Dermatology Practical & Conceptual

Systemic Inflammatory Biomarkers in Alopecia Areata: The Role of SII, SIRI, and CRP/Albumin Ratio

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ABSTRACT

Introduction: Alopecia areata (AA) is an autoimmune disorder, with potential roles of both genetic and environmental factors. Although AA is a specific autoimmune disease targeting hair follicles, its frequent association with other autoimmune diseases supports the notion that the autoinflammation is not confined to the hair follicles but is systemic. Systemic immune-inflammation index (SII), systemic inflammation response index (SIRI), and CRP (C-reactive protein) albumin ratio (CAR) are elevated in various diseases and correlate with disease severity.

Objectives: The aim of this study was to determine SII, SIRI, and CAR levels in alopecia areata patients and investigate their correlation with disease severity.

Methods: The data of 118 AA patients and 122 healthy controls were analyzed. Complete blood count (CBC), CRP, and albumin values were noted during clinic visits. CAR, SII, and SIRI were calculated in both groups.

Results: Although median SIRI and CAR levels were higher in the AA group, the difference was not statistically significant ($P>0.05$). However, median SII was significantly higher in the AA group ($P<0.001$). AA patients were divided into two groups according to disease severity: mild disease group (MD) and moderate and severe disease (MSD) group. SII, SIRI, and CAR did not differ between the two groups.

Conclusions: In AA patients, SIRI and CAR did not show significant differences compared to the normal population, while SII was significantly higher. SII was not found to be associated with disease severity, suggesting that systemic inflammation may occur even in the mildest cases of the disease.

Introduction

Alopecia areata (AA) is an inflammatory disorder characterized by non-scarring alopecia affecting the scalp and/or body hair. The lifetime risk of developing AA is estimated to be 1.7%. While the exact etiology of the disease remains unclear, it is hypothesized to be an organ-specific autoimmune disorder influenced by both genetic and environmental factors [1]. The autoimmune response, triggered by the loss of immune privilege in hair follicles due to various stimuli, leads to hair loss during the anagen phase of the hair growth cycle [2]. Key contributors for the development of auto-inflammation include autoreactive cytotoxic CD8+ NKG2D+ T lymphocytes and IFN-gamma (IFN-γ). Furthermore, studies have demonstrated an increase in Th1 and Th17 lymphocytes, plasmacytoid dendritic cells, and NK cells in the hair follicles of alopecia areata patients, all of which are involved in the pathogenesis of the disease [3]. Despite being a disease specifically targeting hair follicles, the frequent co-occurrence of AA with other autoimmune conditions, such as autoimmune thyroid disease and celiac disease, suggests that the inflammation is systemic rather than localized to the hair follicles alone [4]. In various diseases, inflammatory markers derived from complete blood count (CBC) have been shown to be elevated and correlate with disease severity [5,6]. The systemic immune-inflammation index (SII) and systemic inflammation response index (SIRI) are indices calculated using platelet and leukocyte subtypes that have gained attention in recent years [6]. These indices have also been found to correlate with disease severity in various chronic inflammatory skin diseases and to decrease with treatment [7,8]. Therefore, they are considered simple and accessible biomarkers for monitoring treatment response [8]. A review of the literature did not reveal any study assessing SII and SIRI levels in alopecia areata. CAR (C-reactive protein to albumin ratio) is another easily calculable inflammatory marker similar to CBC-derived inflammation parameters, and the number of studies investigating CAR has increased in recent years. However, only one study has specifically examined CAR levels in alopecia areata patients [9].

Objectives

In this cross-sectional study, the aims were to determine SII, SIRI, and CAR levels in alopecia areata patients and to investigate their correlation with disease severity.

Methods

Study Design and Participant Population

The study was conducted in accordance with the Helsinki Declaration. Following approval from Institutional Ethics Committee (Approval number: KAEK-14/ 07.02.2023), cross-sectional data were collected between March 2023 and March 2024. A total of 118 patients with AA, including alopecia totalis (AT) and alopecia universalis (AU), as well as 122 healthy controls with no history of alopecia areata were enrolled. Informed consent was obtained from all participants.

