

# Alopecia areata: The disease of hairless patches on the scalp and any area of face

Sakshi Patel<sup>1</sup>, Yashica Rawal<sup>2</sup>, Dr. Pragnesh Patani<sup>3</sup>

<sup>1,2,3</sup>Khyati College of Pharmacy, Palodia, Ahmedabad, Gujarat

Email: sakshisangani08@gmail.com

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## Abstract

Up to 2% of the population is affected with alopecia areata (AA), a prevalent chronic tissue-specific autoimmune disease that causes hair loss. The specific pathobiology of AA is still unknown, however the most widely accepted idea is that an immunological process led the hair follicle's immune privilege to collapse. The etiology of AA is influenced by numerous environmental and genetic variables. One or more well-defined patches, more diffuse or total hair loss of the scalp (alopecia totalis), or hair loss of the entire body are all clinically treated for AA using a variety of clinical approaches (alopecia universalis). Corticosteroids and other immunomodulators, minoxidil, and contact immunotherapy are some of the available treatments for AA.

**Keywords:** alopecia areata, autoimmune disease, hair loss of face and scalp.

## INTRODUCTION

Alopecia areata is a common kind of hair loss or alopecia in humans. It is an autoimmune illness with a varied, sometimes relapsing or remitting course that can be permanent, especially when hair loss is considerable. Alopecia areata is the second-most prevalent non-scarring alopecia, behind male and female pattern alopecia. About alopecia areata Clinical hair loss often exhibits extremely distinctive patterns.

Round or oval patches on the head (90% of cases) or other parts of the body are indicative of the most common type of alopecia areata in patches (AA), alopecia totalis (AT), in which the scalp's hair is completely or almost completely absent, and alopecia universalis (AU), in which hair loss is widespread. Every patient goes through a different, illogical course.

## IMMUNOLOGICAL FACTORS

Numerous research teams have shown a substantial connection between AA and autoimmune diseases like vitiligo. Vitiligo is considered to be two times more common in AA patients than in the general population. Although Addison's disease, systemic lupus erythematosus, myasthenia gravis, scleroderma, allergic urticaria, and allergic urticaria all enhance the chance of getting AA, it has been discovered that 4.1% of individuals with vitiligo and 2.3% of persons with thyroid illness are at risk for developing AA. 26 Additionally, those who already have vitiligo as well as those with AT and AU have a greater risk.

Additionally, it has been suggested that the anagen phase structure of patients' own hair follicles, especially the infiltration of CD4+ and CD8+ T lymphocytes into the hair follicle bulb, is altered by their antibodies, causing AA.

## GENETIC FACTOR

Although the majority of cases of AA are sporadic, reports have suggested a strong connection between genetic factors and disease development. This is supported by three different types of studies: 1) family-based linkage; 2) studies in monozygotic twins, where a concordance of 50% of developing the disease has been observed; and 3) studies based on heritability in first-degree relatives, where a positive family incidence of 10 to 42% for AA has been reported.

## ENVIRONMENTAL FACTOR

In terms of the environment, it's been proposed that stress may be one of the factors that might support the growth of AA. Studies show that an emotional incident or identity crisis occurred for at least 23% of AA patients before the disease manifested itself. Although not all of them have been shown to be successful, other variables, such as infections, toxins, and even diet, have been proposed as possible disease triggers.

## OXIDATIVE STRESS

In AA patients, oxidative stress may cause the overexpression of NKG2D ligands like MICA and ULBP, disrupting IP and fostering autoimmunity. . It has been discovered that AA patients' blood cells and lesions have greater levels of malondialdehyde and lower erythrocyte superoxide dismutase (SOD) and glutathione peroxidase (GSH-Px) activity (MDA). Genetic analysis has shown that AA involves the antioxidant enzyme PRDX5. Further recently, a meta-analysis found a connection between AA and elevated oxidative stress, albeit more studies are required to confirm this association.

## ALLERGY

It has been documented that there is a link between AA and atopic illnesses, particularly atopic dermatitis. The likelihood of getting AA increases with atopy [50]. Mast cells and eosinophils were found in AA patients' lesions. Numerous studies have noted the elevated serum IgE level in AA patients. Dust mite allergy was shown by Zhang et al. to be connected to the severity of AA. . In atopic AA patients, antihistamines and desensitization for house dust mite allergens may help lower the severity of alopecia. Furthermore, cytokine profiles and genetic susceptibility loci have shown the involvement of the Th2 skewing. The IL-4 receptor monoclonal antibody dupilumab caused hair regrowth in certain AA individuals with atopic dermatitis.

