

ORIGINAL RESEARCH

Association between androgenetic alopecia and metabolic syndrome – A case control study

¹Dr. Nitish Goyal, ²Dr. Asif Ali Khan, ³Dr. Rajendra Devanda, ⁴Dr. Navneet Kaur Randhawa, ⁵Dr. Khushboo Gupta, ⁶Dr. U.S Agarwal

^{1,2}Junior Resident, ^{3,4}Assistant Professor, ⁵Associate Professor, ⁶Professor and Head of Department, Department of Dermatology, Venereology, Leprology, NIMS Medical College, Jaipur, Rajasthan, India

Corresponding author

Dr. Navneet Kaur Randhawa

Assistant Professor, Department of Dermatology, Venereology, Leprology, NIMS Medical College, Jaipur, Rajasthan, India

Email: navneetrandhawa2110@gmail.com

Received Date: 26 June, 2024

Accepted Date: 30 July, 2024

ABSTRACT

Background: Androgenetic alopecia (AGA) is a prevalent condition characterized by gradual hair loss, predominantly affecting males. Epidemiological studies suggest a high prevalence in Caucasian populations, with significant psychosocial impacts. AGA has been associated with various medical disorders, including metabolic syndrome (MetS), which is a cluster of conditions comprising elevated blood pressure, insulin resistance, glucose intolerance, central obesity, and dyslipidemia. This study aims to investigate the association between AGA and MetS at a tertiary care center in North India. **Methods:** We conducted a hospital-based case-control study from July 2022 to September 2023, including 85 males with clinically diagnosed AGA (Group I) and 85 age-matched controls without AGA (Group II). Patients were selected based on the Modified Norwood-Hamilton scale. We collected data on demographic details, lifestyle factors, medical history, measured body mass index (BMI), waist circumference, blood pressure, fasting glucose, insulin levels and lipid profiles. MetS was diagnosed based on the National Cholesterol Education Program Adult Treatment Panel III criteria. **Results:** The AGA group had a significantly higher mean BMI (23.99 ± 2.56 kg/m²) compared to controls (23.10 ± 2.12 kg/m²). Statistically significant differences were observed in waist circumference (86.88 ± 9.55 cm vs. 82.65 ± 7.33 cm), fasting blood sugar (89.23 ± 11.11 mg/dl vs. 84.45 ± 12.26 mg/dl), total cholesterol (191.95 ± 42.71 mg/dl vs. 176.76 ± 34.11 mg/dl), and LDL cholesterol (120.71 ± 32.48 mg/dl vs. 95.06 ± 32.99 mg/dl). Elevated fasting insulin levels and a higher prevalence of MetS (23.52% vs. 7.05%) were also noted in the AGA group. **Conclusions:** Our study indicates a significant association between AGA and MetS. Higher BMI, increased waist circumference, elevated blood glucose, dyslipidemia, and insulin resistance in AGA patients suggest a potential interrelationship between these conditions. The findings underscore the importance of screening AGA patients for MetS and suggest a need for further research to explore shared pathophysiological mechanisms. This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution-Non Commercial-Share Alike 4.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.

INTRODUCTION

AGA (androgenetic alopecia) is the most frequent cause of gradual loss of hair usually seen after puberty in genetically predisposed individuals. The term AGA has been described for both men and women, however it is predominantly a condition affecting males, although medically benign, it poses a significant psychosocial impact on young adults.¹ Epidemiological data reveals that approximately the prevalence rates in Caucasian populations is around 30% for men in their 30s, 40% for men in their 40s and 50% for men in their 50s.² It has a multifactorial aetiology, however primary role of an interplay between hormonal and genetic factors was first described by Hamilton in 1951. The basic

pathology is the gradual conversion of hair follicles of thick terminal type into finer vellus hair due to androgen influence in people who have a genetic predisposition.

AGA has been found to be connected to a number of medical disorders, including smoking, benign prostatic hyperplasia, coronary artery disease, insulin resistance, abnormal lipid profiles, prostate cancer, hypertension, obesity.¹ Atherosclerotic cardiovascular disease is more likely in people with metabolic syndrome (MetS), which refers to a group of interconnected disorders including elevated systolic and/or diastolic blood pressure, resistance to serum insulin, glucose intolerance, central obesity and dyslipidaemia. The risk rises as MetS's component

count rises. Moreover, elevated testosterone levels have been linked to the development of thrombosis and atherosclerosis, which can result in elevated blood pressure and cholesterol.