Age and sex were recorded for both groups. In the AA group, additional data regarding the age at onset, disease duration, family history of alopecia areata, smoking status, and prior history of infections and medication use were gathered. The type of alopecia areata, affected body areas, presence of nail involvement, and Fitzpatrick skin type were also noted.

Disease severity was assessed using the scoring system defined by Kavak et al. [10]. Patients with three or fewer alopecic patches, each with a widest diameter of 3 cm or less, or disease limited to the eyelashes and eyebrows, were classified as having mild disease. Patients with more than three patches, or a patch with a diameter greater than 3 cm, were classified as moderate disease. Patients with AT or AU were defined as having severe disease. The Severity of Alopecia Tool (SALT) scores were calculated for patients with scalp involvement.

Exclusion criteria included individuals under the age of 18, those with a history of medication affecting CBC and biochemical parameters, individuals with other inflammatory skin diseases, active infections, or with hematologic, rheumatologic, renal, or cardiac conditions.

The control group consisted of healthy volunteers without any medical complaint who had no personal or family history of alopecia areata and who were undergoing laboratory examinations for screening purposes.

Laboratory Analysis

CBC, C-reactive protein (CRP), and albumin values of both the patient and control groups were recorded during their clinic visits. CRP albumin ratio (CAR), systemic immune-inflammatory index (SII), and systemic inflammatory response index (SIRI) were calculated for both groups (SII= neutrophils × platelets / lymphocytes, SIRI = neutrophils × monocytes / lymphocytes) [11].

Statistical Analysis

Data were analyzed using IBM® SPSS® Statistics 25 Software (IBM Corp., Armonk, NY, USA). The Kolmogorov-Smirnov test was applied to assess the normality of the data. For descriptive statistics, mean±standard deviation and median (minimum-maximum) values were used for normally and non-normally distributed data, respectively. The Mann-Whitney U test was used for non-normally distributed variables, while the Student T test was applied for normally distributed variables to compare numeric data. Pearson's

chi-squared test or Fisher's exact test was used to compare categorical data. Spearman correlation test was used to determine the correlation between SALT scores and SII, SIRI, and CAR, A p-value of less than 0.05 was considered statistically significant.

Results

A total of 118 patients with AA and 122 age- and sex-matched healthy controls were included in the study. The median age was 32 (range: 18-67) years for the AA group and 31 (range: 18–86) years for the control group (P=0.89). In the AA group, 48 patients (40.7%) were female and 70 (59.3%) were male, while in control group, 47 individuals (38.5%) were female and 75 (61.5%) were male. No significant difference was observed between the two groups in terms of sex (P=0.73). Mean body mass index (BMI) of the AA patients and control group were 24.15±3.59 kg/m^2 and 24.95 ±3.5 kg/m^2, respectively (P=0.663). There was no significant difference in smoking prevalence between the AA group (55.1%) and the control group (47.6%) (P=0.195). Of the AA patients, 113 had patchy AA, three had patchy alopecia and ophiasis, one had AT, and one had AU. Ninety-three patients (78.8%) had scalp involvement, and 10 (8.5%) patients had nail involvement. Of the AA patents, 50.5% (N=60) had mild disease, 47.5% (N=56) had moderate disease, and 1.7% (N=2) had severe disease. The median duration of AA was two (range: 0.03-246) months, and the median age at onset was 28.5 (range: 2.5–66) years. The median SALT score of the patients with scalp involvement was 4 (1-100). Demographic characteristics of the AA group are summarized in Table 1. SII, SIRI, and CAR levels were compared between the AA and control groups. Although the median SIRI and CAR levels were higher in the AA group, the differences were not statistically significant (P>0.05). However, the median SII was significantly higher in the AA group (P<0.001) (Table 2). AA patients were divided into two groups based on disease severity: mild disease group (MD) or moderate and severe disease (MSD) group, with 60 and 58 patients in MD and MSD groups, respectively. There was no difference between two groups in terms of age, sex, family history of alopecia areata, involved body part, smoking, emotional stress, or infection history (P>0.05). The disease duration was significantly longer in the MSD group compared to the MD group (1.25 years versus 3 years, respectively; P<0.001). No significant difference was found between the two groups in terms of SII, SIRI, or CAR. A comparison between the MD and MSD groups is presented in Table 3. No significant correlation was identified between the SALT scores of 93 patients with scalp involvement and SI, SII, or CAR (p>0.05) (Table 4).

