

A Study on Dermoscopic Findings in Alopecia Areata

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Abstract

Alopecia areata is a common chronic autoimmune inflammatory and non-scarring disease which involves hair follicles, characterised by the hair loss on the scalp and other hairy regions of the body. The reported chance of occurrence is 2% in their lifetime of an individual, with much higher probability in younger individuals aged below 30 years. Depending on the ethnic background and the worldwide regional difference, the prevalence of alopecia areata (AA) varies from 0.1 to 0.2%, with a calculated lifetime risk of 2%. Recent studies on animal models have proposed collapse of hair follicle immune privilege in association with enhanced MHC class I expression and organ-specific autoimmune reactions against hair follicle autoantigens induced by NKG2D+ cytotoxic CD8+ T cells as the probable mechanism for the development of Alopecia areata. In the current study, the average age of the study population was 25.1 ± 11.75 , which was ranging between 3 to 46 years. But the majority of the study participants were in their second decade and third decade of life. There was a slight male preponderance, as 60% of the participants were male. The present study includes to observe the clinical and dermoscopic findings in Alopecia Areata patients.

Keywords : Alopecia areata, ophiasis , scalp , HLA-DR and diffuse patches

1. Introduction

Alopecia areata is a common chronic autoimmune inflammatory and non-scarring disease which involves hair follicles. It is characterized by hair loss on the scalp and/or wherever the hair is present on the body.¹ The chance of occurring is 2% in the lifetime of an individual.² The chance of occurring is 40% higher in younger individuals aged below 30 years.¹⁻³

Clinically, Alopecia areata is differentiated into patchy alopecia, diffuse alopecia, reticulate alopecia, ophiasis, ophiasis inverses (loss of hair in the shape of wave) alopecia totalis or alopecia universalis (loss of all hairs) and perinevoid.⁴ Most commonly around 3% to 30% of patients shows nail changes (diffuse fine nail pitting, longitudinal ridging, thin and

brittle finger and toenails, and trachyonychia).³ Since it is an autoimmune disorder, sudden hair regrowth may occur at any time within the year of hair loss.¹ The immune system is a major player, with T cells and a collapse of the physiological immune privilege (IP) of the hair follicle (HF)⁵ plays a critical role in the pathophysiology of alopecia areata.⁶⁻⁸ The HF represents a site of relative IP, because defined regions of its epithelium (bulge, bulb) do not express MHC class I and class II molecules, and because a number of immunoinhibitory cytokines and neuropeptides create an immunoinhibitory milieu.^{5,9} Even the few intraepithelial Langerhans cells found within the HF epithelium below its stem cell region, the bulge, are immunologically impaired, as they fail to express MHC class II.¹⁰ This collapse of immune privilege (IP) of the hair follicle is a cause for autoimmunity to occur, is a widely accepted theory.¹¹ This IP collapse, which can most effectively be induced by IFN- γ or substance P presumably leads to changes in the quality and quantity of the expressed self-antigen repertoire, rendering HFs, which now ectopically express MHC class I-presented autoantigens, vulnerable to anti-self-immune reactivity.¹²

Clinical diagnosis of Alopecia areata is based on the typical pattern of the hair loss, and the presence of a characteristic exclamation mark hair in microscopy. In some cases, if clinical features are not clear, invasive method (punch biopsy) may be needed for the diagnosis.¹³ The dermoscopic method has been proposed as an alternative to avoid punch biopsy, especially in adolescent girls and children, in whom acceptability of punch biopsy may be very low. Many recent studies have explored the utility of noninvasive diagnostic procedure, dermoscopy in the diagnosis of Alopecia Areata.¹⁴ Apart from these, flow cytometry-based measurement of various inflammatory markers like IFN- γ , IL-13, IL-9, IL-17, and IL-22 cytokines in CD4⁺ and CD8⁺ T cells have been used to supplement or confirm the clinical diagnosis. Inducible co-stimulator molecule (ICOS) and HLA-DR, which are used to define mid- and long-term T-cell activation are also proposed as potential diagnostic markers in Alopecia areata.¹⁵