In this study, we evaluated the association between AGA and metabolic syndrome at a North Indian tertiary care centre to further shed light on the hypothesis of any association between the two individual conditions.

METHODOLOGY

Eighty-five cases (Group I) and eighty-five age-matched controls (Group II) who visited the Dermatology OPD between July 2022 and September 2023 participated in our hospital-based cross-sectional study. Patients aged between 18 and 55 years with clinically diagnosed AGA of grade 3 or more according to Modified Norwood-Hamilton grading were included in group I (i.e. study group) and equal number of age matched patients who had diseases other than androgenetic alopecia were included in group II (i.e. control group). To reduce the impact of compounding factors on the pathophysiology of androgenic alopecia and metabolic syndrome, patients taking hormone replacement treatment with testosterone, corticosteroid medication and diagnosed with psoriasis were removed. Since AGA mostly affects young males and may have different pathophysiology as compared to females and also because androgens may have a debatable role in female pattern hair loss, the study was restricted to men.

The Scientific and Ethical Committee (Proposal no. IEC/P-147/2022) of the Institution gave its time-bound approval for the study to be conducted. Following informed consent, patients underwent a detailed history and clinical examination.

The history included age, area of residence (urban/rural), per capita income, hair fall duration, details of alopecia treatment taken by patient, dietary habits, smoking and alcohol consumption. History for hypertension, diabetes mellitus, dyslipidaemia and any drug prescriptions were obtained. General physical and local cutaneous examination was done. The Modified Norwood Hamilton scale (III–VII) was used to determine the severity of androgenic alopecia. BMI (body mass index), weight, waist circumference and height were all measured. Weight (in kilograms) divided by height (in meters squared) yielded the BMI. The measurement of waist circumference was done at the halfway point of the line starting from the lower border of the last detectable rib to the top of the iliac crest after the conclusion of a typical expiration.

After five minutes of rest, the brachial artery in the right arm was used to measure the systolic and diastolic blood pressure (BP) in a supine posture. The mean value was then recorded. Blood samples were taken between 8 am and 9 am following a 12-hour fast. The fasting insulin level, fasting glucose levels and complete lipid profile were all analysed.

Diagnosis of Metabolic syndrome was made according to National Cholesterol Education Program Adult Treatment Panel III (NCEP: ATP III) criteria (2001) as follows³:

Making a diagnosis requires the presence of three or more of the following:

1. Waist circumferences: > 88 cm for women and > 102 cm for men.
2. Triglycerides in serum 150 mg/dl or higher.
3. The recommended HDL cholesterol levels for men and women are, respectively, less than 40 and 50 mg/dl.
4. A blood pressure of at least 130/85 mmHg.
5. A blood glucose level of 110 mg/dl or more following a fast. The American Diabetes Association (ADA) did, however, change the IFG tolerance standards in 2003, moving them from 110 mg/dl to 100 mg/dl.

The results of observations of individual samples were collected and analysed. Statistical analysis was performed using Statistical Program for Social Sciences (SPSS) software version 20.0 Chicago, Illinois, USA. For categorical variables chi-square test was used for analysis. For rest of the parameters, paired t-test was used for comparison of means.

RESULTS

The study included 85 patients in each group i.e. cases of AGA and control group. The demographic details and descriptive characteristics are given in Table 1.

In our study, the mean body mass index (BMI) in Group I, consisting of patients with androgenetic alopecia (AGA), was higher (23.99 ± 2.56 kg/m²) compared to Group II, the control group, which had a mean BMI of 23.10 ± 2.12 kg/m². Within the AGA group, 27.06% of individuals were classified as overweight, and 3.53% were categorized as obese. In contrast, the control group had 9.41% of individuals classified as overweight and only one individual classified as obese. Statistically, the mean BMI was found to be considerably higher in the AGA group as opposed to the group II (control group).

In the group I, there were 23 smokers (27.06%) compared to 12 smokers (14.12%) in the group II. Statistically the difference was significant, with a p-value of 0.0366.

In addition, there were 29 individuals (34.12%) with a history of alcohol use in Group I, compared to only 9 individuals (10.59%) in Group II. This difference was statistically significant, with a p-value of 0.0002.

Among the cases, the mean waist circumference was 86.88 ± 9.55 cm, whereas in the control group it was 82.65 ± 7.33 cm. The observed difference was statistically significant, with a p-value of 0.001038, indicating a notable discrepancy between the two groups.

