Original Article

Clinico-Epidemiological Profile of Childhood Alopecia Areata Along with Dermoscopic Correlation: A Cross-Section, Observational Study

Abstract

Background: Childhood alopecia areata (AA) is a common cause of dermatologic consultation; however, data is scarce in the present set-up. **Objectives:** To evaluate the clinico-epidemiological profile of childhood AA along with dermoscopic correlation. **Methods:** We conducted a cross-sectional study including 50 new cases of childhood AA for 1 year. Dermoscopy was performed in each child and findings recorded. **Results:** Childhood AA was more common in girls (M: F 1:1.4), mean age being 11.1 ± 3.7 years. Scalp was commonest site of involvement in 86% cases, while 32 (64%) children had mild disease (<25% involvement). Localized circumscribed patch was the commonest presentation in 37 (74%) children, while sisaipho was the least (2%). A positive family history of AA was noted in 5 (10%) children. Twenty-four children (48%) provided a history of atopic disorders, while 30% had a positive family history of atopy. Stress was the commonest precipitating factor in 13 (26%) subjects. Nail involvement was observed in 19 (38%) children (pitting >thinning), while systemic associations like vitiligo and thyroid dysfunction were present in 26% and 24% cases, respectively. Dermoscopy revealed yellow-dots to be the commonest finding in 44 (88%) cases, followed by short vellus hair and black dots in 76% and 28% children, respectively, while exclamation-mark hair was rare. **Conclusion:** Female gender, younger age, nail involvement, and presence of concomitant atopy, vitiligo, and thyroid dysfunction were associated with severe disease, but not statistically significant ($p > 0.05$). Regression model failed to detect any risk factors for severe AA. Dermoscopy is an important non-invasive tool for evaluating childhood AA.

Keywords: *Alopecia areata, atopy, childhood, dermoscopy*

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Introduction

Alopecia areata (AA) is a relatively common autoimmune disorder manifesting as recurrent episodes of non-scarring hair loss. Despite being a benign condition, it can exert tremendous emotional and psychological impact on the patients and their care-givers.[1]

AA has a variable prevalence ranging from 2.1% in the USA and 2.3% in Saudi Arabia to 0.7% in India.[2] Approximately 1.7%–2% of the general population has a life-time risk of developing AA.[2,3] Pediatric alopecia areata (aged ≤16 years) can account for almost 20% of all cases,[4,5] however, a recent study has shown it to be much lower at 5%.[2] This variety is important because a younger age of onset is an established poor prognostic factor.[5,6]

Several pathogenic factors have been identified like infection, autoimmunity, psychological stress, and genetic predisposition,[2] however, T-cell mediated autoimmune destruction of hair follicles is the most accepted theory.[1,2,7] Additionally several contributory factors may worsen its prognosis like personal/family history of atopy, family history of alopecia areata and other autoimmune disorders including vitiligo, autoimmune thyroid disease or pernicious anemia.[7,8]

Based on extent of involvement, there are 3 principle types of AA- patchy, alopecia totalis and alopecia universalis.[1,2,7] Based on pattern of involvement, other subtypes are-ophiasis, sisaipho, and diffuse form.[1,2] According to Thomas *et al*.,[9] severity of AA may be graded as mild (≤3 patches, localized), moderate (>3 patches, localized) and severe (patches and/or alopecia totalis or universalis). Severe disease and ophiasis pattern are poor prognostic factors.

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Despite being a clinical diagnosis, histopathology is confirmatory in doubtful cases.[3] However, biopsy is difficult in the pediatric population; so, dermoscopy has emerged as a non-invasive confirmatory tool with varying degrees of sensitivity and specificity.[10,11]

Although several clinico-epidemiologic studies of AA exist in adults, data on children are limited,[4] especially in the present set-up. So, we undertook this study to assess the clinico-epidemiologic profile of pediatric alopecia areata along with its dermoscopic correlation.

Materials and Methods

We conducted a prospective, cross-sectional study from January 2019 to December 2019 (12 months) at the Dermatology department of a tertiary care center, Eastern India. We enrolled 50 consecutive children (<16 years) with newly diagnosed alopecia areata (AA), whose parents/guardians provided written informed consent to participate in the study. Any sign of inflammation (scaling/oozing) or scarring alopecia and/or fungal infection served as exclusion criteria. Alopecia areata was diagnosed clinically in all cases followed by its dermoscopic confirmation. In doubtful cases, 10% KOH mount was done to rule out fungal infections. Requisite approval was taken from the Institutional ethical committee and study was conducted in accordance with the Declaration of Helsinki Principles of medical research.