Conclusions

Chronic inflammatory skin diseases negatively impact quality of life and require long-term monitoring and treatment. In the follow-up of these diseases, disease severity indices specific to each condition are used. Additionally, various biomarkers have also begun to be utilized. Moreover, a growing body of research has identified various biomarkers to determine prognosis and to monitor treatment responses in dermatological conditions [12]. SII, SIRI, and CAR are inflammatory markers that have gained increasing attention in recent years [13,14]. These markers have shown utility in assessing disease severity and monitoring treatment outcomes in dermatological conditions such as psoriasis, acne, hidradenitis suppurativa, chronic urticaria, and Behçet's disease [8,15-19]. CRP, a positive acute-phase protein produced by the liver in response to inflammation or tissue damage, is often used as a general marker of inflammation [9]. In autoimmune diseases, inflammation occurs when the immune system targets and attacks the body's own tissues. Increased CRP levels can serve as an indicator of an inflammatory response within the body and are commonly used as a general marker of inflammation. [20]. Several studies have reported higher CRP and high-sensitivity CRP levels in alopecia areata (AA) patients compared to healthy controls [20-24]. In a meta-analysis conducted to identify potential biomarkers for alopecia areata, 91 studies and 52 biomarkers were analyzed. The results indicated that serum IL-6 and CRP levels were significantly higher and vitamin D levels were significantly lower in alopecia areata patients compared to controls [25]. CAR, a novel biomarker indicating systemic inflammation, has been shown to predict disease prognosis more effectively than CRP alone [26]. A study by Kemeriz et al. on psoriasis patients found that CAR was significantly elevated and was the most strongly associated marker with psoriasis severity among several other inflammatory markers [27]. In our PubMed search, we identified only one current study on CAR in AA. Kalaycı et al. (2022) analyzed CAR levels in 65 AA patients and 65 controls. They found significantly higher CAR in the AA group and a positive correlation between CAR and disease severity. The optimal CAR value for severe AA was calculated to be 0.38 [9]. However, in our study, while CAR was slightly higher in the patient group, this difference was not statistically significant. In recent years, CBC-derived biomarkers have been extensively studied in various diseases, including AA, with mixed results. Saraç et al. found significantly higher levels of hemoglobin, monocytes, platelets, monocyte/high-density lipoprotein cholesterol (HDL-C) ratio (MHR), monocyte/lymphocyte ratio (MLR), and platelet/lymphocyte ratio (PLR) in AA patients compared to controls [28]. In contrast, Dere et al. found no significant difference in PLR, neutrophil/lymphocyte ratio

Table 1. Demographic Characteristics of AA Patients

Median age-year (minimum- maximum)		32 (18-67)
Sex N (%)	Female	48 (40.7%)
	Male	70 (59.3%)
Alopecia type N (%)	Patchy	113 (95.8%)
	Patchy + ophiasis	3 (2.5%)
	AU+ AT	2 (1.7%)
Family history- n(%)		28 (23.7%)
Disease severity- N (%)	Mild	60 (50.8%)
	Moderate	56 (47.5%)
	Severe	2 (1.7%)
Median disease duration –month (minimum – maximum)		2 (0.03-246)
Mean age of onset - year ±standard deviation		29.2±11.4
Median number of disease episodes (minimum-maximum)		1 (1-14)
Localization – N (%)	Scalp	93 (78.8%)
	Beard	30 (25.4%)
	Mustache	5 (4.2%)
	Eyebrows	4 (3.4%)
	Eyelashes	3 (2.5%)
	Body hair	6 (5.1%)
Median SALT scores of patients with scalp involvement (minimum-maximum)		4 (1-10)
Degree of SALT score –N (%)	S1	83 (89.2)
	S2	4 (3.2)
	S3	4 (4.3)
	S4a	2 (2.2)
	S4b	1 (1.1)
Nail involvement- N (%)		10 (8.5%)
Nail findings- N (%)	Beau's lines+ Median canaliform nail dystrophy	1 (0.8%)
	Leukonychia	1 (0.8%)
	Pitting+ trachyonychia	1 (0.8%)
	Longitudinal ridging	2 (1.7%)
	Pitting	2 (1.7%)
	Trachyonychia	3 (2.5%)
Fitzpatrick skin type- N (%)	2	23 (19.5%)
	3	84 (71.5%)
	4	11 (9.3%)
Smoking- N (%)		65 (55.1%)
Emotional stress –N (%)		93 (78.8%)
Infection- N (%)		9 (%7,6)
Co morbidity- N (%)	Thyroid disease	8 (6.8%)
	Familial Mediterranean fever	1 (0.8%)
	Urticaria	1 (0.8%)
	Hypertension	4 (3.4%)
	Irritable bowel disease	1 (0.8%)

