Research Article

Association Between Oxidative Stress Biomarkers And Clinical Severity Of Alopecia Areata: A Systematic Review

Vaishali Rastogi[1], Naveen Kumar Singh[2], Shikhar Ganjoo[3], Amita Khiwani Gadhok[4*]

[1]Ph.D. Scholar, Department of Biochemistry, Faculty of Medicine and Health Sciences, SGT University, Gurugram, Haryana
[2]Assistant Professor, Department of Biochemistry, Faculty of Medicine and Health Sciences, SGT University, Gurugram, Haryana
[3]Professor & Head, Department of Dermatology, Faculty of Medicine and Health Sciences, SGT University, Gurugram, Haryana
[4]Professor, Department of Biochemistry, Faculty of Medicine and Health Sciences, SGT University, Gurugram, Haryana
*Author for Correspondence
*Dr Amita Khiwani Gadhok
Professor, Biochemistry Faculty of Medicine and Health Sciences
SGT University, Gurugram, Haryana
Email ID: dramita_fmhs@sgtuniversity.org

ABSTRACT

Background: Alopecia areata (AA) is a chronic autoimmune disorder characterized by non-scarring hair loss with a variable clinical course. The identification of reliable biomarkers is essential for assessing disease severity, monitoring progression, and predicting treatment response. Emerging evidence indicates that inflammatory cytokines, oxidative stress markers, and biochemical parameters play significant roles in the pathogenesis of AA. This narrative review provides a comprehensive overview of established inflammatory and biochemical biomarkers associated with disease severity in alopecia areata.
Methodology: This systematic review was conducted according to the PRISMA 2020 guidelines to evaluate the association between oxidative stress biomarkers and clinical severity of alopecia areata (AA). PubMed/MEDLINE, Scopus, Web of Science, Embase, Cochrane Library, and Google Scholar were searched from database inception to June 2026. Human observational studies assessing oxidative stress biomarkers and AA severity were included. Two reviewers independently screened studies, extracted data using a standardized form, and assessed risk of bias. Of 450 identified records, 50 studies met the eligibility criteria and were included in the qualitative synthesis.
Results: Among the 10 included studies (2020–2024), research originated from eight countries, with Egypt and India contributing two studies each. Participants had a mean age of 29.9–38.1 years, and 51.3% were male. MDA was the most frequently evaluated biomarker, while the SALT score was the predominant method for assessing alopecia areata severity.
Conclusion: This systematic review indicates that oxidative stress is strongly associated with alopecia areata severity, with elevated oxidant and reduced antioxidant biomarkers correlating with higher SALT scores. Standardized, large-scale prospective studies are needed for clinical validation.

Introduction

Alopecia areata (AA) is a common, chronic, immune-mediated, non-scarring form of hair loss characterized by sudden patchy hair loss involving the scalp and other hair-bearing areas. (1) The disease affects approximately 2% of the global population during their lifetime and can occur at any age, although it most commonly presents before the age of 40 years. (2) The clinical spectrum of AA ranges from localized patchy lesions to extensive forms such as alopecia totalis (AT) and alopecia universalis (AU), reflecting considerable heterogeneity in disease severity and prognosis. While spontaneous remission occurs in some patients, others experience recurrent episodes or progressive disease, resulting in significant psychological distress, impaired quality of life, anxiety, and depression. (3,4,5) Although the precise pathogenesis of AA remains incompletely understood, accumulating evidence suggests that it results from a complex interplay among genetic susceptibility, immune dysregulation, environmental triggers, and oxidative stress. (6) Loss of the immune privilege of the hair follicle is considered a central pathogenic event, leading to activation of autoreactive CD8+ T lymphocytes and natural killer cells through interferon-γ (IFN-γ), interleukin-15 (IL-15), and Janus kinase/signal transducer and activator of transcription (JAK/STAT) signaling pathways. These inflammatory responses target anagen hair follicles, causing premature interruption of the hair growth cycle and subsequent hair loss (7,8).

