Cardiovascular Risk and Systemic Inflammation in Alopecia Areata: An Observational Case-control Study

Alberto Soto-Moreno[1], Jose Muñoz-Baeza[2], Daniel Muñoz-Barba[1], Manuel Sánchez-Díaz[1,3], Salvador Arias-Santiago[1,2]

[1]Servicio de Dermatología. Hospital Universitario Virgen de Las Nieves. Granada, Spain; [2]Departamento de Dermatología, Facultad de Medicina, Universidad de Granada, Granada, Spain; [3]TECe19-Clinical and Translational Dermatology, Instituto de Investigación Biosanitaria de Granada, Ibs. Granada, Spain

Corresponding author:
Manuel Sánchez-Díaz _
Fuerzas Armadas Avenue, number 15. PC: 18012
Granada, Spain_
manolo.94.sanchez@gmail.com

SUMMARY Alopecia areata (AA) is an autoimmune disorder characterized by non-scarring hair loss, often accompanied by systemic manifestations, suggesting a potential link to systemic inflammation. While previous studies have explored the association between AA and cardiovascular risk (CVR), findings remain inconsistent. The main objectives of the study were to analyze cardiovascular risk (CVR) and systemic inflammatory activity in patients with AA compared with healthy controls. Additionally, the study aimed to investigate associations between systemic inflammation/CVR and baseline clinical variables in patients with AA. The study used a case-control design with patients matched for age, sex, and anthropometric characteristics. Measurements of blood pressure, pulse wave velocity, lipid and carbohydrate metabolism parameters, systemic inflammatory markers, and vitamin D levels were conducted. Seventy-two participants were included in the study (36 patients with AA, 36 healthy controls), of whom 72.2% were women (52/72), with a mean age of 39 years (±2.6). The baseline Severity of Alopecia Tool (SALT) values in patients with AA was 42% (±6). Patients with AA had higher systolic blood pressure than controls, with no differences in diastolic blood pressure, pulse wave velocity, or metabolic profile. Patients with AA showed higher systemic inflammation parameters and lower vitamin D levels. No association was observed between CVR and systemic inflammation; these factors were not associated with disease severity, duration, or type of treatment. Despite presenting similar cardiovascular risk profiles to healthy controls, patients with AA demonstrated elevated systemic inflammatory activity. However, these factors did not appear to be interrelated, and were not associated with disease severity. risk profiles to healthy controls, they had elevated systemic inflammatory activity and lower vitamin D levels.

KEY WORDS: alopecia areata, cardiovascular risk, metabolic risk, inflammation

INTRODUCTION

Alopecia areata (AA) is a chronic, recurring autoimmune disorder that leads to non-scarring hair loss (1). While the primary targets are the hair follicles on the scalp, involvement beyond the scalp, including body hair loss and nail dystrophies, is commonly observed in AA (1). Although AA is believed to be

Acta Dermatovenerol Croat
2025;33(1):9-15

an organ-specific autoimmune process where the hair bulb is attacked by cytotoxic T-cells, comorbidities such as atopy, vitiligo, and thyroid disease have been reported, suggesting the potential for systemic inflammation (1,2large-scale observational studies of baseline incidence rates (IRs).

Alongside its association with other autoimmune disorders, there have been several discussions regarding the link between AA and cardiovascular risk (CVR) or metabolic diseases (3with limited studies evaluating the risk of acute myocardial infarction (AMI). However, findings from studies with large sample sizes have yielded inconsistent conclusions, ranging from suggesting a protective role of AA to identifying it as a risk factor, or even showing no significant differences compared with the healthy population (2-4with limited studies evaluating the risk of acute myocardial infarction (AMI).

Biomarkers obtained from blood count parameters reflect the systemic inflammatory state and have been shown to function as risk markers for cardiovascular events, including the neutrophil-to-lymphocyte ratio (5,6), the monocyte-to-HDL ratio (7) and the systemic immune-inflammation index (8), and acute phase reactants (9allocating transcatheter therapy to the adequate candidates and identifying a reliable and validated risk stratification tool for mortality prediction is still lacking. The C-reactive (CRP). Although biomarkers have been employed in assessing cardiovascular risk in AA (10,11), experience in their use remains limited.