Abbreviations: AA: Alopecia areata; AU: Alopecia universalis; AT: Alopecia totalis; SALT: Severity of Alopecia Tool

(NLR), and mean platelet volume (MPV) levels between AA patients and the normal population [29]. Similarly, İslamoğlu et al. observed comparable red blood cell distribution width (RDW), MPV, plateletcrit (PCT), NLR, and PLR levels between AA patients and controls [22]. Upon reviewing the literature, it was noted that the levels of SII and SIRI, which are also calculated using CBC parameters and which have gained popularity recently, have not previously been examined in AA patients. SII is calculated as platelet count x neutrophil count / lymphocyte count. While NLR also reflect inflammatory states, SII accounts for the interaction between inflammation and thrombosis by incorporating platelet changes, which may provide a more comprehensive view of systemic inflammation. SIRI refers to neutrophil count x monocyte count / lymphocyte count and reveals the balance between inflammatory and anti-inflammatory responses. Both SII and SIRI are thought to be closely associated with the development of autoimmune diseases and tumors [30]. SII has also been shown to be associated with subclinical inflammation [31]. In our study, SII was significantly higher

Table 2. Comparison of The Inflammatory Indexes Between AA and Control Groups

Median (minimum- maximum)	AA group N=118	Control group N=122	*p*
SII	804.49 (342.7-1881.64)	501.29 (149.44-1546.61)	***<0.001****
SIRI	0.86 (0.26-2.60)	0.79 (0.25-4.57)	0.274 *
CRP/albumin ratio	0.178 (0.002-7.674)	0.172 (0.034-1.235)	0.372 *

Abbreviations: *Mann-Whitney U test. AA: Alopecia areata; CRP: C-reactive protein; SII: Systemic immune-inflammatory index; SIRI: Systemic inflammatory response index

Table 3. Comparison of MD and MSD Groups

		MD group N=60	MSD group N=58	*p*
Median age-year (minimum- maximum)		29.5 (18-67)	33.5 (18-53)	0.129*
Sex	Female	21 (35%)	27 (46.6%)	0.202**
	Male	39 (65%)	31 (53.4%)	
Median disease duration –month (minimum – maximum)		1.25 (0.03-40.0)	3.0 (0.1-246)	***<0.01****
Median age of onset (minimum- maximum)		27 (3-66)	29 (2,5-52)	0.588*
Family history of AA		16 (27.6%)	12 (20.7%)	0.445**
Fitzpatrick skin type- N (%)	2	15 (25%)	8 (13.8%)	0.087*
	3	41 (68.3%)	43 (74.1%)	
	4	4 (6.7%)	7(12.1%)	
Localization – N (%)	Scalp	48 (80%)	45 (77.6%)	0.748 **
	Beard	12 (20%)	18 (31%)	0.169 **
	Mustache	1 (1.7%)	4 (6.9%)	0.203 ***
	Eyebrows	1 (1.7%)	3(5.2%)	0.360 ***
	Eyelashes	0	3 (5.2%)	0.116 ***
	Body hair	1 (1.7%)	5 (8.6%)	0.111 ***
	Nail	6 (10%)	4 (6.9%)	0.74 ***
Smoking – N (%)		37 (61.7%)	28 (48.3%)	0.144 **
Emotional stress –N (%)		47 (78.3%)	46 (79.3%)	0.897 **
Infection- N (%)		4 (6.7%)	5 (8.6%)	0.741 ***
Median SIRI (minimum- maximum)		0.847 (0.290-2.137)	0.892 (0.263-2.605)	0.929 *
Median SII (minimum- maximum)		832.88 (342.27-1607.64)	748.68 (448.04-1881.64)	0.329 *
Median CRP/ albumin ratio (minimum- maximum)		0.178 (0.002-3.18)	0.180 (0.019-7.67)	0.708 *