In the study group, 41 patients (48.23%) had grade III AGA which was the most common grade attending our OPD followed by grade IV constituting 29.41%.

In the AGA group, the mean fasting blood sugar (FBS) level was 89.23 ± 11.11 mg/dl, whereas in the control group it was 84.45 ± 12.26 mg/dl. The difference between the two groups was statistically significant, with a p-value of 0.0086. Within the AGA group, 19 patients (22.35%) exhibited FBS levels exceeding 100 mg/dl, which is considered within the pre-diabetic range. In contrast, only 4 patients (4.7%) in the control group had FBS levels above 100 mg/dl (see Table 2).

In the cases, the mean total cholesterol level was 191.95 ± 42.71 mg/dl, whereas in the control group it was 176.76 ± 34.11 mg/dl. This difference was statistically significant, with a p-value of 0.0113.

Additionally, the mean low-density lipoprotein (LDL) cholesterol level in the cases was 120.71 ± 32.48

mg/dl, compared to 95.06 ± 32.99 mg/dl in the control group. This difference was also statistically significant, with a p-value of 0.00418.

Among the androgenetic alopecia (AGA) patients, the mean fasting insulin level was 17.60 ± 11.94 mIU/L, compared to 14.23 ± 7.61 mIU/L in the control group. The fasting insulin levels were significantly elevated in the AGA group, indicating a notable difference between the two groups.

In the case group, 23.52% of patients with androgenetic alopecia (AGA) were diagnosed with metabolic syndrome, whereas only 7.05% of the control group had metabolic syndrome. This difference was statistically significant, with a p-value of 0.0028. (Table 1).

Table 1: Baseline characteristics of patients

Parameters	Variables	Group I	Group II	p value*
Age (years)	18-25	36 (42.35%)	40 (47.05%)	0.1708 (NS)
	26-30	28 (32.94%)	25 (29.41%)	
	31-35	12 (14.11%)	15 (17.65%)	
	36-40	5 (5.88%)	4 (4.70%)	
	41-45	3 (3.53%)	1 (1.18%)	
	46-50	1 (1.18%)	0 (0%)	
	Mean age	28 ± 6.28	26.56 ± 7.33	
Area of Residence	Urban	62 (72.94%)	60 (70.59%)	0.32 (NS)
	Rural	23 (27.05%)	25 (29.41%)	
Per capita income (in Rs)	0-200000	21 (24.7%)	17 (20%)	0.6344 (NS)
	200000-500000	49 (57.64%)	55 (64.70%)	
	>500000	15 (17.64%)	13 (15.30%)	
Body-mass index (Kg/m ²)	Normal (<25)	59 (69.41%)	76 (89.41%)	0.0146 (S)
	Overweight ($\geq 25-29.9$)	23 (27.06%)	8 (9.41%)	
	Obese (≥ 30)	3 (3.53%)	1 (1.18%)	
	Mean BMI	23.99 ± 2.56	23.10 ± 2.12	
Smoking	Smoker	23 (27.06%)	12 (14.12%)	0.0366 (S)
	Non-smoker	62 (72.94%)	73 (85.88%)	
Alcoholism	Absent	56 (65.88%)	76 (89.41%)	0.0002 (S)
	Present	29 (34.12%)	9 (10.59%)	
Dietary habits	Vegetarian	58 (68.23%)	45 (52.94%)	0.0414 (S)
	Non-vegetarian	27 (31.76%)	40 (47.06%)	
Metabolic syndrome	Present	20 (23.52%)	6 (7.05%)	0.0028 (S)
	Absent	65 (76.48%)	79 (92.95%)	
Metabolic syndrome parameters	Waist Circumference (cm)	86.88 ± 9.55	82.65 ± 7.33	0.001038 (S)
	Systolic blood pressure (mm Hg)	121.07 ± 6.83	120.51 ± 6.48	0.588 (NS)
	Diastolic Blood Pressure (mm Hg)	80.09 ± 5.98	81.58 ± 3.63	0.0527 (NS)
Alopecia grading (Modified Norwood Hamilton classification)	Grade III	41 (48.23%)	-	-
	Grade IV	25 (29.41%)	-	
	Grade V	16 (18.82%)	-	
	Grade VI	3 (3.53%)	-	

* P value >0.05= Non-significant (NS); and P value ≤ 0.05 = Significant (S)