Each patient was subjected to a detailed medical history using fixed proforma to obtain their demographic details, duration of disease, sites affected, precipitating/aggravating factors, associated disease with special emphasis on atopic or endocrine disorders and detailed family history regarding the presence of AA, atopic disorders or endocrine disorders. Subsequently dermatological examination was done to assess the morphological pattern, extent and severity of hair loss, nail involvement and detect other cutaneous disorders like vitiligo, atopic dermatitis, or infections. Based on previous studies[1,12] we graded the severity as mild (<25% involved), moderate (25–50% involved), severe (51–75% involved), and extensive (>75% involvement). Based on pattern and extent of hair loss, AA was classified as patchy, ophiasis, sisaipho, alopecia totalis, and universalis. General survey, systemic examination and serum biochemistry were conducted to rule out endocrine disorders like thyroid dysfunction, diabetes mellitus, and anemia. Non-polarized dermoscopy was done in all children using a Heine® Mini 3000 LED Dermatoscope (Germany) as no parent/guardian consented to skin biopsy. All the findings have been preserved for future use.

Statistical analysis

Data were entered in a Microsoft Excel spread sheet. All entries were double-checked for consistency. All the data obtained have been statistically analyzed using MedCalc®v12.5.0 and preserved for future reference. Mean and standard deviation was used for continuous variables. Normal distribution of numerical variables was determined using the Shapiro-Wilk test. Chi-square test was used for non-parametric data while ANOVA and independent t-test were applied for parametric data. Multivariate regression was used to assess the degree of association between subject parameters and severity of alopecia areata. A "p" value <0.05 has been considered significant.

Results

During our study period (1 year) 1,60,784 patients attended our dermatology OPD, 2860 being patients of alopecia areata (AA). Among them, there were 585 children with alopecia areata (<16 years), while there were 60 new cases of childhood alopecia areata. Of these 60 children, 50 fulfilled our inclusion and exclusion criteria and were enrolled in our study. Thus, we recorded a prevalence of 20.5% (585/2860) for pediatric alopecia areata in our set-up.

Among the 50 children with newly diagnosed alopecia areata, mean (SD) age was 11.1 (3.7) years [range 3–16 years]. Girls [29 (58%)] outnumbered boys [21 (42%)], with a male: female ratio of 1:1.4. Most of the children (60%) belonged to 11–16 year age group, while only 6 (12%) children were below 5 years. Most of our patients [30 (60%)] (males: 12, females: 18) presented within 6 months of developing the disease, while the disease persisted for >2 years in 3 (6%) patients [Table 1]. Eleven (22%) patients were from a low socio-economic status, while 46 (92%) children had illiterate parents.

Regarding associated diseases, atopic disorders (atopic dermatitis, bronchial asthma, and allergic rhinitis) were the commonest, present in 24 (48%) children (male: 8, female: 16), while vitiligo and thyroid dysfunction (hypothyroidism >hyperthyroidism) were recorded in 26% and 24% subjects, respectively. Other cutaneous findings included pyoderma (5 patients), scabies, pediculosis, foot-eczema, verruca, dermatophytoses, molluscum contagiosum (2 patients each) and lichen planus, lichen sclerosus et atrophicus, cheilitis and hemangioma (1 patient each), while other systemic co-morbidities included microcytic hypochromic anemia (10,20%) and diabetes mellitus (6,12%). Down's syndrome was present in 1 patient.

A positive family history of AA was obtained in 5 (10%) [Males: 4, female: 1] patients, but was not statistically significant (p = 0.1, Fisher's exact test). This included first-degree relatives in 3 patients (60%) while the remaining 2 (40%) children had multiple family members affected. A positive family history of atopy and vitiligo was noted in 30% and 22% of our patients, respectively. Sixteen (32%) children provided

a positive history of precipitating/aggravating factors including psychological stress in 13 (26%), infection in 2 (4%) [1- urinary tract infection and 1 dental carries] and trauma in 1 (2%). The major causes of stress were school stress, sibling rivalry, hospitalization of family member, and parental conflicts. Eighteen (36%) patients reported self-treatment with indigenous remedies like ayurveda, ginger paste, homeopathic or onion juice before consulting us.

Table 1: Clinico-demographic parameters (*n*=50)