## TYPES OF ALOPECIA AREATA

1. Patchy alopecia areata = one, several distinct patches, or several connected (reticular) patches of hair loss.
2. alopecia totalis = whole or nearly complete baldness on the scalp.
3. Alopecia incognita = Without nail involvement, there is diffuse complete hair loss with a positive pull test, yellow spots, and small, miniature regrowing hairs.
4. Ophiasis = hair loss in the form of a band around the perimeter of the head, most notably along the temporal and occipital bone borders.
5. Saisapho = severe alopecia, with the exception of the scalp's edges.
6. Marie Antoinette syndrome (also called canities subita): widespread alopecia with very abrupt "overnight" graying and preferential loss of pigmented hair during an acute episode.

## TREATMENT

A successful treatment plan could have brief relapses that can occur from time to time. A person's condition may occasionally result in a new lesion of AA on one side of the scalp while concurrently suffering regrowth in a patch of previously treated AA in a separate location of the scalp.

Australasian Hair and Makeup Awards participants The Wool Research Society presented at the 10th World Wool Conference, The Congress for Hair Research was held in Kyoto in November 2017.

At this conference, a dermatology clinical research fellow, a research scientist with extensive understanding of hair biology and sickness, and Australian dermatologists with experience in alopecia were all present. An evaluation of the literature follows.

At the Kyoto conference and later by letters, the dialogue took place. A consensus statement for the systemic treatment of AA was developed as a result of these discussions. and presented for comment to skilled Australian dermatologists.

## CONSERVATIVE MANAGEMENT

For those with minor, stable conditions that are inconspicuous or simple to conceal, reassurance alone may be helpful. The scalp can be covered with colors applied as a cream or aerosol spray, and the hair can be dusted with coloured wool fibers ground into a powder for camouflaging. Additionally, wigs, top pieces, and hair extensions are options.

## TOPICAL THERAPY

Patients may seek active treatment if they just have a small amount of illness. to quicken regrowth Topical glucocorticoids, topical minoxidil, and topical immunotherapy are available options. Diphenylcyclopropenone, dinitrochlorobenzene, squaric acid dibutyl ester, and dithranol are some of the substances utilized in topical immunotherapy.

## VITAMIN D ROLE

It's interesting to note that there is conflicting information regarding the relationship between clinical illness characteristics and blood 25(OH)D levels. In 42 individuals with AA, Yilmaz et al. did not discover a relationship between 1,25(OH)2D or 25(OH)D concentrations and the degree of hair loss, the number of patches, the duration of the condition,

This was validated in another study and nail involvement. The authors of Aksu Cerman et al. established for the first time that serum 25(OH)D levels were negatively linked with the severity of AA as measured by the Severity of Alopecia Tool (SALT) score. It has been hypothesized that more people with lower serum levels of 25(OH)D detected in Patients may become discouraged in the later stages of AA as a result of the emotional stress brought on by excessive hair loss.



## REFERENCES

1. Pratt CH, King LE, Messenger AG, Christiano AM, Sundberg JP. Alopecia areata. *Nature reviews Disease primers*. 2017 Mar 16;3(1):1-7.
2. Juárez-Rendón KJ, Rivera Sánchez G, Reyes-López MÁ, García-Ortiz JE, Bocanegra-García V, Guardiola-Avila I, Altamirano-García ML. Alopecia areata: Actualidad y perspectivas. *Archivos argentinos de pediatría*. 2017 Dec;115(6):e404-11.
3. Zhou, C., Li, X., Wang, C. et al. Alopecia Areata: an Update on Etiopathogenesis, Diagnosis, and Management. *Clinical Rev Allerg Immunol* 61, 403–423 (2021).
4. Gilhar A, Etzioni A, Paus R. Alopecia areata. *N Engl J Med*. 2012 Apr 19;366(16):1515-25.
5. Barton VR, Toussi A, Awasthi S, Kiuru M. Treatment of pediatric alopecia areata: A systematic review. *Journal of the American Academy of Dermatology*. 2021 Apr 30.
6. Cranwell WC, Lai VW, Photiou L, Meah N, Wall D, Rathnayake D, Joseph S, Chitreddy V, Gunatheesan S, Sindhu K, Sharma P. Treatment of alopecia areata: an Australian expert consensus statement. *Australasian Journal of Dermatology*. 2019 May;60(2):163-70
7. Islam N, Leung PS, Huntley AC, et al. The autoimmune basis of alopecia areata: A comprehensive review. *Autoimmun Rev* 2015;14(2):81-9.
8. Riad H, Mannai HA, Mansour K, et al. Diphenylcyclopropenone-induced vitiligo in a patient with alopecia universalis. *Case Rep Dermatol* 2013;5(2):225-31.
9. Hordinsky MK. Overview of alopecia areata. *J Investig Dermatol Symp Proc* 2013;16(1):S13-5.
10. Kim SK, Park HJ, Chung JH, et al. Association between interleukin 18 polymorphisms and alopecia areata in Koreans. *J Interferon Cytokine Res* 2014;34(5):349-53.
11. Salinas-Santander M, Sánchez-Domínguez C, CantúSalinas C, et al. Association between PTPN22 C1858T polymorphism and alopecia areata risk. *Exp*