Likewise, the role of vitamin D as an immunomodulator has been proposed, as there is evidence linking vitamin D deficiency to inflammatory dermatoses (12pleiotropic role in the maintenance of global homeostasis. Its influence goes far beyond the regulation of calcium and phosphorus balance, as diverse activities of vitamin D and its natural metabolites assure proper functioning of major human organs, including skin. Recently, we reviewed the current understanding of vitamin D impact on human health from historical perspective (Wierzbicka *et al.* (2014). Vitamin D deficiencies have also been linked to increased cardiovascular risk (13), indicating that a systemic inflammatory state may heighten CVR. The association between vitamin D deficiency and AA has been previously documented (1,14).

Pulse wave velocity serves as an estimator of the resistance offered by small peripheral arteries; elevated levels of this marker suggest subclinical atherosclerosis and indicate increased risk of cardiovascular events (15). PWV has been utilized as a marker of cardiovascular risk in inflammatory dermatoses, although the available evidence in AA is limited (16).

PATIENTS AND METHODS

Design

We conducted an observational case-control study that included patients diagnosed with AA, matched with healthy controls based on sex, age, and anthropometric parameters. Additionally, a cross-sectional design was implemented to analyze the association between CVR and systemic inflammatory activity and disease severity within the group of patients with AA. STROBE guidelines were followed in designing the study.

Objectives

The main objectives of this study were: (a) To analyze CVR in patients with AA compared with healthy controls. (b) To analyze systemic inflammatory activity in patients with AA compared with healthy controls. The secondary objectives established were as follows: (c) To analyze the association between systemic inflammatory activity and CVR in patients with AA; and (d) To analyze the association between systemic inflammation/CVR and baseline clinical variables in patients with AA.

The inclusion criteria comprised age over 18 years and a diagnosis of AA.

Exclusion criteria comprised pregnancy or lactation, presence of malignancy, chronic coronary artery disease, or autoimmune diseases requiring systemic treatment, history of acute coronary syndrome, coronary revascularization, heart failure with reduced ejection fraction or cardiovascular pathologies requiring surgery, and chronic kidney disease.

Study procedures

Systolic (SBP) and diastolic (DBP) blood pressure and pulse wave velocity (PWV) were measured using the Mobil-O-Graph-PWA® ambulatory blood pressure monitoring device. All measured values were obtained after a 30-minute relative rest period in a seated position, in the same room, at the same time, and under the same temperature conditions.

Lipid metabolism, carbohydrate metabolism, and systemic inflammatory parameters were determined obtained through blood tests performed on the patients and controls. Blood extraction took place in the same room, at the same time, and ensuring an prior 8-hour fasting period.

Ethics:

The present study was approved by the Research Ethics Committee of Hospital Universitario Virgen de

las Nieves and is in accordance with the Declaration of Helsinki.

Variables of interest

We examined baseline sociodemographic and clinical features including age, sex, height and weight (indicated by body mass index; BMI), smoking status (assessed as smoker/non-smoker and quantified by pack-years index), and diagnosis of metabolic syndrome. Among patients diagnosed with AA, disease severity was assessed using the Severity of Alopecia Tool (SALT), along with the duration of disease progression and current treatment regimen.

We also evaluated the following parameters of endothelial dysfunction: SBP and DBP (mmHg) and PWV (m/s).

The analytical lipid metabolism parameters evaluated in the study were total cholesterol (mg/dL), low-density lipoprotein (LDL) (mg/dL), high-density lipoprotein (HDL) (mg/dL), and triglycerides (mg/dL). The analytical parameters of carbohydrate metabolism evaluated were basal glucose (mg/dL), basal insulin (mg/dL), homeostatic model assessment (HOMA) index, and glycated hemoglobin A1C (HbA1C) (%).

We also examined the following analytical parameters of systemic inflammation: erythrocyte sedimentation rate (ESR) (mg/L) and C-reactive protein (CRP) (mm/h). Additionally, vitamin D (ng/mL) was analyzed as an analyte with an immunomodulatory role. The following ratios reflecting systemic inflammatory activity were also analyzed based on hematologic parameters: neutrophil-to-lymphocyte Ratio (NLR), systemic immune-inflammation index (SII), monocyte-to-HDL Ratio (Monocyte/HDL ratio), and monocyte-to-lymphocyte ratio.