Parameters	Males (*n*=21)‡	Females (*n*=29)‡
Age distribution (years)		
Range	3-16	3-16
Mean (SD)	11.5 (3.6)	10.8 (3.8)
Age group		
≤5 years	3 (14.3)	3 (10.3)
6-10 years	5 (23.8)	9 (31)
≥11 years	13 (61.9)	17 (58.6)
Duration of alopecia areata		
<6 months	12 (57.1)	18 (62.1)
6-12 months	6 (28.6)	5 (17.2)
13-24 months	1 (4.8)	5 (17.2)
>24 months	2 (9.5)	1 (3.4)
Severity/extent of alopecia areata		
Mild (<25%)	14 (66.7)	18 (62.1)
Moderate (25-50%)	6 (28.6)	5 (17.2)
Severe and extensive (≥51%)	1 (4.8)	6 (20.7)
Sites of alopecia areata		
Scalp	15 (71.4)	23 (79.3)
Eyebrow	3 (14.3)	0 (0)
Alopecia totalis	0 (0)	2 (6.9)
Alopecia universalis	0 (0)	2 (6.9)
Both scalp and eyebrow	3 (14.3)	2 (6.9)
Nail involvement		
Absent	12 (57.1)	19 (65.5)
Pitting	4 (19)	7 (24.1)
Thinning	2 (9.5)	2 (6.9)
Others	3 (14.3)	1 (3.4)
Associated diseases		
Atopic disorders	8 (38.1)	16 (55.2)
Vitiligo	6 (28.6)	5 (17.2)
Thyroid dysfunction	6 (28.6)	6 (20.7)
Anaemia	9 (42.8)	1 (3.4)
Family history		
Alopecia areata	4 (19)	1 (3.4)
Atopic disorders	5 (23.8)	10 (34.5)
Vitiligo	4 (19)	7 (24.1)
Thyroid dysfunction	1 (4.8)	1 (3.4)

‡All values expressed in *n* (%), unless otherwise mentioned

Scalp was the commonest site involved in our study (43, 86%) [p < 0.001, Chi-square]; 76% showing isolated scalp involvement while 10% showed additional eyebrow involvement. The disease was restricted to eyebrows in only 3 (6%) cases, all males. Alopecia totalis (involvement of whole scalp) and alopecia universalis (scalp plus other body sites) [Figure 1] was recorded in 2 (4%) patients each, all females. According to the pattern of involvement, 74% (37/50) had circumscribed patchy involvement [Figures 2 and 3] while ophiasis and sisaipho were noted in 16% and 2% patients, respectively. This difference was statistically significant (p < 0.001, Chi-square). In circumscribed and ophiasis pattern females predominated, while a single male presented with sisaipho pattern. Ophiasis pattern showed a significant association with the severity of disease (p = 0.0008, Fisher's test).

AA was mild (area of involvement <25%) in most children [32 (64%)] (M: F 14:18) followed by moderate (area involved 25–50%) in 11 (22%) children (M: F 6:5) while 3 (6%) (M: F 1:2) and 4 (8%) children (all females- 2 each of alopecia totalis and universalis) showed severe (51–74% involved) and extensive (≥75%) involvement, respectively. This difference was statistically significant (p = 0.009, Chi-square).