Dermoscopy

Dermoscopy is a non-invasive imaging technique which shows the magnified lesions on the surface of the skin. Skin surface microscopy for pigmented lesions was initially described in the first half of the 20th century based on earlier work done on colposcopy for visualisation of the cervical region.¹⁶ A couple of decades later, the use of oil-immersion fluid as an interface to improve skin surface visualisation and the use of the same for the diagnosis of pigmented lesions was described. Large dedicated dermoscopic devices were first used in the late 1980s, and hand-held devices started to be developed for the same in the early 1990s.¹⁶ Dermoscopy is usually performed with Heine Delta 20 dermoscope, which usually have a magnification of around $\times 10$ to $\times 20$.¹³ The same can be used to take photographs by connecting them to a wide variety of cameras. Dermoscopy worked with the principle of illumination and transillumination of skin with different light sources and observed under the high magnifying lens. Most of the light is scattered due to the reflective property of the stratum corneum. In order to overcome this problem, fluid (liquid paraffin) medium is used as an interface, and a transparent glass contact plate is used for clear vision.

Use of cross- polarized light is another method for this purpose. Both the methods allow looking at the clear image of a deeper section of the skin.¹⁶ Advanced dermoscopes are also increasingly available with polarised light allowing contact and non-contact dermoscopy.¹⁶

The usual findings in dermoscopy assessment of patchy Alopecia areata include exclamation mark hairs and proximal tapering hairs.¹⁷ Even though there are studies assessing the utility of dermoscopy in Alopecia areata, the correlation of dermoscopic features with the severity of disease has not been looked into by many previous studies on AA. Elucidation of dermoscopic features that are highly correlated with severe disease could help in developing dermoscopic predictors of severe disease or poor prognosis.³ The scarcity of studies on the subject is even more conspicuous on the Indian population. Hence there is a strong need to conduct studies evaluating the correlation between dermoscopy and clinical findings. This may enhance the quality of available evidence on the subject and may aid in evidence-based clinical practice. This is even more essential in resource-poor settings like India, where facilities for advanced and invasive interventions may not be accessible to a large section of the affected patients. Hence the present study was conducted to fill this knowledge gap.

2. MATERIALS AND METHODS

Study Design: A Cross-Sectional Study

Study Area: Skin Outpatient Department Sree Balaji Medical College and Hospital

Study Population: All patients with hair loss, attending skin OPD, who are clinically diagnosed as Alopecia Areata

Study Method: Observational study

Sample Size: 30

Exclusion criteria:

- Not consenting for the study.
- Patient not willing for investigations.

Inclusion criteria:

- Consenting for the study.
- The recruited patients were subjected to the following,

(A) Full History Taking

(A) Thorough General

Dermatological Examination

(B) Dermoscopic examination

DOES THIS STUDY INCLUDE HUMAN SUBJECTS OR ANIMALS?

Yes-Human subjects

ARE PATIENTS INVOLVED IN THIS STUDY?

Yes

ARE STUDENTS SUBJECTS OF THIS STUDY?

No

IS THE PROCEDURE INVASIVE? DETAILS ABOUT THE BIOLOGICAL SAMPLES?

No

RISKS OF THE PROCEDURE?

Nil

WILL THE SUBJECTS BE INVOLVED IN ANY OTHER EXPERIMENTATION DURING THE STUDY?

No

PERSONAL EXPERIENCE OF THE INVESTIGATOR WITH THE PROPOSED TECHNIQUE

No

Ethical considerations: Study was approved by the institutional human ethics committee.

Informed written consent was obtained from all the study participants, and only those participants

willing to sign the informed consent were included in the study. The risks and benefits involved in the study and the voluntary nature of participation were explained to the participants before obtaining consent. Confidentiality of the study participants was maintained.

Data collection tools: All the relevant parameters were documented in a structured study proforma.

