordinal properties. The proportional odds assumption was checked for the 7 AGA grades. In addition, the association between family history of AGA and age at onset of AGA was assessed. Because age and family history of AGA were 2 established factors for AGA, they were retained in the model in the multivariate analysis. All odds ratios (ORs) and their 95% confidence intervals (CIs) were computed using statistical software (SAS version 8; SAS Institute, Inc, Cary, North Carolina).

RESULTS

Of 924 invited men, 740 participated in this survey. The mean \pm SD age of participants was 65.2 ± 11.2 (age range, 40-91 years).

PREVALENCE

The age-specific number and percentage of different AGA types are given in **Table 1**. **Figure 1** shows that the age-specific prevalence of AGA (Norwood types III [including IIIv and IIIa], IV [including IVa], V [including Va], VI, and VII) increased with advancing age. The age-specific prevalence of severe hair loss (Norwood types V, Va, VI, and VII) was low. In **Figure 2**, the age-specific prevalence of AGA (Norwood types \geq III) is compared with the results of previous studies.^{2,7,10-15}

Of 740 men examined, 48 (6.5%) were Norwood type A variants, higher than that reported in the study by Norwood (3%), and 13 (1.8%) manifested female pattern AGA. The prevalence did not increase with age for Norwood type A variants or for Ludwig grades L-I to L-III. For the groups aged 40 to 49, 50 to 59, 60 to 69, and 70 years or older, the age-specific prevalences of Norwood type A variants were 5.1%, 1.1%, 8.1%, and 7.4%, respectively, and those of female pattern AGA were 0.0%, 2.1%, 1.6%, and 2.4%, respectively (Table 1).

ASSOCIATION

In the univariate analysis, smokers were at increased risk of having moderate or severe AGA (Norwood types \geq IV) (OR, 1.61; 95% CI, 1.05-2.46). However, no statistically significant associations were found for intensity or duration of smoking (**Table 2**).

There were statistically significant positive associations between moderate or severe AGA and smoking status (OR, 1.77; 95% CI, 1.14-2.76), current cigarette smoking of 20 cigarettes or more per day (OR, 2.34; 95% CI, 1.19-4.59), smoking intensity (OR, 1.78; 95% CI, 1.03-3.07), and dyslipidemia (OR, 1.47; 95% CI, 1.01-2.14) after adjusting for age and family history of AGA (Table 2).

Smoking status and other statistically significant variables (age, family history of AGA, dyslipidemia, and betel nut chewing) were retained in the model and were adjusted for each other. A statistically significant association between smoking status and moderate or severe AGA (OR, 1.63; 95% CI, 1.00-2.65) still remained.

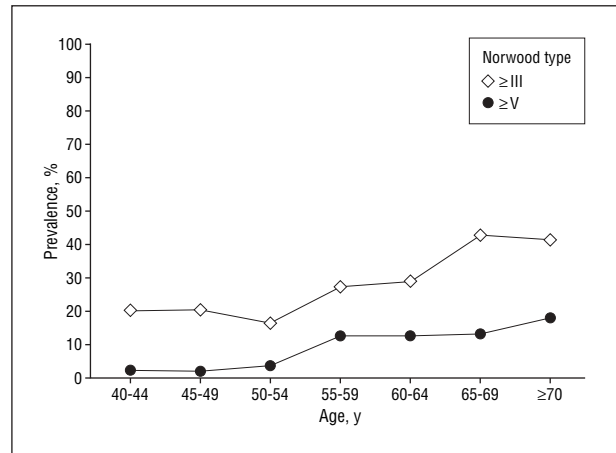


Figure 1. Age-specific prevalence of androgenetic alopecia.