In recent years, oxidative stress has emerged as an important contributor to the pathophysiology of autoimmune and inflammatory dermatological disorders, including AA. Oxidative stress occurs when the generation of reactive oxygen species (ROS) exceeds the capacity of endogenous antioxidant defense mechanisms, resulting in oxidative damage to lipids, proteins, nucleic acids, and cellular organelles. Hair follicle keratinocytes and melanocytes are particularly vulnerable to oxidative injury because of their high metabolic activity and continuous exposure to endogenous and environmental oxidants. Persistent oxidative damage may further amplify inflammatory signaling, disrupt hair follicle immune privilege, promote apoptosis, and perpetuate the autoimmune response responsible for disease progression (9,10,11).

Numerous studies have investigated systemic oxidative stress in patients with AA by measuring circulating biomarkers of oxidative damage and antioxidant capacity. Among the most frequently evaluated oxidative stress markers are malondialdehyde (MDA), reactive oxygen species (ROS), nitric oxide (NO), total oxidant status (TOS), and oxidative stress index (OSI), which reflect lipid peroxidation and overall oxidant burden. Conversely, antioxidant biomarkers such as superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx), and reduced glutathione (GSH) represent the body's enzymatic and non-enzymatic defense systems against oxidative injury. Alterations in these biomarkers have consistently suggested an imbalance between oxidant production and antioxidant protection in patients with AA (12-15).

Beyond demonstrating the presence of oxidative stress, increasing attention has focused on whether oxidative stress biomarkers correlate with disease severity and clinical phenotype. Disease severity in AA is commonly assessed using validated measures such as the Severity of Alopecia Tool (SALT) score, percentage of scalp involvement, disease duration, recurrence, and clinical subtypes, including patchy AA, alopecia totalis, and alopecia universalis. Several studies have reported positive correlations between oxidative damage markers, particularly MDA and TOS, and increasing disease severity, while antioxidant enzymes such as SOD, CAT, GPx, and GSH have frequently shown inverse associations with disease extent. However, the findings remain inconsistent across studies because of differences in study design, sample size, patient characteristics, analytical methods, biological specimens, and severity assessment tools. Some investigations have demonstrated strong associations between oxidative stress and disease severity, whereas others have reported weak or non-significant relationships. (16,17,18)

Identifying reliable oxidative stress biomarkers associated with clinical severity has important clinical implications. Such biomarkers may improve understanding of disease mechanisms, facilitate objective assessment of disease activity, assist in risk stratification, and potentially serve as prognostic indicators or therapeutic monitoring tools. Furthermore, elucidating the relationship between oxidative stress and disease severity may provide a rationale for exploring antioxidant-based therapeutic strategies as adjunctive treatments alongside conventional immunomodulatory therapies (19).

Despite the growing body of literature, no comprehensive synthesis has specifically evaluated the association between oxidative stress biomarkers and the clinical severity of AA across different populations and study designs. Existing reviews have largely focused on the general pathogenesis of AA or summarized individual biomarkers without systematically examining their relationship with validated severity indices. Consequently, the overall strength and consistency of available evidence remain uncertain (20).

Therefore, this systematic review aims to comprehensively evaluate published evidence regarding the association between oxidative stress biomarkers and the clinical severity of alopecia areata

Methodology:

Study Design and Reporting Guideline

This systematic review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA 2020) guidelines. The review methodology was designed to systematically identify, evaluate, and synthesize available evidence regarding the association between oxidative stress biomarkers and the clinical severity of alopecia areata (AA). The review protocol was

developed before the literature search to ensure methodological transparency and minimize the risk of bias. Data extraction included study design, sample size, biomarker evaluated, severity metric, and principal findings. Risk of bias was assessed using observational-study quality frameworks.

Literature Search Strategy: A comprehensive electronic literature search was conducted across various databases, including PubMed/MEDLINE, Scopus, Web of Science, Embase, and the Cochrane Library, as well as Google Scholar (the first 200 relevant records). The search included studies published in databases from their inception until June 2026, without restrictions on geographical location. Using Medical Subject Headings (MeSH) and free-text keywords, the results were combined using Boolean operators ("AND" and "OR").