Statistical Methods:

Scalp hair loss was considered as the primary outcome variable. Demographic findings Primary explanatory variable. Age, age group, gender, clinical type, scalp hair loss, body hair loss, nail involvement, duration of disease (in months), scalp occipital, parietal, vertex, temporal, frontal, tapering hair, and the response was considered as another explanatory variable. Descriptive analysis was carried out by the mean and standard deviation for quantitative variables, frequency and proportion for categorical variables. Data was also represented using appropriate diagrams like a bar diagram, pie diagram and box plots.

The association between explanatory variables and yellow dots, block dots, broken hair, short vellus hairs, exclamatory mark hair was assessed by cross-tabulation and comparison of percentages. Odds ratio along with 95% CI are presented. Chi-square test was used to test statistical significance. P value < 0.05 was considered statistically significant. IBM SPSS version 22 was used for statistical analysis.²³

3. Results

A total of 30 people were included in the analysis.

Table 1: Descriptive analysis of age (in the year) in the study population (N=30)

Parameter	Mean \pm SD	Median	Min	Max	95% CI	
					Lower	Upper
Age (in the year)	25.1 \pm 11.75	27.00	3.00	46.00	20.63	29.57

The mean age was 25.1 \pm 11.75 in the study population, the minimum age was 3, and the maximum age was 46 in the study population (95% CI 20.63 to 29.57). (Table 1)

Table 2: Descriptive analysis of age group in the study population (N=30)

Age group	Frequency	Percentages
Up to 9	5	16.7%
10 to 19	2	6.4%
20 to 29	10	33.30%
30 to 39	10	33.30%
40 and above	3	10.00%

Among the study population, 5 (16.7%) participants were aged up to 9 years, 2 (6.4%) participants were aged 10 to 19 years, 10 (33.30%) participants were aged 20 to 29 years, 10 (33.30%) participants were 30 to 39 years and 3 (10%) participants were 40 and above. (Table 2& Figure 3)

Figure 3: Bar chart descriptive analysis of age group in the study population (N=30)

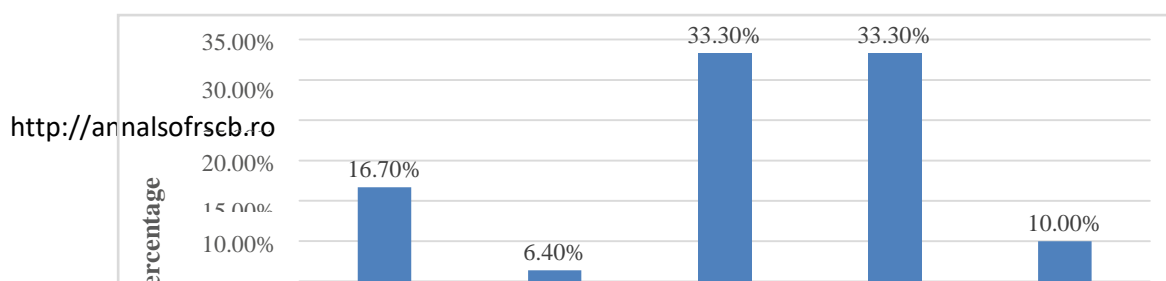


Table 3: Descriptive analysis of gender in the study population (N=30)

Gender	Frequency	Percentages
Male	18	60%
Female	12	40%

Among the study population, 18 (60%) participants were male remaining 12 (40%) were female participants.

(Table 3 & Figure 4)

Figure 4: Pie chart descriptive analysis of gender in the study population (N=30)

