Association of Hypertension and Hyperaldosteronism with Androgenetic Alopecia in Male Patients: Case control study.

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Table of Contents

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Ghadah Alhetheli[1*]; Sulaiman Almarshoud[2*]; Abdurrahman Ghorab[3*]; Rahaf Aldawish[2]; Abdulmalik Aldawsari[4*]; Abdullah Alowais[4*]

[1]Qassim University college of medicine Buraidah SA
[2]King Fahad Specialist Hospital, Dermatology Buraidah SA
[3]Qassim University Medical City, Dermatology Buraidah SA
[4]King Fahad Specialist Hospital, Medicine Buraidah SA
[*]these authors contributed equally

Corresponding Author:
Ghadah Alhetheli
Qassim University
college of medicine
abubaker alsiddiq road, Olaya
building#2084
Buraidah
SA

Abstract

Background: Androgenetic alopecia (AGA), the predominant cause of hair loss (1)(2), is a non-scarring alopecia with a genetic predisposition. AGA manifests through the miniaturization of large terminal follicles into smaller versions due to dihydrotestosterone's (DHT) impact on scalp hair androgen receptors. Characteristically, men experience hair thinning in the vertex and frontotemporal regions, while women tend to see it along the crown (3).

Aldosterone plays a crucial role in maintaining electrolyte balance and blood pressure regulation, which is pivotal for survival. An excess of aldosterone can manifest in a spectrum of symptoms ranging from muscular weakness and cramping to an increase in blood pressure. Intriguingly, recent studies have disclosed the existence of mineralocorticoid receptors in the dermal strata of mice, which when stimulated, lead to hair loss. The discovery of these receptors highlights a novel dermatological dimension to the hormone's activity, pointing to a broader systemic impact that extends to influence hair follicle function (4)(5).

Numerous studies have suggested a relationship between AGA and increased cardiovascular risk, particularly concerning cardiovascular diseases (CVD) and metabolic syndrome (MtS) (6-9). The evidence points to a substantial link, emphasizing AGA as a potential marker for these conditions. However, despite the prevalence of these findings, research exploring the connection between AGA and specific ailments such as hypertension (HTN) and hyperaldosteronism remains comparatively limited and controversial (9)(10).

In exploring the relationship between AGA and cardiovascular morbidity, Mansouri, P. et al. identified a significant relationship between AGA in females and both coronary artery disease (CAD) and a history of myocardial infarction (MI) (6).

As for the relationship between AGA and MtS, a study conducted by Dharam Kumar, et al. demonstrated a notable correlation between the severity of AGA and the presence of MtS (7). Conversely, subsequent recent studies in China and Saudi Arabia had failed to replicate these findings, indicating no significant associations between AGA and MtS (8)(9).

While research into association of AGA with HTN and hyperaldosteronism is relatively nascent, emerging studies provide insightful data. A case-control study from Spain found that women with early-onset AGA had significantly higher systolic and diastolic blood pressure, as well as elevated aldosterone levels, when compared to non-alopecic controls (5). Similarly, a research conducted in Egypt observed that men with AGA exhibited significantly higher systolic blood pressure and aldosterone concentrations; however, unlike the Spanish study, diastolic blood pressure readings did not differ significantly from those without AGA (10).

Objective: Given the paucity of researches on AGA's association with HTN and hyperaldosteronism particularly within the Saudi Arabian, our study endeavors to elucidate this association. We hypothesize that AGA may serve as an early indicator of hypertensive and hyperaldosteronism states.

Methods: This case-control study was conducted at the Department of Dermatology, Qassim University Medical City, from September 2023 to January 2024. The study protocol was approved by the Local Ethical Committee of Qassim University with reference number: (23-42-08) and was performed according to the guidelines of Declaration of Helsinki. All participants were informed about the purpose of the study, and their permission on the forms of written consent was obtained. The study comprised 20 male patients diagnosed with AGA and 20 age and sex-matched healthy controls.
Participants were included based on the following criteria: males aged 18-65 years with clinically established diagnosis of AGA. Exclusion criteria encompassed any history usage of hormonal medications (e.g., testosterone, corticosteroids), diagnosed states of hyperaldosteronism and pre-existing cardiovascular disease including HTN. Controls were matched in age and selected from individuals presenting with other dermatological conditions, excluding those meeting any patient group exclusion criteria.
Data were extracted from electronic medical records and official reports, with confidentiality maintained strictly by investigator access only.