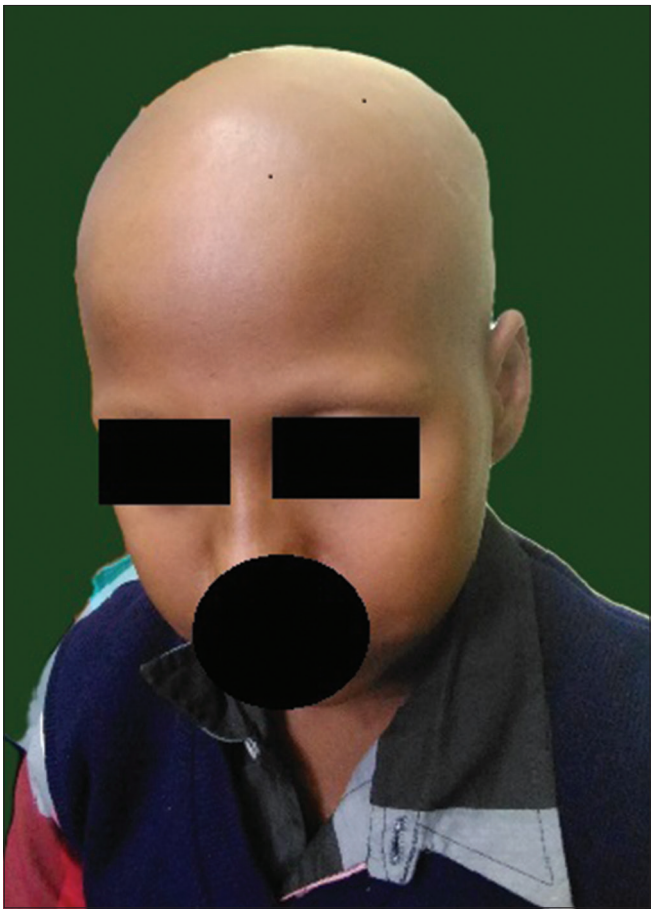

Figure 1: Alopecia universalis

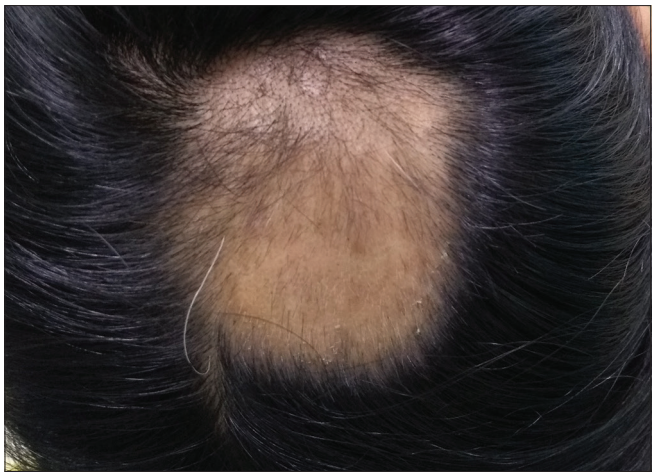

Figure 2: Localized patchy alopecia areata

Nail involvement was present in 19 (38%) children (males: 9, females: 10) but not statistically significant (p = 0.6, Fisher's test). Among them the commonest pathology was pitting (11/19, 57.9%) followed by thinning and other features (21%) each [n = 19]. The later group included leukonychia (n = 1), brownish discoloration (n = 2), and pterygium formation (n = 1) [Table 1].

On correlating disease severity with clinical parameters [Table 2], we observed female gender, atopic disorders, parental illiteracy, low socio-economic status, nail involvement, progression of AA beyond scalp, vitiligo, and thyroid abnormalities (hypo > hyperthyroidism) to be more prevalent in those with moderate/severe/extensive disease (vs. mild disease), however no association was statistically significant (p > 0.05, Fisher's test). We observed a negative correlation between severity of disease and age of patient suggestive of more severe disease in younger patients; however, it was not statistically significant (ρ = - 0.07, *P* = 0.6, Spearman's correlation). Interestingly, history of recurrence, family history of AA and stress were predominant in mild disease, without any statistical significance. Logistic regression model showed none of these factors to be significant predictors of the severity of alopecia areata (p = 0.8).

On dermoscopy, the commonest findings were yellow-dots [YD] (44,88%), short vellus hair [SVH] (38,76%), and black dots [BD] (14,28%) [Figures 4 and 5], while exclamation-mark hair and short broken hair [SBH] were noted in 7 (14%) and 4 (8%) patients, respectively. There was no statistical association between the dermoscopic findings and pattern/severity of alopecia areata in our patients.

Discussion

In our study, we recorded a prevalence rate of 20.4% for pediatric alopecia areata, consistent with most Indian studies (24–26%).[12,13] However, it is higher when compared to previous studies from China (12.8%),[14] Singapore (11.1%),[15] Saudi Arabia (4.2%)[2] and a recent meta-analysis by Lee *et al*. (1.21–2.58%).[16] The variable sample size and ethnic heterogeneity of patients might have contributed to this difference.

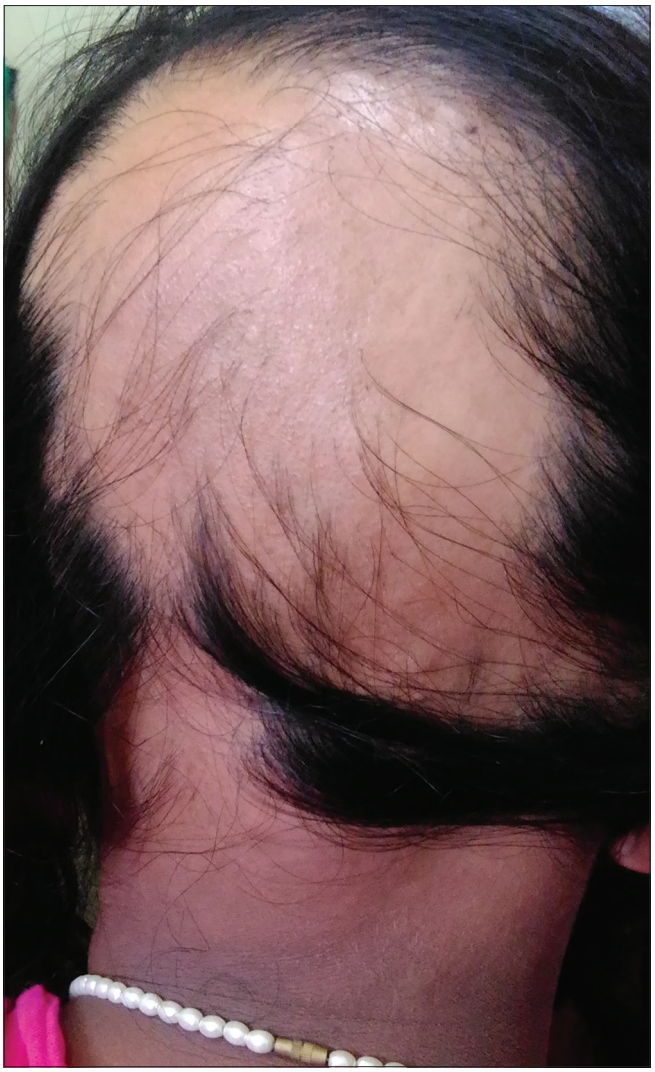

Figure 3: Patchy alopecia areata

In our study, the mean (SD) age of patients was 11.1 (3.7) years, in agreement with previous studies.[4,12,14,15] However, it is slightly higher than studies performed at Kuwait (6.7 years)[5] and Iran (8.9 years).[6]