Statistical analysis

Descriptive statistics were used to evaluate the characteristics of the sample. The Shapiro-Wilk test was used to assess the normality of the variables. Continuous variables were expressed as mean and standard deviation (SD). Qualitative variables were expressed as relative and absolute frequency distributions. The chi-square test or Fisher's exact test, as appropriate, were used to compare nominal variables, and the Student's t-test or Wilcoxon-Mann-Whitney test were used for comparison between

Table 1. Descriptive analysis of the sample

	Alopecia areata N= 36	Controls N=36	P
Age	39 (2.6)	39 (2.6)	1.0
Sex • Male • Female	 27.8% (10/36) 72.2% (26/36)	 27.8% (10/36) 72.2% (26/36)	1.0
Body mass index (kg/m^2)	24.1 (0.5)	23 (0.5)	0.1
Metabolic syndrome • Yes • No	 8.3% (3/36) 91.7% (33/36)	 5.6% (2/36) 94.4% (34/36)	1.0
Smoker • Yes • No Pack/years index	 47.2% (17/36) 52.8% (19/36) 11.3 (1.9)	 25% (9/36) 75% (27/36) 7.5 (2)	0.1 0.7
Clinical features Patients with alopecia areata (n=36)			
SALT (%) Severity • Mild (SALT <20) • Moderate (SALT 20-50) • Severe (SALT >50)	41.9 (6) 47% (17/36) 2% (7/36) 33% (12/36)		
Progression time of the disease (years)	9.24 (1.6)		
Current treatments for AA JAK-inh Oral Cs Topical Cs No treatment	 25% (9/36) 33% (12/36) 27% (10/36) 14% (5/36)		

SALT: Severity of Alopecia Tool; Oral Cs: oral corticosteroids; Topical Cs: topical corticosteroids; JAK-inh: systemic Janus kinase inhibitors

nominal and continuous data. To explore possible associated factors, simple linear regression was used for continuous variables. The β coefficient and SD were used to predict the log odds of the dependent variable. Statistical significance was defined as P<0.05. Statistical analyses were performed using JMP version 14.1.0 (SAS institute).

RESULTS

The study included 72 participants (36 with AA, 36 controls), 72.2% of whom were women (52/72), with a mean age of 39 years (±2.6). The baseline SALT value in patients with AA was 42% (±6) (Table 1). No statistically significant differences were identified between groups regarding BMI, metabolic syndrome, and smoking habits.

Parameters of endothelial dysfunction

Systolic blood pressure was higher in patients with AA (124.5 mmHg in AA vs. 118.8 mmHg in controls) (Table 2), and these differences remained close to significance in multivariate analysis, regardless of BMI and smoking status (P=0.07) (Table 3). Diastolic blood pressure was also higher in patients with AA (79.25 mmHg in AA vs. 75.40 mmHg in controls), but these differences did not remain independent of BMI and smoking habits in multivariate analysis. No differences in PWV were identified between patients with AA and healthy controls.

Parameters of lipid and carbohydrate metabolism

No significant differences between the two groups were identified for any lipid metabolism pa-

Table 2. Univariate and multivariate analysis

Variables	Alopecia areata (n=36)	Controls (n=36)	Univariate analysis
	Mean (SD)	Mean (SD)	P
Endothelial dysfunction			
- SBP (mmHg)	- 124.5 (1.7)	- 118.8 (1.8)	0.02
- DBP (mmHg)	- 79.25 (1.2)	- 75.4 (1.3)	0.03
- PWV (m/s)	- 6.75 (0.21)	- 6.3 (0.24)	0.15
Lipid metabolism			
- Total cholesterol (mg/dL)	- 190 (5.3)	- 188.6 (4.7)	0.76
- LDL (mg/dL)	- 116 (6)	- 110.45 (4.5)	0.34
- HDL (mg/dL)	- 59.1 (2.5)	- 63.7 (2.3)	0.19
- Triglycerides (mg/dL)	- 91.5 (6.7)	- 811.6 (6)	0.27
Glucide metabolism			
- Basal glucose (mg/dL)	- 82.5 (1.8)	- 80.2 (1.18)	0.3
- Basal insulin (mg/dL)	- 12.37 (1.2)	- 8 (3.4)	0.02
- HOMA index	- 2.3 (0.2)	- 1.6 (0.13)	0.02
- HbA1C (%)	- 5.2 (0.06)	- 5.17 (0.05)	0.6
Systemic inflammatory activity			
- ESR (mg/L)	- 14.1 (1.7)	- 8.1 (0.75)	<0.01
- CRP (mm/h)	- 10.5 (3)	- 2.8 (0.63)	0.01
- Vitamin D (ng/mL)	- 25.6 (1.7)	- 30.4 (1.6)	0.04
Systemic inflammatory activity (ratios)			
- NLR	- 1.9 (0.12)	- 1.44 (0.14)	0.02
- SII	- 545.6 (54)	- 360.8 (29.2)	<0.01
- Monocyte/HDL ratio	- 0.01 (<0.01)	- 0.008 (<0.01)	0.02
- Monocyte/ lymphocyte ratio	- 0.23 (0.09)	- 0.22 (0.05)	0.4