Data Collection:
All participants underwent detailed medical history and physical examination including vital signs checking. Weight, height and Body Mass Index (BMI) were obtained. Blood pressure readings and serum aldosterone levels were obtained during morning sessions. For AGA assessment, the Ludwig scale was employed.

Statistical Analysis:
Data were systematically entered into a Microsoft Excel database and subsequently analyzed with SPSS software (version 26). Descriptive statistics, including mean and standard deviation for continuous variables, along with frequencies and percentages for categorical variables, were calculated. The chi-square test and independent samples t-test were applied for comparative analyses of categorical and continuous variables, respectively. One-way ANOVA was utilized for comparisons across multiple groups. A P-value threshold of less than 0.05 was predetermined to signify statistical significance

Results: Demographic Characteristics
Demographic and clinical data of both patients and healthy controls are presented on [Table 1]. The age of the participants ranged from 20 to 62 years, with Body Mass Index (BMI) scores spanning from 17 to 43. Notably, only five participants reported a history of smoking, whereas the majority, comprising thirty-five individuals, did not. Additionally, comorbidities were present in seven participants, in contrast to thirty-three who reported none.

Descriptive Statistics
The blood pressure (Systolic/Diastolic measures), and Aldosterone levels in patients group appeared to be slightly lower than for participants in the control group. Detailed statistical values and comparisons are provided on [Table 2].

Inferential Statistics
Series of statistical analyses were performed to explore the association between androgenetic alopecia, hypertension, and hyperaldosteronism. Initially, three independent sample t-tests were conducted to assess the differences in blood pressure and aldosterone levels relative to the presence of androgenetic alopecia. These analyses yielded no significant differences, with results for aldosterone levels ($t(38) = -0.3$, $p = 0.766$), systolic blood pressure ($t(37) = -1.34$, $p = 0.188$), and diastolic blood pressure ($t(37) = -1.31$, $p = 0.180$) as shown on [Table 3].
Subsequent to the t-tests, point-biserial Pearson correlation analyses were implemented to further investigate the relationships between androgenetic alopecia and both hypertension and hyperaldosteronism. Although these correlations did not reach statistical significance, they illuminated a negative association, suggesting a potential inverse relationship between androgenetic alopecia and these conditions [Table 4].
In light of these findings, additional analyses were conducted to explore the potential influence of age, familial history of androgenetic alopecia, and smoking history on the relationships with androgenetic alopecia and hyperaldosteronism.

Androgenetic alopecia
An independent sample t-test was conducted to compare ages between control and case groups, revealing no significant age difference ($t(38) = 1.08$, $p = 0.287$). Additionally, a chi-square test of association indicated that smoking history does not significantly correlate with androgenetic alopecia ($?2(1) = 0.229$, $p = 0.633$). Conversely, a significant association was found between family history of androgenetic alopecia and the likelihood of developing the condition ($?2(1) = 4.912$, $p = 0.027$). Notably, 32.5% of participants with androgenetic alopecia reported a family history, compared to 17.5% in the control group [Tables 5 and 6] [Figure1].

Hyperaldosteronism
A multiple linear regression test was conducted to evaluate the relationship between age, family history of androgenetic alopecia,

smoking history, and hyperaldosteronism. The analysis revealed that the model as a whole was not significant in predicting hyperaldosteronism: F(3, 36) = .582, p = 631. In addition, none of these factors were significant in predicting one's hyperaldosteronism, as shown in (Table 7).

Degree of Androgenetic Alopecia (Norwood Scale)

To investigate the impact of the severity of androgenetic alopecia (AGA) on physiological measures, one-way ANOVA tests were utilized to analyze differences in blood pressure and aldosterone levels among AGA patients categorized according to the Norwood scale. The analysis encompassed systolic and diastolic blood pressure, as well as aldosterone levels. The results, detailed on [Table 8.1 and 8.2] , indicated no statistically significant differences across the different degrees of AGA severity.