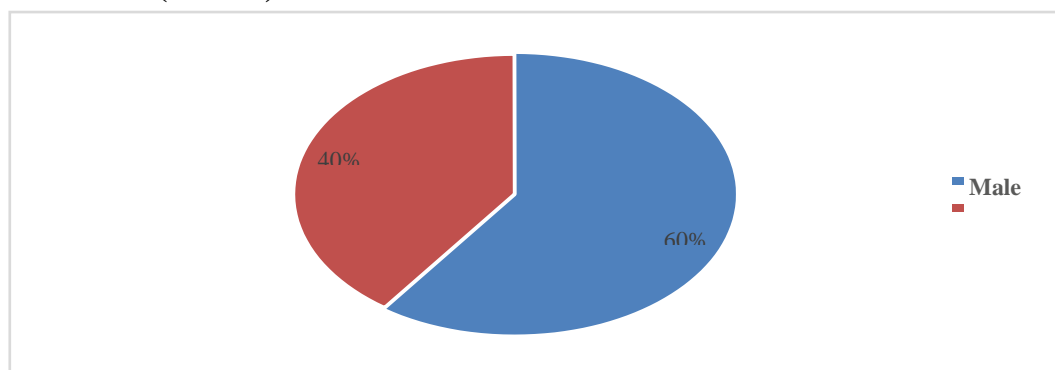


Table 4: Descriptive analysis of clinical types in the study population (N=30)

Clinical types	Frequency	Percentages
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Single Patch	14	46.70%
Multiple Patches	9	30.00%
Diffuse	3	10.00%
Ophiasis	3	10.00%
Alopecia universalist	1	3.30%

Among the study population, 14 (46.70%) participants had a single patch, 9 (30%) participants had multiple patches, 3 (10%) participants had diffused, 3 (10%) participants had ophiasis, and 1 (3.30%) participant had alopecia universalist. (Table 4 & Figure 5)

Figure 5: Bar chart descriptive analysis of clinical types in the study population (N=30)

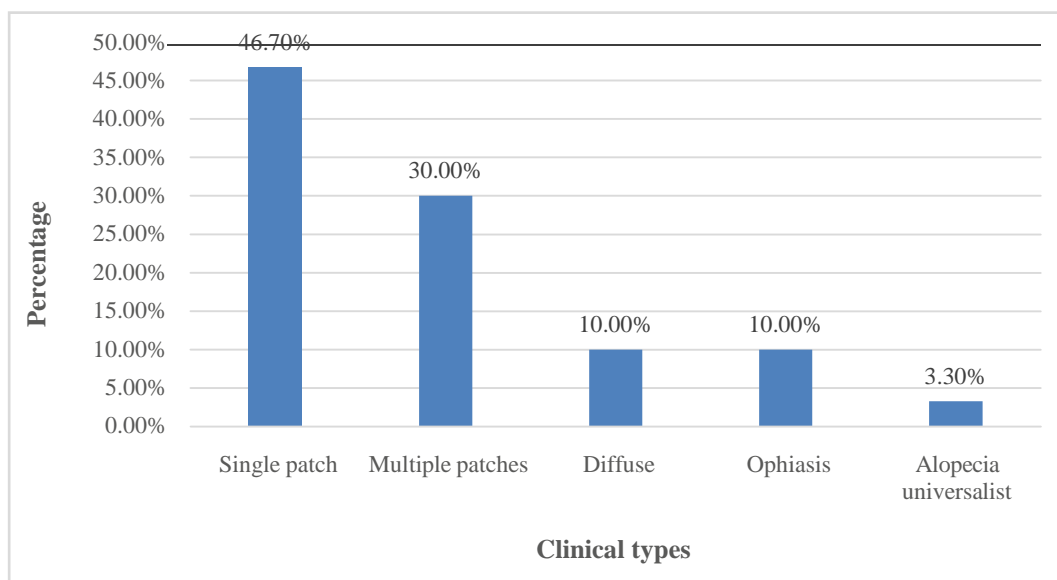


Table 5: Descriptive analysis of scalp hair loss in the study population (N=30)

Scalp hair loss	Frequency	Percentage
S0 (no hair loss)	21	70.00%
S1 (<25% hair loss)	7	23.30%
S3 (51-75% hair loss)	1	3.30%
S5 (100% hair loss)	1	3.30%

Among the study population, 21 (70%) participants had S0, no hair loss, 7 (23.30%) participants had S1, <25% hair loss, 1 (3.30%) participant had S3, 51-75% hair loss and 1 (3.30%) participant had S5, 100% hair loss. (Table 5 & Figure 6)