We observed a female predilection (M: F 1:1.4); consistent with several authors,[4,5,12,13,15] while others recorded a male predominance.[6,14,15] Thus, childhood AA does not have a definite sexual preponderance. We did not find any significant association between gender and severity of AA, contrary to Wohlmuth-Wieser *et al*.[4] and Xiao *et al*.[14] who reported boys to be significantly associated with severe AA, while Tan *et al*.[15] showed increased severity in girls. In the present study 30 (60%) patients presented within 6 months of disease onset, consistent with previous studies.[12,13,15]

Table 2: Clinical parameters and severity/extent of alopecia areata (*n*=50)

Clinical parameters	Extent/severity of alopecia areata		*P*
	Mild (<25% extent) (*n*=32)	Moderate/severe/extensive (*n*=18)	
Gender			0.7
Male	14 (43.8)	7 (38.9)	
Female	18 (56.2)	11 (61.1)	
Atopic disorders			0.08
Present	12 (37.5)	12 (66.7)	
Absent	20 (62.5)	6 (33.3)	
History of recurrent episodes			0.1
Present	5 (15.6)	0 (0)	
Absent	27 (84.4)	18 (100)	
Family history of alopecia areata			0.1
Present	5 (15.6)	0 (0)	
Absent	27 (84.4)	18 (100)	
Site of alopecia areata			0.7
Only scalp	25 (78.1)	13 (72.2)	
Sites other than scalp[ǂ]	7 (21.9)	5 (27.8)	
Nail involvement			0.5
Present[ᵖ]	11 (34.4)	8 (44.4)	
Absent	21 (65.6)	10 (55.6)	
Vitiligo			0.7
Present	7 (21.9)	4 (22.2)	
Absent	25 (78.1)	14 (77.8)	
Thyroid dysfunction			0.09
Present	5 (15.6)	7 (38.9)	
Absent	27 (84.4)	11 (61.1)	
Precipitating factors (stress, infection, trauma etc.)			0.5
Present	2 (6.25)	0 (0)	
Absent	30 (93.8)	18 (100)	

All values presented in n (%) form, P values obtained by Fischer's exact test; [ǂ]includes eyebrow, alopecia totalis, alopecia universalis and involvement of multiple sites; [ᵖ]includes pitting, thinning and their combination.

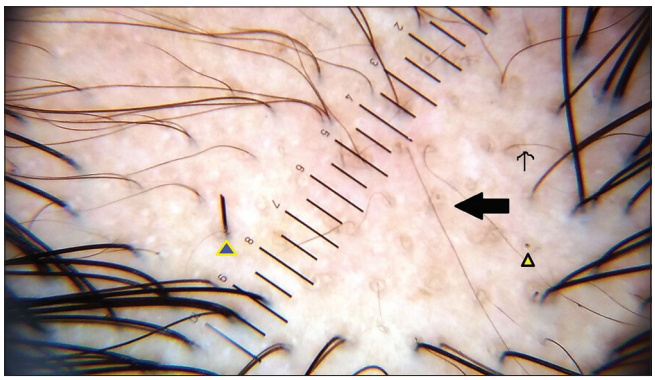

Figure 4: Dermoscopy showing YD (solid-black arrow), SBH (blue arrow-tip), BD (yellow arrow-tip) and SVH (line arrow); 10x, Heine® Mini 3000, Germany [YD – Yellow-dots, SBH- Short-broken hair, BD- Black dot, SVH- Short vellus hair]

We noted associated atopic disorders in 48% patients, while 30% had a family member with atopic disorder. Our finding is in agreement with Aturu *et al*.[12] who recorded atopic