Abbreviations: *Mann-Whitney U test; **Chi-squared test; *** Fisher's exact test; CRP: C-reactive protein; MD group: Mild disease group; MSD group: Moderate-severe disease group; SII: Systemic immune-inflammatory index; SIRI: Systemic inflammatory response index

Table 4. Correlation Analysis between SALT Score and SI, SIRI, and CAR in AA Patients with Scalp Involvement

	N=93	SALT	CAR	SIRI	SII
SALT	r	1.000	0.009	0.112	0.120
	p	.	0.932	0.285	0.254
CAR	r	0.009	1.000	-0.027	0.029
	p	0.932	.	0.798	0.785
SIRI	r	0.112	-0.027	1.000	0.573**
	p	0.285	0.798	.	0.000
SII	r	0.120	0.029	0.573**	1.000
	p	0.254	0.785	0.000	.

Abbreviations: CAR: C-reactive protein / albumin ratio; SALT: Severity of Alopecia tool; SII: Systemic immune-inflammatory index; SIRI: Systemic inflammatory response index

in the AA group compared to controls, while SIRI did not show a significant difference between the two groups. However, no association was found between disease severity and SII levels. These findings suggest that systemic inflammation may be present in AA, even in mild cases, without a direct correlation with disease severity.

Limitations

Our study has several limitations. Firstly, it is a single-center study with a relatively small sample size. Additionally, we did not use the SALT score for severity assessment due to the presence of isolated mustache and beard involvement without scalp involvement among our study population. The insufficient number of patients with alopecia totalis (AT) and alopecia universalis (AU) is another limitation.

In conclusion, while SIRI and CAR did not show any significant differences between AA patients and controls, SII was significantly higher in AA patients. SII was not associated with disease severity, suggesting that systemic inflammation may occur even in mild cases of the disease.