Figure 6: Bar chart descriptive analysis of scalp hair loss in the study population (N=30)

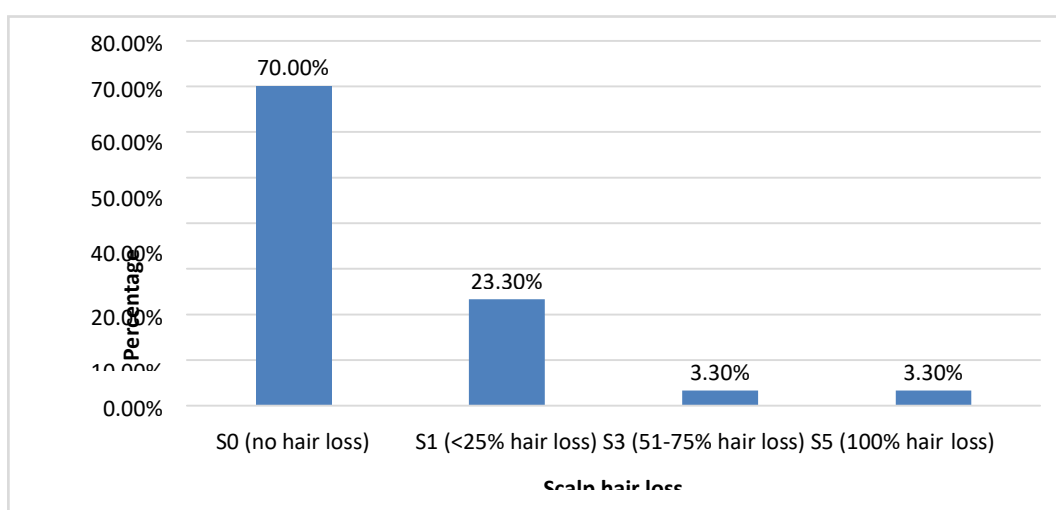


Table 6: Descriptive analysis of body hair loss in the study population (N=30)

Body hair loss	Frequency	Percentages
B0 (no body hair loss)	28	93.30%
B1 (some body hair loss)	2	6.70%

Among the study population, 28 (93.30%) participants had B0, no body hair loss and 2 (6.70%) participants had B1, some body hair loss. (Table 6 & Figure 7)

Figure 7: Pie chart descriptive analysis of body hair loss in the study population (N=30)

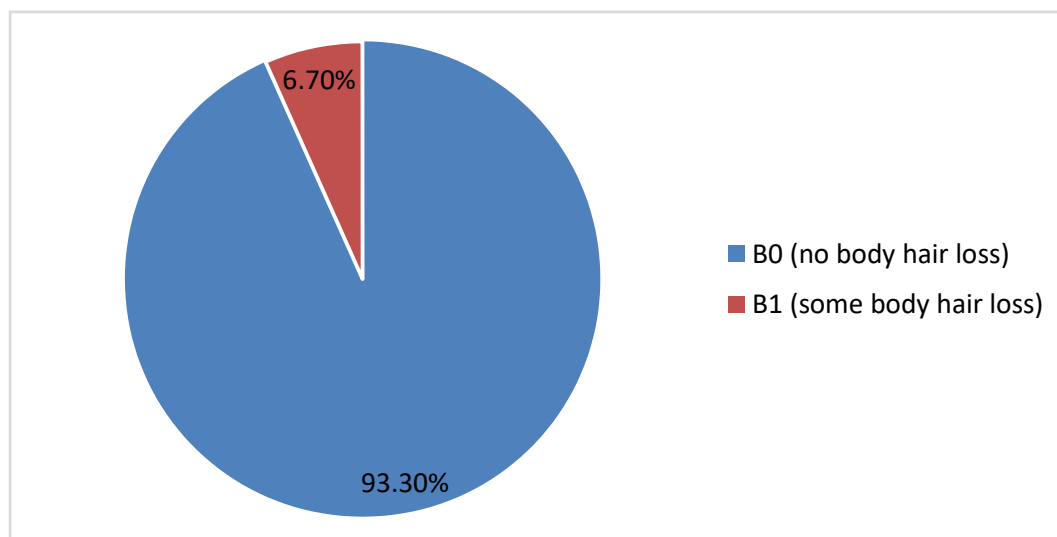


Table 7: Descriptive analysis of nail involvement in the study population (N=30)