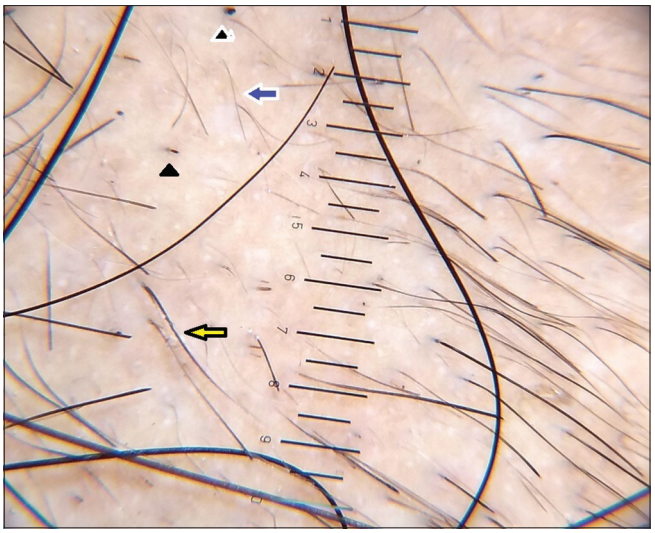

Figure 5: Dermoscopy showing BD (black-arrow tip), SVH (blue arrow) and exclamation-mark hair (yellow hair); 10x, Heine® Mini 3000, Germany [BD- Black dot, SVH- Short vellus hair]

disorders in 44% patients, while it is much higher than studies conducted at Kuwait (24.7%), Chandigarh (17.5%), China (0.88%), and Singapore (0.88%).[4,13-15] Regarding family history of atopy, Aturu *et al*.[12] and Tan *et al*.[15] recorded a lower (17.94%) and similar (33.2%) prevalence respectively. We noted vitiligo and thyroid dysfunction in 26% and 24% patients respectively, higher than other studies.[6,12-15] We failed to find any significant association between associated disorders and severity of AA, consistent with other studies.[6,17]

Family history of AA was present in 5 (10%) patients, comparable to previous studies,[12,14,15] but much lower than an Arab study (51.6%),[5] possibly due to their practice of consanguineous marriages. A positive family history was not related to disease severity, consistent with most authors,[6,18] except Goh *et al*.[19] Vitiligo was most commonly detected in family members of our patients (22%), while other diabetes and hypertension have been reported in other studies.[12,13,15]

Most authors have highlighted psychological stress to precipitate and exacerbate AA in 5–43% patients.[5,6] In the present study, 26% patients reported psychological stress as a precipitating factor; however, there was no association with severity, consistent with an Iranian study.[6] Prior infection was present in 2 (4%) patients (1 each of UTI and dental carries), comparable to Nanda *et al*. (4.7%, all tonsillitis)[5] and slightly lower than Atluru *et al*.[12] (7.7%, all dental carries). However, stress is a subjective phenomenon and more well-designed studies are recommended to address this concern.

Scalp was the commonest site involved in our patients (43, 86%), in agreement with previous studies, ranging from 80 to 100%.[5,6,13-15] Eyebrow was involved in 16%, comparable to Nanda *et al*. (13%)[5] and Farajzadeh *et al*. (10%),[6] but much lower than Kavak *et al*. (2.2%).[18]

Most of our patients [37 (74%)] presented with circumscribed patchy pattern, consistent with previous studies.[5,6,12,14,15] Ophiasis (16%) was significantly associated with severe disease ($p < 0.05$); Atluru *et al*.[12] also reported similar association; however, their prevalence of ophiasis was much low (5.1%). In our cohort, maximum (64%) subjects presented with mild disease (<25% involvement), in agreement with most Asian and Arab studies.[5,6,12-15] We noted extensive involvement (76–100%) [alopecia totalis (AT) and alopecia universalis (AU] in 4 (8%) patients (2 patients each), comparable to studies conducted at China[14] (6.2%) and Iran[6] (2.4%) but lower than other studies (17–31%).[13,20] Western studies have recorded a much higher incidence of extensive AA (>60%)[4,21] probably because they deal only with referred cases at tertiary centers, while in India all patients present to us irrespective of severity.

In our cohort, nails were involved in 19 (38%) patients, consistent with the existing literature (8–40%).[4,5,12,13,15,17] Pitting was our commonest finding in 57.9% patients, in agreement with previous studies (20–90%).[4-6,13,20] Several authors have reported a significant association between nail changes and severity of AA,[5,6,12,13,20]; however we failed to find any significant association similar to Wohlmuth-Weiser *et al*.[4]

Table 3 compares the salient features of different studies concerning childhood alopecia areata.

In the current study, dermoscopy of the alopecic patch revealed yellow dots to be the commonest finding in 44 (88%) cases, followed by short vellus hair and black dots in 76% and 28% cases, respectively. Similar pattern has been reported by Bapu *et al*.[11] and Mane *et al*.,[22] while Jha *et al*.[10] and de Moura *et al*.[23] also noted Yellow-dots to be the commonest finding in 79% and 95% patients, respectively. We observed black dots in only 28% patients, in contrast to Jha *et al*. (70.8%)[10] and Peter *et al*. (75%).[24] Although considered classical, we detected exclamation mark hair in 14% patients, comparable to Bapu *et al*. (20%)[11] and slightly lower than Jha *et al*. (32%).[10] Bapu *et al*. detected short broken hair in 13% cases, similar to us (14%), while Jha *et al*.[10] and Peter *et al*.[24] reported a much high presence of this entity (60–70% cases). Several authors have reported a directly proportional relationship between yellow-dot density per field and severity of AA,[10,11] however, we failed to establish any such association. The higher frequency of yellow-dots and low frequency of black dots or exclamation mark hair in our study are suggestive of less disease activity,[25] which may be explained by more patients in the re-growing phase, thus corroborating the self-limiting nature of childhood alopecia areata.[26]