Conclusions: In this case-control study, we rigorously evaluated the association between AGA and two health conditions—hypertension and hyperaldosteronism. Contrary to previous studies that posited potential correlations (5)(10), our findings reveal no significant disparities in systolic and diastolic blood pressure or aldosterone levels between AGA patients and healthy control. This evidence suggests that AGA may not serve as a reliable clinical predictor for hypertension or hyperaldosteronism.

Our results align with those from similar studies conducted by Danesh-Shakiba et al. (11) in Iran and by Ozbas Gok et al. (12) in Turkey, which also reported no significant association between AGA and health conditions. In contrast, a study from Egypt reported by El Esawy et al. (10), sharing similar objective, and similar study design settings (case-control, male-only participants), indicated a positive association. These results divergence may stem from regional or genetic variations that were not controlled in the study settings.

Additionally, our findings depart from research such as that by Arias-Santiago et al. (5), and Mansouri et al. (6), which documented higher rates of hypertension among female AGA sufferers across various regions. Such inconsistencies highlight a potential gender-specific prevalence that underscores the influence of other non-AGA-related factors on hypertension and hyperaldosteronism, as suggested by research from Akasaka et al. (13), Connelly et al. (14), and others (15-22). These studies imply that factors specific to male physiology may diminish the impact of AGA on the investigated conditions.

Further, our analysis considers the role of geographical variability in health outcomes, as discussed by Dummer et al. (23) and Volinn et al. (24), who emphasized how environmental factors and healthcare accessibility influence health status. Studies by Kershaw et al. (25) and Reddy et al. (20) corroborate this, demonstrating that geographic variation affects both the prevalence and management of hypertension.

Given the comprehensive analysis and corroborative findings from diverse studies, it is prudent for healthcare practitioners, particularly in Saudi Arabia and similar regions, to reassess the predictive significance of AGA concerning hypertension and hyperaldosteronism. The lack of a significant association in our study challenges the traditional view of AGA as an indicator of these conditions. Future research should thus broaden its demographic and regional scope within Saudi Arabia to further explore this relationship, emphasizing the need for larger sample sizes to strengthen the reliability of the data.

This nuanced understanding of AGA's diagnostic value, especially in gender-specific and geographic contexts, offers a refined perspective that can influence clinical approaches and health policy decisions, promoting more tailored and effective healthcare strategies.

Conclusion

This study contributes to a growing body of evidence suggesting that androgenetic alopecia should not be considered as a marker for hypertension or hyperaldosteronism. Our findings are particularly relevant to the medical community, indicating that the presence of AGA alone should not lead healthcare providers to anticipate these conditions in patients. Moving forward, research should focus on elucidating the complex interplay of genetic, environmental, and regional factors that influence these associations, which may inform more tailored and effective clinical assessments.

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Alhetheli G [1], Almarshoud S [2], Ghorab A [3], Aldawish R [2], Aldawsari A [4], Alowais A [4]

Ghadah Alhetheli (MD)
1. Department of Dermatology, College of Medicine, Qassim University, Buraydah, Saudi Arabia. ghthly@qu.edu.sa. https://orcid.org/0000-0002-3896-1873

Sulaiman Almarshoud (MD)
2. Department of Dermatology, King Fahad Specialist Hospital, Buraydah, Saudi Arabia. Sulimanqum@gmail.com

Abdurrahman Ghorab (MD)
3. Department of dermatology and Cutaneous surgery, Qassim University Medical City, Al-Qassim, Saudi Arabia. aghorab@qumc.edu.sa

Rahaf Aldawish (MD)
2. Department of Dermatology, King Fahad Specialist Hospital, Buraydah, Saudi Arabia. Rahafdwish@gmail.com

Abdulmalik Aldawsari (MD)
4. Department of Internal Medicine, King Fahad Specialist Hospital, Buraydah, Saudi Arabia. mlk.aldossari@gmail.com

Abdullah Alowais (MD)
4. Department of Internal Medicine, King Fahad Specialist Hospital, Buraydah, Saudi Arabia. d.abdullahfa@gmail.com

Corresponding Author:

Ghadah Alhetheli
Department of Dermatology
College of Medicine
Qassim University, Buraidah, Saudi Arabia
Tel: +966504956030
Email: ghthly@qu.edu.sa

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Abstract

Background: Androgenetic alopecia (AGA), characterized by patterned hair loss due to genetic predisposition, is widely investigated for potential associations with systemic conditions. Previous studies have suggested a correlation between AGA and both hypertension and hyperaldosteronism, although findings remain inconsistent. This study aimed to evaluate the association between AGA, hypertension, and hyperaldosteronism in a given community in Saudi Arabia.