Table 2: Comparison of biochemical parameters between the groups

Factors	Variables	Group I	Group II	p value*
Metabolic syndrome parameters	Serum Triglycerides(mg/dl)	176.36 ± 83.31	159.26 ± 69.29	0.1476 (NS)
	Serum HDL Cholesterol(mg/dl)	43.18 ± 6.2	48.41 ± 6.86	0.1631 (NS)
	Fasting Blood Sugar (mg/dl)	89.23 ± 11.11	84.45 ± 12.26	0.0086 (S)
	Serum Total Cholesterol(mg/dl)	191.95 ± 42.71	176.76 ± 34.11	0.0113 (S)

	Serum LDL Cholesterol (mg/dl)	120.71±32.48	95.06±32.99	0.00418 (S)
	Serum VLDL Cholesterol (mg/dl)	28.25±15.67	32.08±15.11	0.1071 (NS)
Fasting serum insulin	Normal (2.6 to 25 mIU/L)	64 (75.29%)	71 (83.53%)	0.0296 (S)
	Abnormal (>25 mIU/L)	21 (20.71%)	14 (16.47%)	
	Mean	17.60±11.94	14.23±7.61	

* P value >0.05= Non-significant (NS); and P value ≤0.05= Significant (S)

DISCUSSION

AGA is the leading cause of hair loss among young males, with around 30% for men in their 30s, 40% for men in their 40s and 50% for men in their 50s

The incidence of metabolic syndrome is on the rise worldwide. Atherosclerotic cardiovascular disease is more likely in people with metabolic syndrome (MetS), which refers to a group of interconnected disorders including elevated systolic and/or diastolic blood pressure, resistance to serum insulin, glucose intolerance, central obesity and dyslipidaemia.⁴

Few of the studies done in the past have shown metabolic syndrome to be associated with androgenetic alopecia. But none of them have concluded a one-on-one association between the two. Hence, we undertook this study to further establish whether an association exists between the two or not. Our study concluded a significant link between the two.

In our study, the mean waist circumference was higher amongst the AGA group when compared to controls, which is an indicator of higher abdominal fat which in turn is a risk factor for insulin resistance, hyperinsulinemia, glucose intolerance, dyslipidaemia, hypertension, DM-type 2 and thus posing elevated risk for CVD. Similar findings have been demonstrated by Santiago *et al.* and Gopinath *et al.* in their study.^{5,6}

The mean FBS levels were found to be significantly elevated amongst the cases as compared to controls. Amongst cases, comprising of 85 AGA patients, 19 patients (22.35%) had FBS levels greater than 100mg/dl thereby fulfilling the metabolic syndrome criteria whereas amongst controls only 4 individuals (4.7%) had levels above 100 mg/dl. These findings collaborate with those seen in a study done by Gopinath *et al.*⁶ However, few studies like those by Nabaie *et al.*⁷ and Acibucu *et al.*⁸ contradict such findings.

Our study showed significantly elevated total cholesterol and serum LDL levels amongst cases as compared to controls. These findings are in consistent to those demonstrated by Kim *et al.*⁹ who showed elevated total cholesterol, serum triglycerides and LDL levels amongst AGA patients.

Our study also showed significantly higher fasting serum insulin levels amongst cases when compared to controls. Similar findings have been demonstrated by Bakry *et al.* in their study.¹⁰

In terms of other risk factors, like smoking and alcohol consumption was higher among AGA patients when compared to controls.

Overall, our study concluded that 23.52 % of the patients with AGA had metabolic syndrome while

only 7.05 % of the control group were diagnosed with metabolic syndrome.

Thus, many studies including ours concludes a significant association between metabolic syndrome and AGA. Such an association signifies that there might be some similar pathological pathways involved in the pathogenesis of the two.

Visceral or central adiposity is regarded as the main initiator of the majority of the metabolic syndrome pathways, highlighting the significance of consuming a high caloric intake combined with sedentary lifestyle as major causative factors which are on the rise in today's times.^{4,11} Insulin resistance, chronic inflammation and neurohormonal activation are involved in the initiation, progression, and transition of metabolic syndrome to cardiovascular disease.^{4,12,13,14}