References

1. Gönül M, Gül Ü, Piskin E, et al. Alopesi Areatali Hastalarin Geriye Dönük Degerlendirilmesi/Retrospective Evaluation of Alopecia Areata Patients. *Turk Dermatoloji Dergisi*. 2011;5(2): 43-47. DOI: 10.5152/tdd.2011.10.
2. Fukuyama M, Ito T, Ohyama M. Alopecia areata: Current understanding of the pathophysiology and update on therapeutic approaches, featuring the Japanese Dermatological Association guidelines. *J Dermatol*. 2022;49(1):19-36. DOI: 10.1111/1346-8138.16207. PMID: 34709679.
3. Šutić Udović I, Hlača N, Massari LP, Brajac I, Kaštelan M, Vičić M. Deciphering the Complex Immunopathogenesis of Alopecia Areata. *Int J Mol Sci*. 2024;25(11). DOI: 10.3390/ijms25115652. PMID: 38891839.
4. Simakou T, Butcher JP, Reid S, Henriquez FL. Alopecia areata: A multifactorial autoimmune condition. *J Autoimmun*. 2019;98: 74-85. DOI: 10.1016/j.jaut.2018.12.001. PMID: 30558963.
5. Shi C, Cao H, Zeng G, Yang L, Wang Y. The relationship between complete blood cell count-derived inflammatory biomarkers and benign prostatic hyperplasia in middle-aged and elderly individuals in the United States: Evidence from NHANES 2001-2008. *PLoS One*. 2024;19(7):e0306860. DOI: 10.1371/journal.pone.0306860. PMID: 38980876.
6. Ma R, Cui L, Cai J, Yang N, Wang Y, Chen Q, et al. Association between systemic immune inflammation index, systemic inflammation response index and adult psoriasis: evidence from NHANES. *Front Immunol*. 2024;15:1323174. DOI: 10.3389/fimmu.2024.1323174. PMID: 38415255.
7. Tamer F, Edek YC, Aksakal AB. Effect of Treatment With Biologic Agents on the Novel Inflammatory Biomarkers Systemic Immune Inflammation Index and Systemic Inflammation Response Index for Psoriasis. *Dermatol Pract Concept*. 2024;14(1). DOI: 10.5826/dpc.1401a65. PMID: 38364414.
8. Kulakli S, Oguz ID, Aksan B. Could Blood Cell-Based Inflammatory Markers Be Used to Monitor Response to Biologic Therapy in Psoriasis? *Sisli Etfal Hastan Tip Bul*. 2023;57(4):536-542. DOI: 10.14744/semb.2023.43569. PMID: 38268655.
9. Kalaycı BN, Balta İ. Evaluation of the serum C-reactive protein-albumin ratio and its relationship with disease severity IN alopecia areata: A prospective case-control study. *J Cosmet Dermatol*. 2022;21(12):7194-7199. DOI: 10.1111/jocd.15460. PMID: 36225083.
10. Kavak A, Baykal C, Ozarmağan G, Akar U. HLA in alopecia areata. *Int J Dermatol*. 2000;39(8):589-592. DOI: 10.1046/j.1365-4362.2000.00921.x. PMID: 10971726.
11. Zeng X, Liu G, Pan Y, Li Y. Development and validation of immune inflammation-based index for predicting the clinical outcome in patients with nasopharyngeal carcinoma. *J Cell Mol Med*. 2020;24(15):8326-8349. DOI: 10.1111/jcmm.15097. PMID: 32603520.
12. Tan IJ, Podwojniak A, Parikh A, Cohen BA. Precision Dermatology: A Review of Molecular Biomarkers and Personalized Therapies. *Curr Issues Mol Biol*. 2024;46(4):2975-2990. DOI: 10.3390/cimb46040186. PMID: 38666916.
13. Gürbüzer N, Özcan Tozoğlu E. Inflammation, Immunonutritive, and Cardiovascular Risk Biomarkers in Men With Alcohol Use Disorder. *Cureus*. 2024;16(5):e59522. DOI: 10.7759/cureus.59522. PMID: 38826899
14. Çakin Ö, Karaveli A, Yüce Aktepe M, Gümüş A, Yildirim Ö E. Comparison of Inflammatory Marker Scoring Systems and Conventional Inflammatory Markers in Patients over 65 Years of Age Admitted to the Intensive Care Unit: A Multicenter, Retrospective, Cohort Study. *J Clin Med*. 2024;13(14). DOI: 10.3390/jcm13144011. PMID: 39064051.
15. Morariu SH, Cotoi OS, Tiucă OM, Baican A, Gheucă-Solovăstru L, Decean H, et al. Blood-Count-Derived Inflammatory Markers as Predictors of Response to Biologics and Small-Molecule Inhibitors in Psoriasis: A Multicenter Study. *J Clin Med*. 2024;13(14). DOI: 10.3390/jcm13143992. PMID: 39064032.
16. Utlu Z. Evaluation of systemic immune and inflammatory biomarkers in hidradenitis suppurativa. *Eur Rev Med Pharmacol Sci*. 2023;27(19):9267-9272. DOI: 10.26355/eurrev_202310_33954. PMID: 37843340.