Methods: We conducted a case-control study involving 20 males with AGA and 20 age and sex-matched healthy controls. Blood pressure and serum aldosterone levels were measured then analyzed.

Results: Statistical analysis revealed no significant differences between the two groups in aldosterone levels ($t(38) = -0.3$, $p = 0.766$), systolic blood pressure ($t(37) = -1.34$, $p = 0.188$), or diastolic blood pressure ($t(37) = -1.31$, $p = 0.180$).

Conclusion: Our findings do not support the assumption that AGA is associated with higher risks of hypertension or hyperaldosteronism. This is align with other studies questioning the purported link, suggesting that AGA should not be considered as an indicator of these conditions. Further clinical studies are needed to fully elucidate the relationship between AGA and systemic health.

Keywords

AGA, Androgenetic alopecia , Hyperaldosteronism, Hypertension, Male pattern hair loss

Introduction:

Androgenetic alopecia (AGA), the predominant cause of hair loss [(1)(2)], is a non-scarring alopecia with a genetic predisposition. AGA manifests through the miniaturization of large terminal follicles into smaller versions due to dihydrotestosterone's (DHT) impact on scalp hair androgen receptors. Characteristically, men experience hair thinning in the vertex and frontotemporal regions, while women tend to see it along the crown [(3)].

Aldosterone plays a crucial role in maintaining electrolyte balance and blood pressure regulation, which is pivotal for survival. An excess of aldosterone can manifest in a spectrum of symptoms ranging from muscular weakness and cramping to an increase in blood pressure. Intriguingly, recent studies have disclosed the existence of mineralocorticoid receptors in the dermal strata of mice, which when stimulated, lead to hair loss. The discovery of these receptors highlights a novel dermatological dimension to the hormone's activity, pointing to a broader systemic impact that extends to influence hair follicle function [(4)(5)].

Numerous studies have suggested a relationship between AGA and increased cardiovascular risk, particularly concerning cardiovascular diseases (CVD) and metabolic syndrome (MtS) [(6-9)]. The evidence points to a substantial link, emphasizing AGA as a potential marker for these conditions. However, despite the prevalence of these findings, research exploring the connection between AGA and specific ailments such as hypertension (HTN) and hyperaldosteronism remains comparatively limited and controversial [(9)(10)].

In exploring the relationship between AGA and cardiovascular morbidity, Mansouri, P. et al. identified a significant relationship between AGA in females and both coronary artery disease (CAD) and a history of myocardial infarction (MI) [(6)].

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While research into association of AGA with HTN and hyperaldosteronism is relatively nascent, emerging studies provide insightful data. A case-control study from Spain found that women with early-onset AGA had significantly higher systolic and diastolic blood pressure, as well as elevated aldosterone levels, when compared to non-alopecic controls [(5)]. Similarly, a research conducted in Egypt observed that men with AGA exhibited significantly higher systolic blood pressure and aldosterone concentrations; however, unlike the Spanish study, diastolic blood pressure readings did not differ significantly from those without AGA [(10)].

Given the paucity of researches on AGA's association with HTN and hyperaldosteronism particularly within the Saudi Arabian, our study endeavors to elucidate this association. We hypothesize that AGA may serve as an early indicator of hypertensive and hyperaldosteronism states.

Methodology:

This case-control study was conducted at the Department of Dermatology, Qassim University Medical City, from September 2023 to January 2024. The study protocol was approved by the Local Ethical Committee of Qassim University with reference number: (23-42-08) and was performed according to the guidelines of Declaration of Helsinki. All participants were informed about the purpose of the study, and their permission on the forms of written consent was obtained. The study comprised 20 male patients diagnosed with AGA and 20 age and sex-matched healthy controls.

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Data were extracted from electronic medical records and official reports, with confidentiality maintained strictly by investigator access only.