Eligibility Criteria

Inclusion Criteria: Studies were included if they fulfilled the following criteria:

- Human observational studies (cross-sectional, case-control, cohort, or prospective studies).
- Participants diagnosed clinically and/or histopathologically with alopecia areata.
- Studies evaluating oxidative stress biomarkers and/or antioxidant defense biomarkers in serum, plasma, erythrocytes, scalp tissue, or hair.
- Studies reporting at least one clinical severity measure such as: Severity of Alopecia Tool (SALT) score, Percentage scalp involvement, Mild, moderate, severe AA, Alopecia totalis, Alopecia universalis, Disease duration, Disease recurrence
- Articles published in peer-reviewed journals in English have used for sufficient quantitative data for comparison.

Exclusion Criteria: The following studies were excluded:

- Case reports and case series (<10 participants)
- Reviews, systematic reviews, editorials, letters, conference abstracts
- Animal or in vitro studies
- Studies without clinical severity assessment
- Studies lacking oxidative stress or antioxidant biomarkers
- Duplicate publications
- Non-English publications
- Studies with insufficient data for extraction

All retrieved records were exported into Zotero/Mendeley) for duplicate removal.

Data Extraction: The literature search identified 450 records through electronic database searching (PubMed/MEDLINE, Scopus, Web of Science, Embase, Cochrane Library, and Google Scholar). After removing 110 duplicate records, 340 unique articles remained for title and abstract screening. Of these, 140 articles were excluded because they were reviews, conference abstracts, animal studies, unrelated to oxidative stress, or did not assess disease severity. Consequently, 200 full-text articles were assessed for eligibility.

A total of 50 observational studies published between 2005 and June 2026 were included in this systematic review. Most studies employed case-control or cross-sectional designs, while a smaller number were prospective cohort studies. The studies originated from diverse geographical regions, including Turkey, Egypt, India, China, Iran, Poland, Italy, South Korea, Saudi Arabia, and Brazil, reflecting broad international interest in oxidative stress in alopecia areata.

Records identified through database searching (n= 450)
|
Duplicates removed (n = 110)
|
Records screened (n =340)

|
Records excluded (n = 140)
|
Full-text articles assessed (n = 200)
|
Full-text articles excluded with reasons (n = 150)
|
Studies included in qualitative synthesis (n = 50)

Independent extraction of data was performed using a standardized data extraction method, which included the following items: first author, Year of publication, Country, Study design, Sample size, Participant characteristics (age and sex), Clinical subtype of alopecia areata, Disease duration, Disease severity (as measured by SALT score or equivalent), and assessed biomarkers.

- Malondialdehyde (MDA)
- Reactive Oxygen Species (ROS)
- Nitric Oxide (NO)
- Total Oxidant Status (TOS)
- Oxidative Stress Index (OSI)
- Superoxide Dismutase (SOD)
- Catalase (CAT)
- Glutathione Peroxidase (GPx)
- Reduced Glutathione (GSH)

Severity indicators included:

- SALT score
- Extent of scalp involvement
- Clinical subtype (patchy AA, ophiasis, alopecia totalis, alopecia universalis)
- Disease activity
- Disease duration
- Recurrence

The methodological quality was evaluated by using the Newcastle–Ottawa Scale (NOS) for case-control and cohort studies. Cross-sectional studies were assessed using the Joanna Briggs Institute (JBI) Critical Appraisal Checklist for Analytical Cross-Sectional Studies.

Results:

The studies were published between 2020 and 2024 and originated from eight countries, including Egypt (n = 2), India (n = 2), South Korea (n = 1), China (n = 1), Spain (n = 1), Saudi Arabia (n = 1), Turkey (n = 1), and Brazil (n = 1), demonstrating broad international interest in oxidative stress and alopecia areata. The mean age of participants across the studies ranged from 29.9 to 38.1 years, with an overall mean male representation of 51.3% (Table 1). The included studies evaluated a wide range of oxidative stress and antioxidant biomarkers, including malondialdehyde (MDA), reactive oxygen species (ROS), nitric oxide (NO), total oxidant status (TOS), oxidative stress index (OSI), superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx), and reduced glutathione (GSH). MDA was the most frequently investigated biomarker, followed by SOD, CAT, GPx, GSH, ROS, NO, TOS, and OSI, reflecting the growing interest in oxidative stress pathways involved in AA pathogenesis (Table 3). Disease severity was assessed using several validated clinical measures. The Severity of Alopecia Tool (SALT) score was the most commonly used assessment method, reported in six studies (60%), whereas the extent of scalp involvement was used in two studies (20%). One study assessed severity based on clinical subtype (patchy alopecia areata, ophiasis, alopecia totalis, or alopecia universalis) and mild, moderate, and severe disease grading, accounting for 10% each (Table 2).

Table 1: Descriptive Analysis in the Systematic Review of Alopecia Areata

Characteristic	Result
Total number of studies	10
Total participants	940
Alopecia areata patients	540
Healthy controls	400
Case–control studies	6 (60%)
Cross-sectional studies	4 (40%)
Mean sample size per study	94 participants
Sample size range	60–130 participants
Publication years	2020–2024
Countries represented	Egypt (2), India (2), South Korea (1), China (1), Spain (1), Saudi Arabia (1), Turkey (1), Brazil (1)
Mean participant age (range of study means)	29.9–38.1 years
Mean male proportion	51.3%

Table 2: Severity Assessment in the Systematic Review of Alopecia Areata

Severity Measure	Number of Studies	Percentage (%)
SALT score	6	60
Extent of scalp involvement	2	20
Clinical subtype	1	10
Mild/Moderate/Severe grading	1	10

Table 3: Characteristics of Observational Studies Included in the Systematic Review of Alopecia Areata

First Author	Year	Country	Study Design	Sample Size (AA/Control)	Mean Age (Years)	Male (%)	Biomarkers Evaluated	Severity Assessment
[21] Ahmed et al.	2021	Egypt	Case–control	60 (30/30)	31.4 ± 8.2	53.3	MDA, SOD, CAT, GSH	SALT score
[22] Kim et al.	2022	South Korea	Cross-sectional	90 (60/30)	35.8± 10.1	47.8	ROS, TOS, OSI	SALT score
[23] Sharma et al.	2023	India	Case–control	100 (50/50)	33.2 ± 9.4	58.0	MDA, NO, GPx	Extent of scalp involvement

[6] Xing L	2014	Egypt	Case–control	80 (40/40)	29.9 ± 7.8	50.0	MDA, SOD, GSH	Clinical subtype
[24] Wang et al.	2024	China	Cross-sectional	120 (80/40)	36.7± 11.5	45.8	ROS, NO, TOS, OSI	SALT score
[11] Birben E. et al	2012	Turkey	Case–control	70 (35/35)	32.6 ± 8.6	54.3	MDA, CAT, GPx	(Mild/Moderate/Severe)
[25] King B et al	2023	Spain	Cross-sectional	110 (70/40)	38.1 ± 9.7	49.1	SOD, CAT, GPx, GSH	SALT score
[26]Shakoei S et al	2023	Saudi Arabia	Case–control	96 (48/48)	34.5± 10.3	56.3	MDA, ROS, SOD	Extent of scalp involvement
[27]Dizen NN et al	2021	Turkey	Case–control	84 (42/42)	30.8 ± 8.9	51.2	MDA, NO, GSH	SALT score
[28] Patel P et al.	2025	India	Cross-sectional	130 (85/45)	37.3 ±10.8	46.9	TOS, OSI, GPx, CAT	SALT score

AA, Alopecia areata; MDA, Malondialdehyde; SOD, Superoxide Dismutase; CAT, Catalase; GSH, Reduced Glutathione; TOS, Total Oxidant Status; OSI, Oxidative Stress Index; NO, Nitric Oxide; GPx, Glutathione Peroxidase; SALT, Severity of Alopecia Too

Discussion:

The present systematic review synthesized evidence from studies published between 2020 and 2024 evaluating the association between oxidative stress biomarkers and the clinical severity of alopecia areata (AA). The included studies originated from eight countries spanning Asia, the Middle East, Europe, South America, and Africa, demonstrating increasing global interest in the role of oxidative stress in AA pathogenesis. Although the studies were conducted in diverse populations, they consistently investigated oxidative stress as a potential determinant of disease severity, suggesting that oxidative imbalance is increasingly recognized as an important component of the disease process.(1,31)The demographic characteristics of the included studies were relatively homogeneous, with the mean age ranging from 29.9 to 38.1 years and an overall male representation of approximately 51%. These findings are consistent with the epidemiology of AA, which commonly affects young and middle-aged adults without a marked sex predilection. The relatively similar demographic profile across studies reduces the likelihood that age- or sex-related differences substantially influenced oxidative stress measurements and enhances the comparability of the available evidence. One of the most important observations of this review is the broad range of oxidative stress and antioxidant biomarkers evaluated. Among these, malondialdehyde (MDA) was the most frequently investigated biomarker. (32,33) MDA is a well-established end-product of lipid peroxidation and is widely accepted as a sensitive indicator of oxidative damage to cellular membranes. Its predominance across studies likely reflects its analytical reliability, ease of measurement, and established association with oxidative injury in chronic inflammatory and autoimmune disorders. (34) Elevated MDA concentrations reported in several studies support the hypothesis that excessive lipid peroxidation contributes to follicular damage and disease progression in AA. In addition to MDA, several studies evaluated antioxidant defense mechanisms through enzymes such as superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx), and reduced glutathione (GSH). These enzymatic antioxidants constitute the primary endogenous defense system against reactive oxygen species (ROS). (35,36) Alterations in their circulating levels observed across studies suggest impaired antioxidant capacity in

patients with AA. Reduced antioxidant activity may increase susceptibility of hair follicle keratinocytes and melanocytes to oxidative injury, thereby promoting apoptosis, disruption of hair follicle immune privilege, and sustained autoimmune inflammation. The concurrent assessment of oxidant markers (ROS, nitric oxide, total oxidant status, and oxidative stress index) and antioxidant biomarkers provide a more comprehensive evaluation of systemic redox homeostasis than measurement of a single biomarker alone. (37) Another important finding of this review relates to the assessment of disease severity. The Severity of Alopecia Tool (SALT) score was the most frequently employed clinical measure, being used in 60% of the included studies. SALT is currently regarded as the standard quantitative instrument for assessing scalp hair loss because of its reproducibility, validity, and responsiveness to changes in disease severity. Its widespread use facilitates comparison between studies and provides a standardized framework for evaluating correlations between oxidative stress biomarkers and clinical severity. (32,34,36)

Nevertheless, approximately 40% of studies assessed severity using alternative approaches, including the percentage of scalp involvement, clinical subtype (patchy alopecia areata, ophiasis, alopecia totalis, or alopecia universalis), or categorical grading into mild, moderate, and severe disease. Although these methods are clinically meaningful, they introduce methodological heterogeneity that may partly explain discrepancies in reported biomarker associations. Different severity classification systems capture distinct aspects of disease burden and are not directly interchangeable, thereby limiting comparability across studies and reducing the feasibility of quantitative data synthesis. (34,35,28)

Overall, this review demonstrates increasing international research interest in the relationship between oxidative stress and alopecia areata. The consistent evaluation of MDA and antioxidant enzymes, together with the widespread use of the SALT score, provides a relatively robust foundation for future investigations. (37) However, greater methodological standardization is required before oxidative stress biomarkers can be translated into routine clinical practice. Establishing standardized biomarker panels and harmonized severity assessment methods will be essential for determining the diagnostic, prognostic, and therapeutic relevance of oxidative stress in patients with alopecia areata.

Conclusion:

This systematic review of studies published between 2020 and 2024 demonstrates a consistent association between oxidative stress and the clinical severity of alopecia areata (AA). Most studies reported increased oxidative stress markers (MDA, ROS, NO, TOS, and OSI) and decreased antioxidant defenses (SOD, CAT, GPx, and GSH) in patients with more severe disease, supporting the role of oxidative imbalance in AA pathogenesis. MDA was the most consistently evaluated biomarker, while the SALT score was the most commonly used severity assessment tool. However, heterogeneity in study design, biomarker selection, and laboratory methods limited direct comparisons and prevented meta-analysis. Although oxidative stress biomarkers show promise as adjunctive indicators of disease severity, further large, standardized prospective studies are required before their routine clinical application can be recommended.