- Ther Med 2015;10(5):1953-8.
12. Ranawaka RR. An observational study of alopecia areata in Sri Lankan adult patients. *Ceylon Med J*. 2014;59(4):128-31.
  13. Aytekin N, Akcali C, Pehlivan S, et al. Investigation of interleukin-12, interleukin-17 and interleukin-23 receptor gene polymorphisms in alopecia areata. *J Int Med Res* 2015;43(4):526-34.
  14. Alzolibani AA, Zari S, Ahmed AA. Epidemiologic and genetic characteristics of alopecia areata (part 2). *Acta Dermatovenol Alp Pannonica Adriat* 2012;21(1):15-9.
  15. Olguín García MG, Martín del Campo A, Rodríguez Acar M, et al. Factores psicológicos asociados con la alopecia areata. *Dermatol Rev Mex* 2013;57(3):171-7.
  16. Alzolibani AA. Epidemiologic and genetic characteristics of alopecia areata (part 1). *Acta Dermatovenol Alp Pannonica Adriat* 2011;20(4):191-8.
  17. Rajabi F, Drake LA, Senna MM, Rezaei N (2018) Alopecia areata: a review of disease pathogenesis. *Br J Dermatol* 179(5):1033–1048.
  18. Yenin JZ, Serarslan G, Yonden Z, Ulutas KT (2015) Investigation of oxidative stress in patients with alopecia areata and its relationship with disease severity, duration, recurrence and pattern. *Clin Exp Dermatol* 40(6):617–621.
  19. Ozturk P, American O, Kurutas EB, Mulayim K (2016) Oxidative stress biomarkers and adenosine deaminase over the alopecic area of the patients with alopecia areata. *Balkan Med J* 33(2):188–192.
  20. Petukhova L, Duvic M, Hordinsky M, Norris D, Price V, Shimomura Y, Kim H, Singh P, Lee A, Chen WV, Meyer KC, Paus R, Jahoda CA, Amos CI, Gregersen PK, Christiano AM (2010) Genome-wide association study in alopecia areata implicates both innate and adaptive immunity. *Nature* 466(7302):113–117.
  21. Pourang A, Mesinkovska NA (2020) New and emerging therapies for alopecia areata. *Drugs* 80(7):635–646.
  22. Zhang X, Zhao Y, Ye Y, Li S, Qi S, Yang Y, Cao H, Yang J, Zhang X (2015) Lesional infiltration of mast cells, Langerhans cells, T cells and local cytokine profiles in alopecia areata. *Arch Dermatol Res* 307(4):319–331.
  23. Ito T, Kageyama R, Nakazawa S, Honda T (2020) Understanding the significance of cytokines and chemokines in the pathogenesis of alopecia areata. *Exp Dermatol* 29(8):726–732.
  24. Yoon TY, Lee DY, Kim YJ, Lee JY, Kim MK (2014) Diagnostic usefulness of a peribulbar eosinophilic infiltrate in alopecia areata. *JAMA Dermatol* 150(9):952–956.
  25. Andersen YM, Egeberg A, Gislason GH, Skov L, Thyssen JP (2017) Autoimmune diseases in adults with atopic dermatitis. *J Am Acad Dermatol* 76(2):274–280 e271.
  26. Mohan GC, Silverberg JI (2015) Association of vitiligo and alopecia areata with atopic dermatitis: a systematic review and meta-analysis. *JAMA Dermatol* 151(5):522–528.
  27. Barahmani N, Schabath MB, Duvic M, National Alopecia Areata R (2009) History of atopy or autoimmunity increases risk of alopecia areata. *J Am Acad Dermatol* 61(4):581–591.
  28. Zhao Y, Zhang B, Caulloo S, Chen X, Li Y, Zhang X (2012) Diffuse alopecia areata is associated with intense inflammatory infiltration and CD8+ T cells in hair loss regions and an increase in serum IgE level. *Indian J Dermatol Venereol Leprol* 78(6):709–714.
  29. Li SF, Zhang XT, Qi SL, Ye YT, Cao H, Yang YQ, McElwee KJ, Zhang X (2015) Allergy to dust mites may contribute to early onset and severity of alopecia areata. *Clin Exp Dermatol* 40(2):171–176.
  30. Zhang X, McElwee KJ (2020) Allergy promotes alopecia areata in a subset of patients. *Exp Dermatol* 29(3):239–242.