Insulin resistance and hence hyperinsulinemia has been proposed to amplify peripheral testosterone conversion to its active form, dihydrotestosterone (DHT), as well as local androgen synthesis. In addition to increased levels of testosterone, there is also increased peripheral sensitivity of androgen receptors, these factors combined contribute to miniaturization of hair follicles. Obesity and insulin resistance associated microvascular changes is another suggested possible mechanism in the pathogenesis of AGA. These microvascular changes are further explained by increased production of Angiotensin II associated with obesity and insulin resistance.¹⁵ Angiotensin II is already known for its vasoconstrictive properties through its receptors in the heart, kidney, lungs, adrenal glands as well as peripheral blood vessels. Moreover, angiotensin II activates the type 1 receptors which results in the enzyme nicotinamide adenine dinucleotide phosphate oxidase activation, which causes ROS (reactive oxygen species) to be produced.¹⁶ The expression of redox-sensitive transcription factor nuclear factor kappa-light-chain-enhancer of activated B cells (NF-κB), endothelial damage, aggregation of platelets, expression of lectin-like oxidized low-density lipoprotein receptor-1 (LOX-1) on the endothelium and vascular smooth muscle cells, and oxidation of LDL are among the numerous effects that follow.¹⁷ This starts a vicious cycle of damage to endothelium, inflammation and proliferation of fibroblasts that leads to the development of CVD, dyslipidaemia, hypertension, diabetes, and cardiac hypertrophy. All these changes may also be extended to the dermal vessels thereby compromising the blood flow and hence the hair growth.

Also in a recent study done by Ahouansou S et al.¹⁸, suggests a potential link in pathogenesis of AGA and hypertension through the mineralocorticoid receptors. Spironolactone, a mineralocorticoid receptor antagonist, is known for its effects on reducing androgen activity, the predominant known pathogenic pathway involved in AGA. Their study's observation in a double transgenic mouse model, showed the overexpression mineralocorticoid receptors in the skin, which leads to alopecia, supporting the hypothesis of similar possible pathogenetic pathways involved in AGA and metabolic syndrome. Arias-Santiago et al. in their study have also shown that, in comparison to controls, women with early-onset androgenetic alopecia had significantly higher levels of aldosterone and blood pressure.¹⁹

Alarin is a peptide belonging to the family of galanin that exhibits biological activity which is vasoactive in nature, this activity is dose dependant. The main hypothesized mechanisms by which alarin may exert its vasoactive response in the dermis are reduction in flow of blood to dermis and dermal vascular permeability. Its receptors have been found in the microvasculature of dermal papillae.^{20,21,22,23} In line with findings from Zhou et al., Hu et al., and Fang et al., Hamed et al.'s study demonstrated an association between serum cholesterol, TG, LDL, VLDL, fasting blood sugar, waist circumference, weight and BMI in patients with AGA with serum alarin levels.

Thus, the raised alarin levels associated with both AGA and components of metabolic syndrome could be another link between the pathogenesis of the two diseases and their association.

Therefore, the development of metabolic syndrome and androgenetic alopecia (AGA) appears to be interconnected. Various diagnostic criteria for metabolic syndrome may contribute to the onset of AGA, while elevated androgen levels in AGA might influence the development of certain aspects of metabolic syndrome.

AGA can thus be used an indicator for risk for metabolic syndrome, and these patients can be investigated and followed up according to their risk for developing the same.