SBP: systolic blood pressure; DBP: diastolic blood pressure; PWV: pulse wave velocity; LDL: low-density lipoprotein; HDL: high-density lipoprotein; HbA1C: glycated hemoglobin A1C; ESR: erythrocyte sedimentation rate; CRP: C-reactive protein; NLR: neutrophil-to-lymphocyte ratio; SII: systemic immune-inflammation index

rameter. In terms of carbohydrate metabolism and peripheral insulin resistance, the AA group exhibited higher baseline insulin levels (12.37 vs. 8.00 mg/dL) and HOMA index (2.3 vs. 1.6), with these differences being statistically significant (P<0.02 in both univariate analyses) (Table 2). However, this observation lost significance after adjusting the analysis using a multivariate model (Table 3).

Systemic inflammatory activity

Patients with AA exhibited higher serum levels of acute-phase reactants, both ESR (14.1 mg/L in AA vs. 1.7 mg/L in controls, P<0.01) and CRP (10.5 mm/h in AA vs. 2.8 mm/h in controls, P=0.01). However, blood vitamin D levels were lower than those found in healthy controls (25.6 ng/mL in AA vs. 30.4 ng/mL in controls, P=0.04) (Table 2). The differences observed in ESR, CRP, and vitamin D remained independent in the multivariate analysis (P<0.05) (Table 3).

Regarding hematological indices of systemic inflammatory activity, univariate analysis showed significative higher NLR in patients with AA (1.00 in AA vs. 1.44 in controls), as well as higher SII (545.6 in AA vs. 360.8 in controls) and monocyte/HDL ratio (0.010 in AA vs. 0.008 in controls), whereas no differences were identified in the monocyte/lymphocyte ratio (Table 2). These differences did not persist in the subsequent multivariate analysis, which showed that they were mainly dependent on smoking habits (Table 3).

The association between systemic inflammation and cardiovascular risk in patients with AA

Univariate analysis was conducted in the AA group to assess the association between cardiovascular risk variables and systemic inflammation variables that showed significant differences compared with healthy controls. No statistically significant association was found between ESR, CRP, vitamin D, and SBP and DBP (P>0.05 in all analyses).

The association between systemic inflammation and baseline clinical variables in patients with AA

Univariate analysis was performed in the AA group to evaluate the association between systemic inflammation parameters that showed significant differences and baseline clinical characteristics. Acute phase reactants and vitamin D were not significantly associated with any examined clinical variables (baseline SALT, disease severity, current treatment, or years of AA progression). Additionally, there were no differences in PWV, SPB, or DBP that were associated with the clinical characteristics assessed.

DISCUSSION

This case-control study with 72 participants (36 with AA, 36 controls) found higher SBP in patients with AA compared with controls, with differences close to statistical significance in multivariate

Table 3. Multivariate analysis

Multivariate analysis (n=72)	AA/Controls	BMI	Smoking status (pack/years index)
	Beta (SD); p		
Endothelial dysfunction			
- SBP (mmHg)	2.3 (1.3); p= 0.07	1.2 (0.4); p< 0.01	0.04 (0.1); p= 0.8
- DBP (mmHg)	1.52 (0.9); p= 0.12	0.6 (0.3); p= 0.05	-0.02 (0.1); p= 0.8
Glucide metabolism			
- Basal insulin (mg/dl)	1.9 (1); p= 0.07	0.55(0.3); p=0.1	-0.05 (0.1); p= 0.7
- HOMA index	0.29 (0.2); p=0.13	0.07(0.06); p=0.24	<0.01 (<0.01); p= 0.7
Systemic inflammatory activity			
- ESR (mg/l)	2.84 (1); p<0.01	-0.1 (<0.3); p=0.8	0.27 (0.2); p=0.06
- CRP (mm/h)	3.4(1.6); p=0.04	0.9(0.54); p=0.08	-0.03 (0.2); p= 0.88
- Vitamin D (ng/ml)	0.4 (0.15); p<0.01	1.2 (1); p= 0.3	0.42 (0.3); p= 0.3
Systemic inflammatory activity (ratios)			
- NLR	0.13(0.1); p= 0.16	0.02(0.01); p= 0.37	0.05(0.01); p< 0.01
- SII	5.3 (29); p= 0.07	6 (5.3); p= 0.5	3.7 (1.5); p< 0.01
- Monocyte/HDL ratio	6.5(5.3); p= 0.23	3.5 (0.1); p= 0.05	1.9 (<0.01); p< 0.01