Limitations

Our major limitations were a small sample size, lack of control group, and a cross-sectional study design. We need future studies with a larger sample size, control group, and longitudinal design, to assess the impact of different clinic-epidemiologic parameters on the prognosis of AA. We were also unable to test for thyroid antibodies to confirm autoimmune thyroiditis due to financial constraints.

Conclusion

To conclude, pediatric alopecia is relatively common in the present set-up; however, most cases are mild showing isolated scalp involvement in the form of localized, circumscribed patches, without any sexual preponderance. Personal history of atopy was detected in 50% of our patients, while one-fourth had associated immunological disorders; this evaluation is critical because they are suggestive of more severe disease. One should also enquire about possible precipitating factors and family history, though they are not related to the severity of

Table 3: Comparison of the salient features of previous studies concerning pediatric alopecia areata

Name of author, place, year	Prevalence of childhood alopecia areata (%)	Most common age group (years); Gender distribution	Severity of disease	Most common clinical presentation; Most common site	Family history of alopecia areata (%)	History of associated disorder (most common)	Nail involvement (%)
Sharma *et al*.,[13] Chandigarh, India, 1996	23.9	6-10 years; Female > male (1.4:1)	Severe AA (total, universal or extensive) → 16.9%	60%- Single patch; Scalp	12.4	17.5% → atopic disorders	30
Nanda *et al*.,[5] Kuwait, 2002	6.7	2-6 years; Girls>boys (2.5:1)	80.5% → mild disease, 13% → extensive (>50% scalp affected)	Localised patch; Scalp	51.6%	20% → Atopic disorders	26.5
Tan *et al*.,[15] Singapore, 2002	11.1	11-15 years; Males > females (1.4:1)	82.4% → limited AA (<50% involvement); AT/AU→2.6%	Localised patch; Scalp	8.4	26.6% → Atopic disorders	8.4
Xiao *et al*.,[14] China, 2006	12.8	11-15 years; Males > females (1.4:1)	84.9% → limited AA (<50% involvement), 8.9% → 50-99% involvement, AT (3.5%) and AU (2.6%)	Localised patch; Scalp	11.1	2.65% → Associated disorders	-
Farajzadeh *et al*.,[6] Iran, 2016	-	6-10 years; Males > females (1.3:1)	84.1% → patchy AA (localized), AT→2.4%, AU→nil	Localised patch; Scalp	21	14% → Eczema	10
Wohlmuth-Wieser *et al*.,[4] USA, 2018	-	6-10 years; Females > males (1.5:1)	37% → lost all scalp hair, 19.4% → upto 75% hair loss, 14.9% → <25% hair loss	Scalp	26.1	32.7% → Atopic dermatitis	43.8
Atluru *et al*.,[12] Chennai, India	26	13-18 years; Female > male (1.4:1)	75% → mild disease (<25% scalp involved), 15.4% → 25-50% hair loss, Opiasis (5.1%), Sisaipho (2.5%), AT (2.5%)	Patchy hair loss; Scalp	7.7	43.6% → Atopic disorders	38.5
Present study, West Bengal, India	20.5	11-16 years; Female>male (1.4:1)	64% → mild disease (<25% scalp involved), 22% → 25% -50% scalp involved, AT→4%, AU→4%	Patchy hair loss; Scalp	10	48% → Atopic disorders	38%

AA- Alopecia areata, USA United States of America, AT alopecia totalis, AU alopecia universalis

disease. Ophiasis pattern and nail involvement must be actively pursued because of their association with severe disease. Dermoscopy is an important non-invasive in-vivo diagnostic tool in this age group, thus dermatologists must be aware of the common dermoscopic findings in this disease. Dearth of similar literature in the present set-up has prompted this study.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

References

1. Achar A, Rathi SK, Kumrah L, Biswas R, Bisai S. Clinico-epidemiological study of alopecia areata. J Pak Assoc Dermatol 2018;28:168-74.
2. Alshahrani AA, Al-Tuwaijri R, Abuoliat ZA, Alyabsi M, AlJasser MI, Alkhodair R. Prevalence and clinical characteristics of alopecia areata at a tertiary care center in Saudi Arabia.