REFERENCES

- Dharam Kumar KC, Kishan Kumar YH, Neladimmanahally V. Association of Androgenetic Alopecia with Metabolic Syndrome: A Case-control Study on 100 Patients in a Tertiary Care Hospital in South India. *Indian J Endocrinol Metab*. 2018 Mar-Apr;22(2):196-199. doi: 10.4103/ijem.IJEM_650_17. PMID: 29911030; PMCID: PMC5972473.
- Kaliyadan F, Nambiar A, Vijayaraghavan S. Androgenetic alopecia: An update. *Indian J Dermatol Venereol Leprol* 2013;79:613-625
- Gruday et al. (2005). Diagnosis and management of the metabolic syndrome: an American Heart Association/National Heart, Lung and Blood Institute scientific statement. *Circulation* 112, 2735-52.
- Rochlani Y, Pothineni NV, Kovelamudi S, Mehta JL. Metabolic syndrome: pathophysiology, management, and modulation by natural compounds. *Ther Adv Cardiovasc Dis*. 2017 Aug;11(8):215-225. doi: 10.1177/1753944717711379. Epub 2017 Jun 22. PMID: 28639538; PMCID: PMC5933580.
- Arias-Santiago S, Gutiérrez-Salmerón MT, Castellote-Caballero L, Buendía-Eisman A, Naranjo-Sintes R. Androgenetic alopecia and cardiovascular risk factors in men and women: A comparative study. *J Am Acad Dermatol*. 2010;63:420-9.
- Gopinath H, Upadya GM. Metabolic syndrome in androgenetic alopecia. *Indian J Dermatol Venereol Leprol* 2016;82:404-8.
- Nabaie L, Kavand S, Robati RM, Sarrafi-Rad N, Kavand S, Shahgholi L, et al. Androgenic alopecia and insulin resistance: Are they really related? *Clin Exp Dermatol*. 2009;34:694-7.
- Acibucu F, Kayatas M, Candan F. The association of insulin resistance and metabolic syndrome in early androgenetic alopecia. *Singapore Med J*. 2010;51:931-6.
- Kim MW, Shin IS, Yoon HS, Cho S, Park HS. Lipid profile in patients with androgenetic alopecia: a meta-analysis. *J Eur Acad Dermatol Venereol*. 2017 Jun;31(6):942-951. doi: 10.1111/jdv.14000. Epub 2016 Nov 2. PMID: 27717019.
- Bakry OA, Shoeib MA, El Shafiee MK, Hassan A. Androgenetic alopecia, metabolic syndrome, and insulin resistance: Is there any association? A case-control study. *Indian Dermatol Online J*. 2014 Jul;5(3):276-81. doi: 10.4103/2229-5178.137776. PMID: 25165643; PMCID: PMC4144211.
- Matsuzawa Y, Funahashi T, Nakamura T. The concept of metabolic syndrome: contribution of visceral fat accumulation and its molecular mechanism. *J Atheroscler Thromb* 2011; 18(8): 629-639.
- Juhan-Vague I, Alessi MC, Mavri A, et al. Plasminogen activator inhibitor-1, inflammation, obesity, insulin resistance and vascular risk. *J Thromb Haemost* 2003; 1(7): 1575-1579.
- Wallace AM, McMahon AD, Packard CJ, et al.; on behalf of the WOSCOPS Executive Committee. Plasma leptin and the risk of cardiovascular disease in the west of Scotland coronary prevention study (WOSCOPS). *Circulation* 2001; 104(25): 3052-3056.
- Pant S, Deshmukh A, Gurumurthy GS, et al. Inflammation and atherosclerosis – revisited. *J Cardiovasc Pharmacol Ther* 2014; 19(2): 170-178.
- Vanecková I, Maletínská L, Behuliak M, et al. Obesity-related hypertension: possible pathophysiological mechanisms. *J Endocrinol* 2014; 223(3): R63-R78.
- Mehta PK, Griendling KK. Angiotensin II cell signaling: physiological and pathological effects in the cardiovascular system. *Am J Physiol Cell Physiol* 2007; 292(1): C82-C97.
- Gobal F, Deshmukh A, Shah S, et al. Triad of metabolic syndrome, chronic kidney disease, and coronary heart disease with a focus on microalbuminuria: death by overeating. *J Am Coll Cardiol* 2011; 57(23): 2303-2308.
- Ahouansou S, Le Toumelin P, Crickx B, Descamps V. Association of androgenetic alopecia and hypertension. *Eur J Dermatol* 2007; 17:220-2.
- Arias-Santiago S, Gutiérrez-Salmerón MT, Buendía-Eisman A, Girón-Prieto MS, Naranjo-Sintes R. Hypertension and aldosterone levels in women with early-onset androgenetic alopecia. *Br J Dermatol* 2010; 162:786-9.

20. Hamed AM, Fatah MA, Shams GM. Androgenetic Alopecia and Metabolic Syndrome: Is Alarin a Missing Link? *J Clin Aesthet Dermatol*. 2022 Jul;15(7):32-37. PMID: 35942015; PMCID: PMC9345189.
21. Zhou X, Luo M, Zhou S et al. Plasma Alarin Level and Its Influencing Factors in Obese Newly Diagnosed Type 2 Diabetes Patients. *Diabetes Metab Syndr Obes*. 2021;14:379–385.
22. Chew E, Tan J, Bahta A et al. Differential Expression between Human Dermal Papilla Cells from Balding and Non-Balding Scalps Reveals New Candidate Genes for Androgenetic Alopecia. *J Investig Dermatol*. 2016;136(8):1559–1567.
23. Vora RV, Kota R, Singhal RR et al. Clinical profile of androgenic alopecia and its association with cardiovascular risk factors. *Indian J Dermatol*. 2019;64(1):19–22.