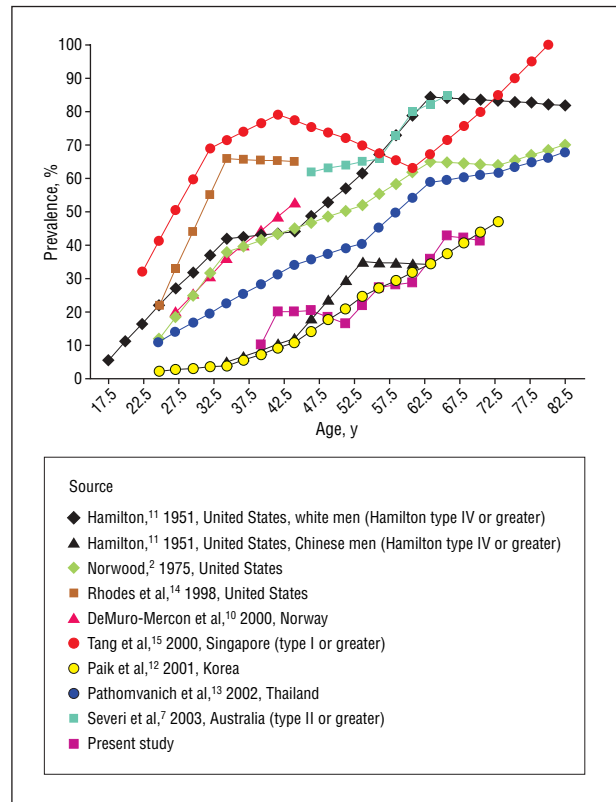


Figure 2. Prevalence of androgenetic alopecia in previous studies and in the present study.

EARLY-ONSET AGA AND AGA GRADES

The incremental effect of early-onset on severity of AGA was demonstrated by using the cumulative logits model. Findings suggest that earlier onset of AGA was associated with severe AGA. In those with early-onset AGA, AGA grades were statistically significantly worse (OR, 2.09; 95% CI, 1.14-3.85). Early-onset AGA was also related to family history of AGA (OR, 2.89; 95% CI, 1.42-5.88) using the logistic regression model.

Risk for moderate or severe AGA increased with degree of relationship for family history (first degree [OR, 13.38; 95% CI, 4.80-37.27], second degree [OR, 6.33; 95%

Table 2. Univariate and Multivariate Analyses of Risk Factors

Variable	Norwood Type, No.		Univariate Analysis		Multivariate Analysis ^a	
	I-III	IV-VII	OR (95% CI)	P Value	Adjusted OR (95% CI)	P Value
Smoking status						
Never	446	103	1 [Reference]		1 [Reference]	
Quit and current	105	39	1.61 (1.05-2.46)	.03	1.77 (1.14-2.76)	.01
Cigarette smoking						
Never	471	112	1 [Reference]		1 [Reference]	
Quit	27	12	1.92 (0.94-3.93)	.07	2.05 (0.98-4.32)	.06
Current, <20 cigarettes/d	43	12	1.21 (0.62-2.37)	.58	1.21 (0.60-2.44)	.59
Current, ≥20 cigarettes/d	35	15	1.86 (0.98-3.53)	.06	2.34 (1.19-4.59)	.01
Smoking intensity, duration × amount per day						
<350	24	5	1 [Reference]		1 [Reference]	
≥350	63	23	1.75 (0.60-5.14)	.31	1.78 (1.03-3.07)	.04
Duration of smoking, y						
<25	14	3	1 [Reference]		1 [Reference]	
≥25	76	27	1.66 (0.44-6.22)	.45	0.53 (0.11-2.46)	.42
Age, continuous variable, y			1.04 (1.02-1.06)	<.001	NA	
Family history of androgenetic alopecia						
No	548	125	1 [Reference]			
Yes	28	26	4.07 (2.31-7.18)	<.001	NA	
Educational achievement						
≥College	29	11	1 [Reference]		1 [Reference]	
<College	538	140	0.69 (0.33-1.41)	.30	0.55 (0.25-1.20)	.13
Waist circumference, cm						
≤90	397	103	1 [Reference]		1 [Reference]	
>90	176	48	1.05 (0.72-1.55)	.80	1.16 (0.78-1.73)	.47
Hip circumference, cm						
≤100	409	121	1 [Reference]		1 [Reference]	
>100	110	29	0.89 (0.56-1.41)	.62	0.98 (0.61-1.58)	.93
Body mass index ^b						
≤27	457	122	1 [Reference]		1 [Reference]	
>27	116	29	0.94 (0.60-1.47)	.78	1.12 (0.70-1.81)	.63
Hypertension						
No	261	57	1 [Reference]		1 [Reference]	
Yes	152	50	1.51 (0.98-2.31)	.06	1.42 (0.90-2.24)	.13
Diabetes mellitus						
No	493	131	1 [Reference]		1 [Reference]	
Yes	82	20	0.92 (0.54-1.55)	.75	0.98 (0.57-1.69)	.94
Cardiovascular disease						
No	64	16	1 [Reference]		1 [Reference]	
Yes	39	18	1.85 (0.84-4.04)	.12	1.64 (0.72-3.71)	.24
Dyslipidemia						
No	293	66	1 [Reference]		1 [Reference]	
Yes	283	85	1.33 (0.93-1.91)	.12	1.47 (1.01-2.14)	.047
Benign prostatic hypertrophy						
No	414	109	1 [Reference]		1 [Reference]	
Yes	98	35	1.36 (0.87-2.11)	.17	1.09 (0.68-1.74)	.72
Alcohol drinking						
No	492	122	1 [Reference]		1 [Reference]	
Yes	56	18	1.30 (0.74-2.30)	.37	1.62 (0.90-2.93)	.11
Betel nut chewing						
No	514	130	1 [Reference]		1 [Reference]	
Yes	30	11	1.45 (0.71-2.97)	.31	1.98 (0.93-4.20)	.08
Pubertal growth spurt, y						
>16	270	75	1 [Reference]		1 [Reference]	
≤16	291	70	0.87 (0.60-1.25)	.44	0.90 (0.61-1.32)	.60