Dermatol Res Pract 2020;2020:1-4. doi: 10.1155/2020/7194270.
3. Raju BT, Rani MS, Vindhya K, Suvarna G. A clinico-epidemiological study on alopecia areata. Indian J Appl Res 2019;9:55-7.
4. Wohlmuth-Wieser I, Osei JS, Norris D, Price V, Hordinsky MK, Christiano A, *et al*. Childhood alopecia areata—data from the National Alopecia Areata Registry. Pediatr Dermatol 2018;35:164-9.
5. Nanda A, Al-Fouzan AS, Al-Hasawi F. Alopecia areata in children: A clinical profile. Pediatr Dermatol 2002;19:482-5.
6. Farajzadeh S, Rahnama Z, Esfandiarpour I, Tardast A, Hasheminasab S, Damavandi FD, *et al*. Clinical and demographic profile of childhood alopecia areata in Iran. J Pak Assoc Dermatol 2016;23:20-7.
7. Pratt CH, King LE Jr, Messenger AG, Christiano AM, Sundberg JP. Alopecia areata. Nat Rev Dis Primers 2017;3:17011. doi: 10.1038/nrdp. 2017.11. PMID: 28300084; PMCID: PMC5573125.
8. Sharma VK, Dawn G, Kumar B. Profile of alopecia areata in Northern India Int J Dermatol 1996;35:22-7.
9. Thomas EA, Kadyan RS. Alopecia areata and autoimmunity: A clinical study. Indian J Dermatol 2008;53:70-4.
10. Jha AK, Udayan UK, Roy PK, Amar AK, Chaudhary RK. Dermoscopy of alopecia areata—A retrospective analysis. Dermatol Pract Concept 2017;7:53-7.
11. Bapu 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;6:156-9.
12. Atluru NC, Gaddam V, Rangarajan S, Muralidhar K, Veeraraghavan M. A clinical profile of childhood alopecia areata in a tertiary care hospital in Chennai, India. Indian J Pediatr Dermatol 2019;20:320-4.
13. Sharma VK, Kumar B, Dawn G. A clinical study of childhood alopecia areata in Chandigarh, India. Pediatr Dermatol 1996;13:372-7.
14. Xiao FL, Yang S, Liu JB, He PP, Yang J, Cui Y, *et al*. The epidemiology of childhood alopecia areata in China: A study of 226 patients. Pediatr Dermatol 2006;23:13-8.
15. Tan E, Tay YK, Giam YC. A clinical study of childhood alopecia areata in Singapore. Pediatr Dermatol 2002;19:298-301.
16. Lee HH, Gwillim E, Patel KR, Hua T, Rastogi S, Ibler E, *et al*. Epidemiology of alopecia areata, ophiasis, totalis, and universalis: A systematic review and meta-analysis. J Am Acad Dermatol 2020;82:675-82.
17. Ejaz A, Jameel K, Suhail M. Pattern and profile of alopecia areata in Pakistan. J Pak Assoc Dermatol 2016;19:136-40.
18. Kavak A, Yeşildal N, Parlak AH, Gökdemir G, Aydoğan İ, Anul H, *et al*. Alopecia areata in Turkey: Demographic and clinical features. J Eur Acad Dermatol 2008;22:977-81.
19. Goh C, Finkel M, Christos PJ, Sinha AA. Profile of 513 patients with alopecia areata: Associations of disease subtypes with atopy, autoimmune disease and positive family history. J Eur Acad Dermatol 2006;20:1055-60.
20. Tosti A, Morelli R, Bardazzi F, Peluso AM. Prevalence of nail abnormalities in children with alopecia areata. Pediatr Dermatol 1994;11:112-5.
21. De Waard-van der Spek FB, Oranje AP, De Raeymaecker DM, Peereboom-Wynia JD. Juvenile versus maturity-onset alopecia areata-a comparative retrospective clinical study. Clin Exp Dermatol 1989;14:429-33.
22. Mane M, Nath AK, Thappa DM. Utility of dermoscopy in alopecia areata. Indian J Dermatol 2011;56:407-10.
23. De Moura LH, Duque-Estrada B, Abraham LS, Barcaui CB, Sodre CT. Dermoscopy findings of alopecia areata in an African-American patient. J Dermatol Case Rep 2008;2:52-4.
24. Peter D, George L, Pulimood SA. Trichoscopic features of various types of alopecia areata in India: Application of a hand-held dermoscope. Aust J Dermatol 2013;54:198-200.
25. Kibar M, Aktan Ş, Lebe B, Bilgin M. Trichoscopic findings in alopecia areata and their relation to disease activity, severity and clinical subtype in Turkish patients. Aust J Dermatol 2015;56:e1-6.
26. Sotiriou E, Patsatsi A, Fotiadou C, Sotiriadis D, Panagiotidou D. Tacrolimus ointment 0.1% in the treatment of active patchy alopecia areata of childhood. Eur J Pediatr Dermatol 2007;17:227-30.