AA: alopecia areata; SBP: systolic blood pressure; DBP: diastolic blood pressure; PWV: pulse wave velocity; ESR: erythrocyte sedimentation rate; CRP: C-reactive protein; NLR: neutrophil-to-lymphocyte ratio; SII: systemic Immune-Inflammation Index; HDL: high-density lipoprotein

analysis. While no disparities were detected in DBP or PWV, patients with AA displayed elevated levels of acute-phase reactants and reduced vitamin D levels. However, no significant associations were found between systemic inflammation variables and cardiovascular risk or baseline clinical characteristics in patients with AA. There were no differences in lipid or carbohydrate metabolism between the two groups. The size and baseline characteristics of the sample in our study were similar to those of other studies with the same design (11,16,17).

Patients with AA exhibited elevated SBP compared with healthy controls, with these differences approaching statistical significance in multivariate analysis. A case-control study involving 52 patients with AA revealed a higher prevalence of endothelial dysfunction among in the case group (16). In the same study, consistent with the absence of differences in PWV in our sample, no differences in arterial stiffness were observed (16). An Indian case-control study of 106 patients with AA described higher blood pressure values in patients than in controls, with no significant differences found either (18).

The importance of the differences identified in our study is difficult to predict; when drawing conclusions, it would be advisable to consider the results of studies with larger sample sizes or higher quality evidence: the retrospective cohort studies by Huang *et al.* (1377 patients with AA vs. 4131 controls), Lee *et al.* (3770 patients with AA vs. 18850 controls) and George *et al.* (8784 patients with AA vs. 26352 controls) found no differences in the cumulative incidence of cardiovascular disease in patients with AA (2,3,19with limited studies evaluating the risk of acute myocardial infarction (AMI).

Patients with AA in our study showed no differences in lipid metabolism parameters or peripheral insulin resistance compared with healthy controls. The absence of metabolic alterations was previously described in other case-control studies, where the prevalence of metabolic disorders was almost identical to that of controls (17,18).

Higher systemic inflammatory activity was found in patients with AA in our study. Glickman *et al.* reported increased expression of inflammatory proteins along with proteomic markers of cardiovascular risk in patients with AA, particularly in those with greater disease severity (11). This led to the hypothesis that the severity and duration of AA might correlate with systemic inflammation and subsequent cardiovascular risk (11). However, our study did not find any association between markers of systemic inflammation and other cardiovascular risk factors, disease duration, or severity. Thus, we cannot provide support for this hypothesis based on our findings.

Finally, vitamin D deficiency was more prevalent in patients with AA. This deficit has been previously described in other studies, as reflected in the systematic review and meta-analysis by Lee *et al.* (1). It has been proposed that hypovitaminosis D may play a pathogenic role in AA given the regulatory role it has with regard to inflammation (14).

The present study comprehensively analyzed a wide range of variables and explored their potential associations. However, it is important to acknowledge several limitations. Firstly, the sample size was small, which may limit the generalizability of the findings. Additionally, the case-control design introduces inherent biases, particularly potential selection bias. Furthermore, no follow-up data for the sample were provided, limiting our ability to assess longitudinal outcomes. These limitations should be considered when interpreting the results of the study.

In conclusion, the lack of differences in the metabolic profile, coupled with the absence of any association between inflammation parameters and cardiovascular risk factors, as well as the lack of correlation with disease severity or duration, suggests that cardiovascular risk in patients with AA may be influenced by factors beyond the disease itself.

In conclusion, we found that patients with AA presented a cardiovascular risk similar to that of healthy controls, together with greater systemic inflammatory activity; however, these two factors did not appear to be correlated. Furthermore, cardiovascular risk and systemic inflammation did not show any association with disease severity or duration or with the type of treatment received.