Abbreviations: CI, confidence interval; NA, not available; OR, odds ratio.

^aAdjusted for age and family history.

^bCalculated as weight in kilograms divided by height in meters squared.

CI, 2.37-16.91], and third degree [OR, 5.32; 95% CI, 0.80-35.48]) (**Table 3**). The association between moderate or severe AGA and family history of AGA from paternal

relatives was statistically significant (OR, 12.69; 95% CI, 4.65-34.60), whereas the corresponding association from maternal relatives was not (OR, 3.07; 95% CI, 0.30-

31.32). The OR for the association between family history in both paternal and maternal relatives could not be estimated because there was no case with a positive family history from both paternal and maternal relatives among the subjects without AGA.

COMMENT

In our study, the age-specific prevalence of male pattern AGA was comparable with that among Korean men.¹² Compared with the results of another Asian study¹⁵ conducted in Singapore, our prevalence rate of AGA was lower. However, the comparison should be interpreted with caution because the definition of cosmetically significant AGA by Tang et al¹⁵ was Norwood type I or greater, which was different from the definition (Norwood types \geq III) used in our study. This may also explain why the age-specific prevalence of AGA in their study was much higher than that in our study. The age-specific prevalence of AGA in another Asian study¹³ conducted in Thailand was also higher than that in our study. However, race/ethnicity in Taiwan is different from that in Thailand.

The prevalence of AGA increased steadily with advancing age but was lower than that among persons of white race/ethnicity. An increase in risk of AGA with age reflects the natural progression of this condition. Advancing age is a risk factor for AGA in all men, irrespective of their family history. In addition, female pattern hair loss was observed in 13 of 740 men (1.8%) examined. Because AGA in some men may follow the female (Ludwig⁹) pattern, we suggest that the female pattern should be included in the classification of AGA in men.

A positive association between smoking and AGA was demonstrated in our study. Our results were consistent with findings by Mosley and Gibbs⁵ using a cross-sectional survey in a general surgical outpatient clinic in the United Kingdom. Unlike our study that was based on the general population, the generalizability of their study may be limited because respondents came from a general surgical outpatient clinic. In addition, no confounders were considered, and the dose-response relationship to AGA with respect to amount and duration of smoking was not elucidated.