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Statistical Analysis:

Data were systematically entered into a Microsoft Excel database and subsequently analyzed with SPSS software (version 26). Descriptive statistics, including mean and standard deviation for continuous variables, along with frequencies and percentages for categorical variables, were calculated. The chi-square test and independent samples t-test were applied for comparative analyses of categorical and continuous variables, respectively. One-way ANOVA was utilized for comparisons across multiple groups. A P-value threshold of less than 0.05 was predetermined to signify statistical significance

Results

Demographic Characteristics

Demographic and clinical data of both patients and healthy controls are presented on [Table 1]. The

age of the participants ranged from 20 to 62 years, with Body Mass Index (BMI) scores spanning from 17 to 43. Notably, only five participants reported a history of smoking, whereas the majority, comprising thirty-five individuals, did not. Additionally, comorbidities were present in seven participants, in contrast to thirty-three who reported none.

Table 1. Demographic characteristics

	Cases		**Controls**	
	n = 20	*Percentages %*	*n* = 20	*Percentages %*
Gender	Male	100	Male	100
BMI (Range)	17 - 37		20 - 43	
Smoking history				
Yes	2	10	3	15
No	18	90	17	85
Comorbidities				
Yes	4	20	3	15
No	16	80	17	85

BMI - Body Mass Index

Descriptive Statistics

The blood pressure (Systolic/Diastolic measures), and Aldosterone levels in patients group appeared to be slightly lower than for participants in the control group. Detailed statistical values and comparisons are provided on [Table 2].

Table 2. Descriptive Statistics

		Control	**Cases**
Age	Mean	30.80	34.50
	Range	28	42
Systolic	Mean	126.25	120.95
	Range	48	38
Diastolic	Mean	81.15	77.42
	Range	32	43
Aldosterone level (ng/dL)	Mean	7.35	6.90
	Range	24	12

Inferential Statistics

Series of statistical analyses were performed to explore the association between androgenetic

alopecia, hypertension, and hyperaldosteronism. Initially, three independent sample t-tests were conducted to assess the differences in blood pressure and aldosterone levels relative to the presence of androgenetic alopecia. These analyses yielded no significant differences, with results for aldosterone levels (t(38) = -0.3, p = 0.766), systolic blood pressure (t(37) = -1.34, p = 0.188), and diastolic blood pressure (t(37) = -1.31, p = 0.180) as shown on [Table 3].

Table 3. Independent sample t-test

	t-statistic	Degree of Freedom	p-value
Aldosterone level (ng/dL)	-0.3	38	0.766
Systolic	-1.34	37	0.188
Diastolic	-1.31	37	0.180

Subsequent to the t-tests, point-biserial Pearson correlation analyses were implemented to further investigate the relationships between androgenetic alopecia and both hypertension and hyperaldosteronism. Although these correlations did not reach statistical significance, they illuminated a negative association, suggesting a potential inverse relationship between androgenetic alopecia and these conditions [Table 4].

Table 4. Correlation between serum Aldosterone levels and Systolic/Diastolic blood pressure.

A: Correlation - Systolic

		Group	Systolic blood pressure
Group	Pearson Correlation	1	-.214
	Sig. (2-tailed)		.191
Systolic blood pressure	Pearson Correlation	-.214	1
	Sig. (2-tailed)	.191	

Sig: Significance

B: Correlations - Diastolic

		Group	Diastolic blood pressure
Group	Pearson Correlation	1	-.219
	Sig. (2-tailed)		.180
Diastolic blood pressure	Pearson Correlation	-.219	1
	Sig. (2-tailed)	.180	

Sig: Significance

C: Correlation - Aldosterone

	Group	Aldosterone level (ng/dL)

Group	Pearson Correlation	1	-.049
	Sig. (2-tailed)		.766
Aldosterone level (ng/dL)	Pearson Correlation	-.049	1
	Sig. (2-tailed)	.766	

Sig: Significance

In light of these findings, additional analyses were conducted to explore the potential influence of age, familial history of androgenetic alopecia, and smoking history on the relationships with androgenetic alopecia and hyperaldosteronism.