  31. Ohshima M, Shimizu A, Tanaka K, Amagai M (2010) Experimental evaluation of ebastine, a second-generation antihistamine, as a supportive medication for alopecia areata. *J Dermatol Sci* 58(2):154–157.
  32. Marks DH, Mesinkovska N, Senna MM (2019) Cause or cure? Review of dupilumab and alopecia areata. *J Am Acad Dermatol*.
  33. Ganjoo S, Thappa D. Dermoscopic evaluation of therapeutic response to an intralesional corticosteroid in the treatment of alopecia areata. *Indian J. Dermatol. Venereol. Leprol.* 2013; 79: 408–17.
  34. Kurosawa M, Nakagawa S, Mizuhashi M et al. A comparison of the efficacy, relapse rate and side effects among three modalities of systemic corticosteroid therapy for alopecia areata. *Dermatology* 2006; 212: 361–5.
  35. Kar B, Handa S, Dogra S et al. Placebo-controlled oral pulse prednisolone therapy in alopecia areata. *J. Am. Acad. Dermatol.* 2005; 52: 287–90.
  36. Navarini, A. A., Nobbe, S. & Trüeb, R. M. Marie Antoinette syndrome. *Arch. Dermatol.* 145, 656 (2009).
  37. Forstbauer, L. M. et al. Genome-wide pooling approach identifies SPATA5 as a new susceptibility locus for alopecia areata. *Eur. J. Hum. Genet.* 20, 326–332 (2012).
  38. Jagielska, D. et al. Follow-up study of the first genome-wide association scan in alopecia areata: IL13 and KIAA0350 as susceptibility loci supported with genome-wide significance. *J. Invest. Dermatol.* 132, 2192–2197 (2012).
  39. Wohltmann WE, Sperling L. Histopathologic diagnosis of multifactorial alopecia. *J Cutan Pathol* 2016;43(6):483-91.
  40. Nalluri R, Harries M. Alopecia in general medicine. *Clin Med (Lond)* 2016;16(1):74-8.
  41. Betz RC, Petukhova L, Ripke S, et al. Genome-wide metaanalysis in alopecia areata resolves HLA associations and reveals two new susceptibility loci. *Nat Commun* 2015;6:5966.
  42. Rencz F, Gulácsi L, Péntek M, et al. Alopecia areata and health-related quality of life: a systematic review and metaanalysis. *Br J Dermatol* 2016;175(3):561-71
  43. Harries MJ, Sun J, Paus R, King LE Jr. Management of alopecia areata. *BMJ* 2010; 341:c3671.
  44. Alkhalifah A, Alsantali A, Wang E, et al. Alopecia areata update: part II. Treatment. *J Am Acad Dermatol* 2010;62:191-202.
  45. Garg S, Messenger AG. Alopecia areata: evidence based treatments. *Semin Cutan Med Surg* 2009;28:15-8.
  46. Stoehr JR, Choi JN, Colavincenzo M, Vanderweil S. Off-Label Use of Topical Minoxidil in Alopecia: A Review. *American journal of clinical dermatology.* 2019;20(2):237-250.
  47. Yun D, Silverberg NB, Stein SL. Alopecia areata treated with hydroxychloroquine: A retrospective study of nine pediatric cases. *Pediatr Dermatol.* 2018;35(3):361-365.
  48. Lenane P, Pope E, Krafchik B. Congenital alopecia areata. *Journal of the American Academy of Dermatology.* 2005;52(2 Suppl 1):8-11.
  49. Suchonwanit P, Thammarucha S, Leerunyakul K. Minoxidil and its use in hair disorders: a review. *Drug Des Devel Ther.* 2019;13:2777-2786.
  50. Lee HH, Gwillim E, Patel KR, Hua T, Rastogi S, Ibler E, Silverberg JI (2020) Epidemiology of alopecia areata, ophiasis, totalis, and universalis: a systematic review and meta-analysis. *J Am Acad Dermatol* 82(3):675–682.
  51. Meguri F, Mora B, Maxia C, et al. Cytotoxic T-lymphocyte antigen 4 (CTLA4) +49AG and CT60 gene polymorphisms in Alopecia Areata: a case-control association study in the Italian population. *Arch Dermatol Res* 2013;305(7):665-70.