References:

1. Lee S, Lee H, Lee CH, Lee W-S. Comorbidities in alopecia areata: A systematic review and meta-analysis. J Am Acad Dermatol. 2019;80:466-77. e16.
2. George P, Jagun O, Liu Q, Wentworth C, Napatalung L, Wolk R, *et al.* Incidence Rates of Infections, Malignancies, Thromboembolism, and Cardiovascular Events in an Alopecia Areata Cohort from a US Claims Database. Dermatol Ther (Heidelb). 2023;13:1733-46.
3. Huang KP, Joyce CJ, Topaz M, Guo Y, Mostaghimi A. Cardiovascular risk in patients with alopecia areata (AA): A propensity-matched retrospective analysis. J Am Acad Dermatol. 2016;75:151-4.
4. Shin J-W, Kang T, Lee JS, Kang MJ, Huh CH, Kim

MS, *et al.* Time-Dependent Risk of Acute Myocardial Infarction in Patients With Alopecia Areata in Korea. JAMA Dermatology. 2020;156:763-71.

5. Li S, Chen H, Zhou L, Cui H, Liang S, Li H. Neutrophil-to-lymphocyte ratio predicts coronary artery lesion severity and long-term cardiovascular mortality in patients with unstable angina pectoris. Acta Cardiol. 2022;77:708-15.
6. Onalan E, Dönder E. Neutrophil and platelet to lymphocyte ratio in patients with hypothyroid Hashimoto's thyroiditis. Acta Biomed. 2020;91:310-4.
7. Dogan A, Oylumlu M. Increased monocyte-to-HDL cholesterol ratio is related to cardiac syndrome X. Acta Cardiol. 2017;72:516-21.
8. Esenboğa K, Kurtul A, Yamantürk YY, Tan TS, Tutar DE. Systemic immune-inflammation index predicts no-reflow phenomenon after primary percutaneous coronary intervention. Acta Cardiol. 2022;77:59-65.
9. Avci Y, Demir AR, Duran M, Bulut U, Demirci G, Uygur B, *et al.* The prognostic value of C-reactive protein to albumin ratio in patients undergoing transcatheter aortic valve implantation. Acta Cardiol. 2022;77:930-6.
10. Wang EH, Santos L, Li XY, Tran A, Kim SSY, Woo K, *et al.* Alopecia Areata is Associated with Increased Expression of Heart Disease Biomarker Cardiac Troponin I. Acta Derm Venereol. 2018;98:776-82.
11. Glickman JW, Dubin C, Renert-Yuval Y, Dahabreh D, Kimmel GW, Auyeung K, *et al.* Cross-sectional study of blood biomarkers of patients with moderate to severe alopecia areata reveals systemic immune and cardiovascular biomarker dysregulation. J Am Acad Dermatol. 2021;84:370-80.
12. Piotrowska A, Wierzbicka J, Żmijewski MA. Vitamin D in the skin physiology and pathology. Acta Biochim Pol. 2016;63:17-29.
13. Casado Cerrada J, Parra Caballero P, Vega Piris L, Suárez Fernández C. Relation between parathyroid hormone and cardiovascular risk in patients with vitamin D deficiency. Med Clin (Barc). 2013;141:292-4.
14. Lin X, Meng X, Song Z. Vitamin D and alopecia areata: possible roles in pathogenesis and potential implications for therapy. Am J Transl Res. 2019;11:5285-300.
15. Fagundes RR, Vitorino PVO, Lelis E de S, Jardim PCBV, Souza ALL, Jardim TSV, *et al.* Relationship between Pulse Wave Velocity and Cardiovascular Biomarkers in Patients with Risk Factors. Arq Bras Cardiol. 2020;115:1125-32.
16. Waśkiel-Burnat A, Kotowska M, Dorobek W, Smyk JM, Gasecka A, Niemczyk A, *et al.* Patients with alopecia areata are at risk of endothelial dysfunction: results of a case-control study. Clin Exp Dermatol. 2022;47:1517-22.
17. Incel-Uysal P, Akdogan N, Alli N, Oktem A, Candar T, Topcuoglu C, *et al.* Assessment of Metabolic Profile and Ischemia-modified Albumin Level in Patients with Alopecia Areata: A Case-Control Study. Indian J Dermatol. 2019;64:12-8.
18. Singdia H, Bhargava P, Nijhawan S, Mathur DK. A Study of Correlation of Alopecia Areata and Metabolic Syndrome in Northwest Indian Population: A Case-Control Study. Int J Trichology. 2023;15:63-9.
19. Lee H, Kim YC, Choi JW. Alopecia areata is not a risk factor for heart diseases: A 10-year retrospective cohort study. PLoS One. 2021;16:e0250216.