Androgenetic alopecia

An independent sample t-test was conducted to compare ages between control and case groups, revealing no significant age difference ($t(38) = 1.08$, $p = 0.287$). Additionally, a chi-square test of association indicated that smoking history does not significantly correlate with androgenetic alopecia ($\chi2(1) = 0.229$, $p = 0.633$). Conversely, a significant association was found between family history of androgenetic alopecia and the likelihood of developing the condition ($\chi2(1) = 4.912$, $p = 0.027$). Notably, 32.5% of participants with androgenetic alopecia reported a family history, compared to 17.5% in the control group [Tables 5 and 6] [Figure1].

Table 5. Cross-tabulation between family history of AGA and case/control groups

		Family History of AGA		
		Family history of AGA absent	Family history of AGA present	Total
Group	Case Group	7	13	20
	Control Group	14	6	20
Total		21	19	40

AGA - Androgenetic alopecia

Table 6. Chi-square test of family history and case/control groups

	Value	Degree of Freedom	Asymptotic Significance (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	4.912[a]	1	.027		

Continuity Correction[b]	3.609	1	.057			
Likelihood Ratio	5.019	1	.025			
Fisher's Exact Test				.056	.028	
Linear-by-Linear Association	4.789	1	.029			
Number of Valid Cases	40					

a. 0 cells (.0%) have expected count less than 5. The minimum expected count is 9.50.
b. Computed only for a 2x2 table
Sig: Significance

Model		Sum of Squares	Degree of Freedom	Mean Square	F-statistic	P-value
1	Regression	39.594	3	13.198	.582	.631[b]
	Residual	816.781	36	22.688		
	Total	856.375	39			

a. Dependent Variable: Aldosterone level (ng/dL)

b. Predictors: (Constant), Family History of AGA, Age, Smoking history

Hyperaldosteronism

A multiple linear regression test was conducted to evaluate the relationship between age, family history of androgenetic alopecia, smoking history, and hyperaldosteronism. The analysis revealed that the model as a whole was not significant in predicting hyperaldosteronism: $F(3, 36) = .582$, $p = 631$. In addition, none of these factors were significant in predicting one's hyperaldosteronism, as shown in (Table 7).

Table 7. Relationship between Age, family history of AGA, smoking history, and hyperaldosteronism.

a- ***ANOVA***

b- ***Coefficients[a]***

Model		Unstandardized Coefficients		Standardized Coefficients		
		Beta	Standard Error	Beta	t-statistic	P-value
1	(Constant)	5.873	2.705		2.171	.037

	Age	.040	.071	.092	.558	.581
	Smoking history	-2.523	2.328	-.180	-1.083	.286
	Family History of AGA	.566	1.565	.061	.361	.720

a. Dependent Variable: Aldosterone level (ng/dL)

Degree of Androgenetic Alopecia (Norwood Scale)

To investigate the impact of the severity of androgenetic alopecia (AGA) on physiological measures, one-way ANOVA tests were utilized to analyze differences in blood pressure and aldosterone levels among AGA patients categorized according to the Norwood scale. The analysis encompassed systolic and diastolic blood pressure, as well as aldosterone levels. The results, detailed in Table 8.1 and 8.2 , indicated no statistically significant differences across the different degrees of AGA severity.

Table 8.1 The descriptive statistics of Blood pressure and Aldosterone levels based on degree of AGA

		Number	Mean	Standard Deviation
Systolic	Stage 1	1	130.00	.
	Stage 2	4	120.00	3.651
	Stage 3	1	136.00	.
	Stage 3 Vertex	2	115.50	2.121
	Stage 4	3	116.33	8.963
	Stage 5	5	126.40	12.720
	Stage 6	2	107.50	2.121
	Stage 7	1	125.00	.
	Total	19	120.95	10.052
Diastolic	Stage 1	1	83.00	.
	Stage 2	4	79.75	12.093
	Stage 3	1	83.00	.
	Stage 3 Vertex	2	74.00	1.414
	Stage 4	3	71.00	14.731
	Stage 5	5	79.20	4.658
	Stage 6	2	74.50	4.950
	Stage 7	1	80.00	.
	Total	19	77.42	8.355
Aldosterone level (ng/dL)	Stage 1	1	3.00	.
	Stage 2	4	6.25	3.500
	Stage 3	1	2.00	.
	Stage 3 Vertex	2	7.00	5.657