17. Cosansu NC, Yuksekal G, Turan U, Umitfer F, Yaldiz M, Sevimli Dikicier B. Investigation of systemic immune-inflammation index and systemic inflammation response index as an indicator of the anti-inflammatuary effect of isotretinoin in patients with acne vulgaris. *Cutan Ocul Toxicol*. 2022;41(2):174-178. DOI: 10.1080/15569527.2022.2081700. PMID: 35658795.
18. Bakay OSK, Kacar N, Gul B, Goksin S, Gural Y. What can be learned from real-world data about chronic spontaneous urticaria? *Allergy Asthma Proc*. 2024;45(4):255-261. DOI: 10.2500/aap.2024.45.240041. PMID: 38982597.
19. Pala E, Bayraktar M. Relationship between C-reactive protein/albumin ratio and mucocutaneous symptom frequency and disease severity in Behçet's disease. *Turk J Med Sci*. 2024;54(2):384-390. DOI: 10.55730/1300-0144.5803. PMID: 39050391.
20. AbdElneam AI, Al-Dhubaibi MS, Bahaj SS, Mohammed GF, Alantry AK, Atef LM. C-reactive protein as a novel biomarker for vitamin D deficiency in alopecia areata. *Skin Res Technol*. 2024;30(3):e13657. DOI: 10.1111/srt.13657. PMID: 38528743.
21. Mokhtari F, Ganjei Z, Yazdanpanah M, Hosseini M. Inverse correlation between vitamin D and CRP levels in alopecia areata: A pilot study. *J Cosmet Dermatol*. 2023;22(11):3176-3180. DOI: 10.1111/jocd.15994. PMID: 37674473.
22. İslamoğlu ZGK, Demirbaş A. Evaluation of complete blood cell and inflammatory parameters in patients with alopecia areata: Their association with disease severity. *J Cosmet Dermatol*. 2020;19(5):1239-1245. DOI: 10.1111/jocd.13131. PMID: 31502748.
23. Gade VKV, Mony A, Munisamy M, Chandrashekar L, Rajappa M. An investigation of vitamin D status in alopecia areata. *Clin Exp Med*. 2018;18(4):577-584. DOI: 10.1007/s10238-018-0511-8. PMID: 29869122.
24. Yousefi M, Namazi MR, Rahimi H, Younespour S, Ehsani AH, Shakoei S. Evaluation of Serum Homocysteine, High-Sensitivity CRP, and RBC Folate in Patients with Alopecia Areata. *Indian J Dermatol*. 2014;59(6):630. DOI: 10.4103/0019-5154.143567. PMID: 25484412.
25. Zaaroura H, Gilding AJ, Sibbald C. Biomarkers in alopecia Areata: A systematic review and meta-analysis. *Autoimmun Rev*. 2023;22(7):103339. DOI: 10.1016/j.autrev.2023.103339. PMID: 37087083.
26. Ranzani OT, Zampieri FG, Forte DN, Azevedo LC, Park M. C-reactive protein/albumin ratio predicts 90-day mortality of septic patients. *PLoS One*. 2013;8(3):e59321. DOI: 10.1371/journal.pone.0059321. PMID: 23555017.
27. Kemeriz F, Tuğrul B, Tuncer SÇ. C-reactive protein to albumin ratio: Is a new parameter for the disease severity in patients with psoriasis vulgaris? *Dermatologica Sinica*. 2020;38(4):199-204. DOI: 10.4103/ds.ds_42_20.
28. Aksoy Sarac G, Acar O, Nayır T, Hararcı Yıldırım P, Dinçer Rota D. The Use of New Hematological Markers in the Diagnosis of Alopecia Areata. *Dermatol Pract Concept*. 2023;13(2). DOI: 10.5826/dpc.1302a118. PMID: 37196257.
29. Dere G, Gündoğdu M. Investigation of the relationship between alopecia areata and inflammatory blood parameters. *J Cosmet Dermatol*. 2021;20(12):4048-4051. DOI: 10.1111/jocd.14048. PMID: 33682292.
30. Wang C, Yan W, Ren M, Zhong L. Screening significance of systemic immune-inflammation index (SII) and systemic inflammation response index (SIRI) in coronary heart disease of symptomatic youth. *Immun Inflamm Dis*. 2024;12(8):e1369. DOI: 10.1002/iid3.1369. PMID: 39110067.
31. Ustundag Y, Huysal K, Gecgel SK, Unal D. Relationship between C-reactive protein, systemic immune-inflammation index, and routine hemogram-related inflammatory markers in low-grade inflammation. *Int J Med Biochem*. 2018;1(1):24-28. DOI: 10.14744/ijmb.2017.08108.