Our positive results were not comparable with the findings from 2 studies.^{6,7} Severi et al⁷ reported a lower risk of AGA among current smokers (OR, 0.86; 95% CI, 0.54-1.38) and exsmokers (OR, 0.91; 95% CI, 0.65-1.29), although the results were not statistically significant. Nevertheless, details of smoking history were lacking, and the age at onset of AGA was unknown. In addition, different AGA classification categories instead of the Norwood classification were used. Therefore, it was difficult to compare their results with those of other studies. Matilainen et al⁶ found that the prevalence of current smoking among women with extensive hair loss (Ludwig grades \geq L-II to L-III) did not differ from those with normal or minimal hair loss ($P=.92$). However, the main objective of the study was not tailored to smoking history, and no definition of smoking was addressed.

The mechanisms by which smoking causes hair loss may be multifactorial.¹ First, cigarette smoking may be

Table 3. Association Between Family History of Androgenetic Alopecia (AGA) and Moderate or Severe AGA After Adjusting for Age

Variable	OR (95% CI)	P Value
Family history of AGA in different degrees of relatives		
None	1 [Reference]	
Third-degree relatives	5.32 (0.80-35.48)	.08
Second-degree relatives	6.33 (2.37-16.91)	<.001
First-degree relatives	13.38 (4.80-37.27)	<.001
Parental family history of AGA		
None	1 [Reference]	
Paternal family history positive	12.69 (4.65-34.60)	<.001
Maternal family history positive	3.07 (0.30-31.32)	.34
Both positive	NA	NA

Abbreviations: CI, confidence interval; NA, not available; OR, odds ratio.

deleterious to the microvasculature of the dermal hair papilla. Second, smoke genotoxicants may do damage to DNA of the hair follicle. Third, smoking may lead to an imbalance in the follicular protease or antiprotease system. Smoking-induced oxidative stress may lead to the release of proinflammatory cytokines that, in turn, results in follicular microinflammation and fibrosis. Fourth, cigarette smoking may yield a relative hypoestrogenic state by inducing increased hydroxylation of estradiol and inhibition of aromatase.¹

Although the genetic basis of AGA is well documented in the medical community and among the general population, there are few studies investigating the familial aggregation of AGA. Chumlea et al¹⁶ reported that the risk of AGA increased among men with a positive maternal grandfather history and even more with a history of hair loss in their father. In our study, moderate or severe AGA was statistically significantly associated with family history of AGA among the first-degree and second-degree relatives but not among the third-degree relatives after adjusting for age. The highest OR was associated with a family history among first-degree relatives. In addition, family history of AGA among paternal relatives was statistically significantly predictive of moderate or severe AGA after adjusting for age. Our findings do not support an association between moderate or severe AGA and family history of AGA among maternal relatives. The effect of family history among both paternal and maternal older relatives could not be assessed because no case was found among subjects without AGA.

Moreover, we found that family history of AGA is statistically significantly associated with the risk of early-onset AGA. This implies that those with a family history of AGA may have a higher risk of early-onset AGA and a higher risk of developing severe AGA. Most important, early-onset AGA (age at onset \leq 40 years) showed a statistically significant dose-dependent association with AGA grade after adjusting for age and family history. From the clinical point of view, this suggests that patients with early-onset AGA should receive early advice to prevent the deterioration of AGA.

In conclusion, our study showed that the prevalence of AGA among men in Taiwan was lower than that among persons of white race/ethnicity. The severity of AGA was also milder in Taiwan. We suggest that a female pattern should be included in the classification because AGA in some men may follow this Ludwig pattern. We also confirmed that smoking status, current amount of cigarette smoking, and smoking intensity play important roles in the development of moderate or severe AGA.

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Author Contributions: Drs Su and Chen had full access to all the data in the study and take responsibility for the integrity of the data and the accuracy of the data analysis. *Study concept and design:* Chen. *Acquisition of data:* Su. *Analysis and interpretation of data:* Chen and Su. *Drafting of the manuscript:* Su. *Critical revision of the manuscript for important intellectual content:* Chen. *Statistical analysis:* Chen. *Administrative, technical, and material support:* Chen. *Study supervision:* Chen.

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