	Stage 4	3	9.00	6.083
	Stage 5	6	6.33	1.366
	Stage 6	2	12.50	2.121
	Stage 7	1	4.00	.
	Total	20	6.90	3.865

Table 8.2 The Inferential statistics of Blood pressure and Aldosterone levels based on degree of AGA. *ANOVA*

		Sum of Squares	Degree of Freedom	Mean Square	F-statistic	P-value
Systolic BP	Between Groups	962.081	7	137.440	1.764	.192
	Within Groups	856.867	11	77.897		
	Total	1818.947	18			
Diastolic BP	Between Groups	270.582	7	38.655	.431	.863
	Within Groups	986.050	11	89.641		
	Total	1256.632	18			
Aldosterone level (ng/dL)	Between Groups	127.217	7	18.174	1.393	.293
	Within Groups	156.583	12	13.049		
	Total	283.800	19			

BP - Blood Pressure

Discussion

In this case-control study, we rigorously evaluated the association between AGA and two health conditions—hypertension and hyperaldosteronism. Contrary to previous studies that posited potential correlations [5][10], our findings reveal no significant disparities in systolic and diastolic blood pressure or aldosterone levels between AGA patients and healthy control. This evidence suggests that AGA may not serve as a reliable clinical predictor for hypertension or hyperaldosteronism.

Our results align with those from similar studies conducted by Danesh-Shakiba et al. [11] in Iran and by Ozbas Gok et al. [12] in Turkey, which also reported no significant association between AGA and health conditions. In contrast, a study from Egypt reported by El Esawy et al. [10], sharing similar objective, and similar study design settings (case-control, male-only participants), indicated a

positive association. These results divergence may stem from regional or genetic variations that were not controlled in the study settings.

Additionally, our findings depart from research such as that by Arias-Santiago et al. [5], and Mansouri et al. [6], which documented higher rates of hypertension among female AGA sufferers across various regions. Such inconsistencies highlight a potential gender-specific prevalence that underscores the influence of other non-AGA-related factors on hypertension and hyperaldosteronism, as suggested by research from Akasaka et al. [13], Connelly et al. [14], and others [15-22]. These studies imply that factors specific to male physiology may diminish the impact of AGA on the investigated conditions.

Further, our analysis considers the role of geographical variability in health outcomes, as discussed by Dummer et al. [23] and Volinn et al. [24], who emphasized how environmental factors and healthcare accessibility influence health status. Studies by Kershaw et al. [25] and Reddy et al. [20] corroborate this, demonstrating that geographic variation affects both the prevalence and management of hypertension.

Given the comprehensive analysis and corroborative findings from diverse studies, it is prudent for healthcare practitioners, particularly in Saudi Arabia and similar regions, to reassess the predictive significance of AGA concerning hypertension and hyperaldosteronism. The lack of a significant association in our study challenges the traditional view of AGA as an indicator of these conditions. Future research should thus broaden its demographic and regional scope within Saudi Arabia to further explore this relationship, emphasizing the need for larger sample sizes to strengthen the reliability of the data.

This nuanced understanding of AGA's diagnostic value, especially in gender-specific and geographic contexts, offers a refined perspective that can influence clinical approaches and health policy decisions, promoting more tailored and effective healthcare strategies.

Conclusion

This study contributes to a growing body of evidence suggesting that androgenetic alopecia should not be considered as a marker for hypertension or hyperaldosteronism. Our findings are particularly relevant to the medical community, indicating that the presence of AGA alone should not lead healthcare providers to anticipate these conditions in patients. Moving forward, research should focus on elucidating the complex interplay of genetic, environmental, and regional factors that influence these associations, which may inform more tailored and effective clinical assessments.

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Consent statement

The study protocol was approved by the Local Ethical Committee of Qassim University with reference number: (23-42-08) and was performed according to the guidelines of Declaration of Helsinki. All participants were informed about the purpose of the study, and written informed consent was obtained from each.

Conflict of interest statement

There are no conflicts of interest.

Data availability statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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Supplementary Files

Untitled.
URL: http://asset.jmir.pub/assets/2aa279b2c31742ab38a9adac889c2851.docx

Figures

Family history of AGA and frequency of participants with AGA.