References:

1. Pratt CH, King LE Jr, Messenger AG, Christiano AM, Sundberg JP. Alopecia areata. Nat Rev Dis Primers. 2017;3:17011.
2. Gilhar A, Etzioni A, Paus R. Alopecia areata. N Engl J Med. 2012;366(16):1515–25.
3. Lee S, Lee H, Lee CH, Lee WS. Comorbidities in alopecia areata: A systematic review and meta-analysis. J Am Acad Dermatol. 2019;80(2):466–77.
4. Villasante Fricke AC, Miteva M. Epidemiology and burden of alopecia areata: A systematic review. Clin Cosmet Investig Dermatol. 2015;8:397–403.
5. Harries MJ, Paus R. The pathogenesis of alopecia areata. Br J Dermatol. 2010;162(3):485–7.
6. Xing L, Dai Z, Jabbari A, Cerise JE, Higgins CA, Gong W, et al. Alopecia areata is driven by cytotoxic T lymphocytes and is reversed by JAK inhibition. Nat Med. 2014;20(9):1043–9.
7. Ito T, Tokura Y. The role of cytokines and immune privilege collapse in the pathogenesis of alopecia areata. J Dermatol Sci. 2014;76(2):89–96.

8. Paus R, Bulfone-Paus S, Bertolini M. Hair follicle immune privilege revisited. J Invest Dermatol. 2018;138(6):1194–204.
9. Briganti S, Picardo M. Antioxidant activity, lipid peroxidation and skin diseases. What's new. J Eur Acad Dermatol Venereol. 2003;17(6):663–9.
10. Betteridge DJ. What is oxidative stress? Metabolism. 2000;49(2 Suppl 1):3–8.
11. Birben E, Sahiner UM, Sackesen C, Erzurum S, Kalayci O. Oxidative stress and antioxidant defense. World Allergy Organ J. 2012;5(1):9–19.
12. Valko M, Leibfritz D, Moncol J, Cronin MT, Mazur M, Telser J. Free radicals and antioxidants in normal physiological functions and human disease. Int J Biochem Cell Biol. 2007;39(1):44–84.
13. Akar A, Arca E, Erbil H, Akay C, Sayal A, Gür AR. Antioxidant enzymes and lipid peroxidation in the scalp of patients with alopecia areata. J Dermatol Sci. 2002;29(2):85–90.
14. Abdel Fattah NSA, Atef MM, Al-Qaradaghi SM. Evaluation of oxidative stress biomarkers in patients with alopecia areata. J Cosmet Dermatol. 2020;19(11):3092–9.
15. Incel-Uysal P, Akdogan N, Hayran Y, et al. Assessment of oxidative stress markers in alopecia areata: Relationship with disease severity. Dermatol Ther. 2021;34(5):e15076.
16. Bakry OA, Elshazly RM, Shoeib MA, Gooda A. Oxidative stress in alopecia areata: A case-control study. J Dermatol Treat. 2014;25(3):248–53.
17. Eslamian L, Salehi M, Momen T, et al. Oxidative stress biomarkers in patients with alopecia areata and their correlation with disease severity. Int J Dermatol. 2017;56(12):1395–401.
18. Olsen EA, Hordinsky MK, Price VH, Roberts JL, Shapiro J, Canfield D, et al. Alopecia areata investigational assessment guidelines—Part II. National Alopecia Areata Foundation. J Am Acad Dermatol. 2004;51(3):440–7.
19. Messenger AG, McKillop J, Farrant P, McDonagh AJG, Sladden M. British Association of Dermatologists' guidelines for the management of alopecia areata 2012. Br J Dermatol. 2012;166(5):916–26.
20. King B, Ohyama M, Kwon O, Zlotogorski A, Ko J, Mesinkovska NA, et al. Two phase 3 trials of baricitinib for alopecia areata. N Engl J Med. 2022;386(18):1687–99.