Nail involvement	Frequency	Percentages
N0 (No nail involvement)	24	80%
N1 (Some nail involvement)	6	20.00%

Among the study population, 24 (80%) participants had N0 (no nail involvement), and 6 (20%) participants had N1 (Some nail involvement). (Table 7 & Figure 8)

Discussion

Results are seen in the case-control study, conducted by Park J, et al 17 and Shim WH, et al.14 among alopecia patients reported, the mean age was 34.0 ± 18.2 and 30 years respectively which is higher than our results. Our study results showed that, 5 (16.7%) participants were aged up to 9 years, 2 (6.4%) participants were aged 10 to 19 years, 10 (33.30%) participants were aged 20 to 29 years, 10 (33.30%) participants were 30 to 39 years and 3 (10%) participants were 40 and above (18-23). Among our results it is seen that 90% of the study people belong to age below 40 years only 10% belongs to above 40 years, similar results were shown by SharmaVK, et al,23 in their prospective hospital based study, among 808 patients and concluded that 88% of the study population belonged to below 40 years of age which is similar to our results. Our study results showed that 18 (60%) participants were male remaining 12 (40%) participants were female participants. Similar results were seen in Sharma

VK, et al,²³ prospective hospital based study and noticed that chance of development of alopecia areata in males are having a risk of 2 folds more than females, whereas Shim WH, et al,¹⁴ noticed that the development of alopecia areata is more often in females than males (24-25).

Whereas 14 (46.70%) participants had single patch, 9 (30%) participants had multiple patches, totally 76.70% were reported with patches, which is less than 50 per cent in the study conducted by Park J, et al²⁷, in the same study reported 2.8% of the study population were with ophiasis which is similar to our results with 3 (10%) participants, similarly alopecia universalis (7%), which is double than our results with (3.30%), Senila SC, et al⁵⁴ reported that 68.8% of the study population were reported with patches and similarly 8 patients (25%) had alopecia universalis.

Our results concluded that 24 (80%) participants had no nail involvement and 6 (20%) participants had Some nail involvement. Similar results were noticed in Sharma V K., et al²³ in his prospective hospital- based study, reveals that 20% of the study population developed nail involvement.

Among the study population, 10 (33.30%) participants had yellow dots which is one of the symptoms and are reported as a specific finding of Alopecia areata. Similar results are seen in a study conducted by Park J, et al,²⁷ among 327 patients and reported 34.6% of the study population which is close to our results. Whereas Shim WH, et al,¹⁴ in his study concluded that 60% of his study population had yellow dots which is double than our report and reported 80% in a prospective study to evaluate various dermoscopic patterns by Guttikonda A, et al.¹⁰ Black dots are believed to be remnants of hair shafts arising from tapering hairs among the study population, 11 (36.70%) participants had black dots whereas Park J, et al,²⁷ reported as 50.5% which is higher than 1.5 times than our results whereas Shim WH, et al,¹⁴ reported 42% of the study population have black dots, and 58% were reported by Guttikonda, A et al,²⁰ with their team contribution. Broken hair is one of the symptoms for alopecia areata, and Our study results show that 15 (50%) participants had broken hair where a Park J, et al,³⁷ and Guttikonda A, et al,⁵⁰ also concluded that 44.4% and 56% of the study population were reported with broken hair respectively. ShimWH, et al,¹⁴ reported only 2% of patients to have broken hair which is very less than compared to others.

Among the study population, 11 (36.70%) participants had short vellus hair whereas Park J, et al²³ and Shim WH et al.¹⁴ reported 59.8% and 48% respectively which is slightly higher than ours, whereas Guttikonda A, et al,¹⁰ reported 66% that seems to be double than our reports. Among the study population, 7 (23.30%) participants had exclamation mark hairs, which could not be observed with the naked eye; however, their characteristic feature of the hair shaft towards the hair follicle is more readily perceived with dermoscopy (25). Under the dermoscopy which is one of the major findings for the diagnosis of alopecia areata a similar study was conducted by a Park J, et al, ²² revealed that 41% of his study population was reported with Exclamatory mark hair. Among the study population, 21 (70%) participants had S0 (no hair loss), 7 (23.30%) participants had S1 (<25% hair loss), 1 (3.30%) participant had S3 (51- 75% hair loss) and 1 (3.30%) participant had S5(100% hair loss). The proportion of subjects showing B1 (Some body hair loss) was 6.70%, and 20% had some nail involvement. The mean duration of disease was 4.45 ± 11.75 months in the study population, the minimum duration was two months, and the maximum duration was 12 months in the study population. Among the study

population, 12 (40%) participants had occipital involvement. And 18 (60%) participants who never had occipital involvement. Among the study population, 16 (53.30%) participants had the parietal area and 14 (46.70%) participants who never had the parietal area. Among the study population, 10 (33.30%) participants had vertex area 50% of the participants had temporal area and 8 (26.70%) participants had a frontal distribution of the disease. Among the study population, 10 (33.30%) participants had yellow dots and 20 (66.70%) participants had no yellow dots. Among the study population, 11 (36.70%) participants had black dots and 19 (63.30%) participants had no black dots. Among the study population, 50% of the participants had broken hair. Among the study population, 7 (23.30%) participants had exclamatory mark hair. Among the study population, 16 (53.30%) participants had a good response, 9 (30%) participants had a minimal response and 5 (16.70%) participants had a poor response to treatment. In people with yellow dots group, 5 (23.8%) participants never had S0, 3 (42.9%) participants had S1, 1 participant had S3, and 1 participant had S5 (100% hair loss).

4. CONCLUSION

Among the study population, 14 (46.70%) participants had a single patch, 9 (30%) participants had multiple patches, 3 (10%) participants had diffuse patches, 3 (10%) participants had ophiasis, and 1 (3.30%) participant had alopecia universalis. Among the black dots group, 7 (33.3%) participants never had S0 (no hair loss), 3 (42.9%) participants had S1 (<25% hair loss) and 1 (100%) participant had S5 (100% hair loss). Among the broken hairs group, 9 (42.9%) participants never had S0 (no hair loss), 5 (71.4%) participants had S1 (<25% hair loss) and 1 (100%) participant had S3, 51-75% hair loss. Among the short vellus hairs group, 6 (28.6%) participants never had S0 (no hair loss), and 5 (71.4%) participants had S1 (<25% hair loss). Among the exclamatory mark hairs group, 5 (23.8%) participants never had S0 (no hair loss) and 2 (28.6%) participants had S1 (<25% hair loss).

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Ethical approval: The study was approved by the Institutional Ethics Committee

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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REFERENCES

1. Harries MJ, Sun J, Paus R, King LE. Management of alopecia areata. *BMJ*. 2010;341:c3671.
2. Finner AM. Alopecia areata: Clinical presentation, diagnosis, and unusual cases. *Dermatol Ther*. 2011;24(3):348-54.
3. Babu NG, Chandrashekar L, Munisamy M, Thappa DM, Mohanan S. Dermoscopic findings of alopecia areata in dark-skinned individuals: an analysis of 116 cases. *Int J Trichology*. 2014;6(4):156-9.
4. Yesudian P, Thambiah AS. Perinevoid alopecia. An unusual variety of alopecia areata. *Arch*