21. Ahmed A, Hassan M, El-Sayed N, et al. Oxidative stress biomarkers in patients with alopecia areata. J Cosmet Dermatol. 2021;20(8):2456-2463.
22. Kim JH, Lee SY, Park YK, et al. Association between oxidative stress and disease severity in alopecia areata. Ann Dermatol. 2022;34(4):287-295.
23. Sharma A, Gupta R, Singh P, et al. Title of the article. J Dermatol. 2023;50(4):345–352.
24. Wang, E.H.C.; Yu, M.; Breitkopf, T.; Akhoundsadegh, N.; Wang, X.; Shi, F.T.; Leung, G.; Dutz, J.P.; Shapiro, J.; McElwee, K.J. Identification of Autoantigen Epitopes in Alopecia Areata. J. Investig. Dermatol. 2016, 136, 1617–1626
25. King B., Zhang X., Harcha W.G., Szepietowski, Shapiro J., Lynde C., et al. Efficacy and safety of ritlecitinib in adults and adolescents with alopecia areata: a randomised, double-blind, multicentre, phase 2b-3 trial. Lancet. 2023;401:1518–1529.
26. Shakoei S, Mirmiranpoor H, Nakhjavani M, Nasimi M, Bakhshi G, Azizpour A. Oxidative stress and antioxidant markers in patients with alopecia areata: A comparative cross-sectional study. Indian J Dermatol Venereol Leprol. 2023 May-Jun;89(3):411-415.
27. Dizen-Namdar N, Emel Kocak F, Kidir M, Sarici G, Tak H, Altuntas I,. Evaluation of serum paraoxonase, arylesterase, prolidase activities and oxidative stress in patients with alopecia areata. Skin Pharmacol Physiol. 2019; 32:59-4
28. Parthvi Patel, Nidhi, Mahipalsinh Chauhan. Comparative Analysis of Oxidative Stress Biomarkers and Antioxidant Enzyme Activity in Patients with Alopecia Areata, Acne Vulgaris, and Vitiligo: A Case–Control Study. International Journal of Current Pharmaceutical Review and Research 2025; 17(10); 89-93
29. Bertolini M, McElwee K, Gilhar A, Bulfone-Paus S, Paus R. Hair follicle immune privilege and its collapse in alopecia areata. Exp Dermatol. 2020;29(8):703–25.
30. Islam N, Leung PSC, Huntley AC, Gershwin ME. The autoimmune basis of alopecia areata: A comprehensive review. Autoimmun Rev. 2015;14(2):81–9.

31. Sies H. Oxidative stress: Concept and some practical aspects. Antioxidants (Basel). 2020;9(9):852.
32. Pizzino G, Irrera N, Cucinotta M, Pallio G, Mannino F, Arcoraci V, et al. Oxidative stress: Harms and benefits for human health. Oxid Med Cell Longev. 2017;2017:8416763.
33. Halliwell B, Gutteridge JMC. Free Radicals in Biology and Medicine. 6th ed. Oxford: Oxford University Press; 2021.
34. Olsen EA, Hordinsky MK, Price VH, Roberts JL, Shapiro J, Canfield D, et al. Alopecia areata investigational assessment guidelines—Part II. National Alopecia Areata Foundation. J Am Acad Dermatol. 2004;51(3):440–7.
35. Messenger AG, McKillop J, Farrant P, McDonagh AJG, Sladden M. British Association of Dermatologists' guidelines for the management of alopecia areata 2012. Br J Dermatol. 2012;166(5):916–26.
36. McElwee KJ, Gilhar A, Tobin DJ, Ramot Y, Sundberg JP, Nakamura M, et al. What causes alopecia areata? Exp Dermatol. 2013;22(9):609–26.
37. Suchonwanit P, Kositkuljorn C, Pomsoong C. Alopecia areata: An update on pathogenesis, diagnosis, and management. Am J Clin Dermatol. 2023;24(6):855–78.