- Dermatol. 1976;112(10):1432-4.
5. Paus R, Nickoloff BJ, Ito T. A 'hairy' privilege.
 6. Trends Immunol. 2005;26(1):32-40.
 7. Gilhar A, Etzioni A, Paus R. Alopecia areata. N Engl J Med. 2012;366(16):1515-25.
 8. Alkhalifah A, Alsantali A, Wang E, McElwee KJ, Shapiro J. Alopecia areata update: part I. Clinical picture, histopathology, and pathogenesis. J Am Acad Dermatol. 2010;62(2):177-88.
 9. Guo H, Cheng Y, Shapiro J, McElwee K. The Role of Lymphocytes in the Development and Treatment of Alopecia Areata. Expert review of clinical immunology. 2015;11(12):1335-51.
 10. Ito T, Ito N, Saatoff M, Hashizume H, Fukamizu H, Nickoloff BJ, et al. Maintenance of hair follicle immune privilege is linked to prevention of NK cell attack. J Invest Dermatol. 2008;128(5):1196-206.
 11. Gilhar A. Collapse of immune privilege in alopecia areata: coincidental or substantial? J Invest Dermatol. 2010;130(11):2535-7.
 12. Paus R, Slominski A, Czarnecki BM. Is alopecia areata an autoimmune-response against melanogenesis-related proteins, exposed by abnormal MHC class I expression in the anagen hair bulb? Yale J Biol Med. 1993;66(6):541-54.
 13. Gilhar A, Schrum AG, Etzioni A, Waldmann H, Paus R. Alopecia areata: Animal models illuminate autoimmune pathogenesis and novel immunotherapeutic strategies. Autoimmun Rev. 2016;15(7):726-35.
 14. Mane M, Nath AK, Thappa DM. Utility of dermoscopy in alopecia areata. Indian J Dermatol. 2011;56(4):407- 11.
 15. Shim W-H, Jwa S-W, Song M, Kim H-S, Ko H-C, Kim B, et al. Dermoscopic Approach to a Small Round to Oval Hairless Patch on the Scalp 2014. 214-20 p.
 16. Czarnowicki T, He HY, Wen HC, Hashim PW, Nia JK, Malik K, et al. Alopecia areata is characterized by expansion of circulating Th2/Tc2/Th22, within the skin-homing and systemic T-cell populations. Allergy. 2018;73(3):713-23.
 17. Kaliyadan F. The scope of the dermoscope. Indian Dermatol Online J. 2016;7(5):359-63.
 18. Nigam G, Pathak C, Riaz M. Alopecia areata and narcolepsy: a tale of obscure autoimmunity. BMJ Case Rep. 2016;2016.
 19. Suzuki T, Tokura Y, Ito T. Similarities of dermoscopic findings in alopecia areata between human and C3H/HeJ mouse. J Dermatol Sci. 2016;83(2):154-7.
 20. Lie C, Liew CF, Oon HH. Alopecia and the metabolic syndrome. Clin Dermatol. 2018;36(1):54-61.
 21. Seetharam K. Alopecia areata: An update. Indian J Dermatol Venereol Leprol. 2013;79(5):563-75.
 22. Wasserman D, Guzman-Sanchez DA, Scott K, McMichael A. Alopecia areata. Int J Dermatol. 2007;46(2):121-31.
 23. Rodriguez TA, Fernandes KE, Dresser KL, Duvic M. National Alopecia Areata Registry. Concordance rate of alopecia areata in identical twins supports both genetic and environmental factors. J Am Acad Dermatol. 2010;62(3):525-7.
 24. Sharma VK, Dawn G, Kumar B. Profile of alopecia areata in Northern India. Int J Dermatol. 1996;35(1):22-7.
 25. Barahmani N, Schabath MB, Duvic M. National Alopecia Areata Registry. History of atopy or autoimmunity increases risk of alopecia areata. J Am Acad Dermatol. 2009;61(4):581-91.
 26. Leung MC, Sutton CW, Fenton DA, Tobin DJ. Trichohyalin is a potential major autoantigen in human alopecia areata. J Proteome Res. 2010;9(10):5153-63