International Journal of Dermatology, Venereology and Leprosy Sciences

E-ISSN: 2664-942X
P-ISSN: 2664-9411
Derma 2019; 2(1): 13-15
Received: 10-11-2018
Accepted: 13-12-2018

Dr. Daniel Lewis
Department of Dermatology, King's College Hospital NHS Foundation Trust, London, UK

Correspondence
Dr. Daniel Lewis
Department of Dermatology, King's College Hospital NHS Foundation Trust, London, UK

Assessment of cases of Alopecia areata- A clinical study

Dr. Daniel Lewis

DOI: https://doi.org/10.33545/26649411.2019.v2.i1a.19

Abstract
Background: Alopecia areata is a common, clinically heterogenous, immune-mediated, non-scarring hair loss disorder. The present study was conducted to assess Alopecia areata (AA) cases in known population.
Materials & Methods: The present study was conducted on 162 patients of both genders. The site was selected and lesion was observed through the dermoscope. The photographs of the lesion were taken and recorded. Grading was done to evaluate extent of scalp hair loss, body hair loss and nail involvement.
Results: Out of 162 patients, males were 92 and females were 70. Pattern was patchy seen in 142, Sub totalis in 14 and ophiasis pattern in 6 patients. The difference was significant ($P< 0.05$). Long ridges were seen in 96, pitting in 32, Beau's lines in 20, Onychorrhexis in 6, Onychodystrophy in 6, Trachyonychia in 2 and other pattern in 2 patients. The difference was significant ($P< 0.05$). YD was seen in 78, TH in 45, BH in 13, BD in 15 and SVH in 11 patients.
Conclusion: Alopecia areata is a common disease affecting hairs and nails. Most common pattern was patchy. Most common nail findings were long ridges.

Keywords: Alopecia areata, hair, Nail

Introduction
Alopecia areata (AA) is a common, clinically heterogenous, immune-mediated, non-scarring hair loss disorder. At any given time approximately 0.2% of world population suffers from Alopecia Areata (AA) with an estimated lifetime risk of 1.7% [1] There is no clear conclusion about whether the disease varies according to sex. There appears to be no significant difference in the incidence of AA between males and females as both formal population studies found none, and hospital-based studies are mixed in citing a female vs male predominance [2].
Alopecia areata (AA) is a common form of non-scarring alopecia involving the scalp and/or body, characterized by hair loss without any clinical inflammatory signs [2]. It is one of the most common form of hair loss seen by dermatologists and accounts for 25% of all the alopecia cases. The exact incidence and prevalence of the disease is not available. It accounts for 2-3% of the new dermatology cases in UK and USA, 3.8% in China, and 0.7% in India [4].
The etiology of AA is considered to be chronic, organ specific autoimmune disease, probably mediated by auto-reactive CD8+ T cells, which affects the hair follicles and sometimes the nails [5]. In AA, CD4+ and CD8+ T-cells violate the immune privilege of the anagen hair follicle, leading to loss of the growing hair shaft. CD8+ T-cells are present in significantly greater quantities than CD4+ cells, and a subset of them known as CD8+ NKG2D+ T-cells has been found both necessary and sufficient to induce AA in C3H/HeJ mice. A predominant Th1 cytokine profile has been discovered at the site of AA lesions [6].
The present study was conducted to assess Alopecia areata (AA) cases in known population.

Materials & Methods
The present study was conducted in the department of Dermatology. It comprised of 162 patients of both genders. The study was approved from institutional ethical committee. All participants were informed regarding the study and written consent was obtained from parents.

Information such as name, age, gender etc. was recorded. The site was selected and lesion was observed through the dermoscope. The photographs of the lesion were taken and recorded. Grading was done to evaluate extent of scalp hair loss, body hair loss and nail involvement. P value less than 0.05 was considered significant.

Results

Table I: Distribution of patients

Total- 162		
Gender	**Males**	**Females**
Number	92	70

Table I shows that out of 162 patients, males were 92 and females were 70.

Table II: Pattern of lesion

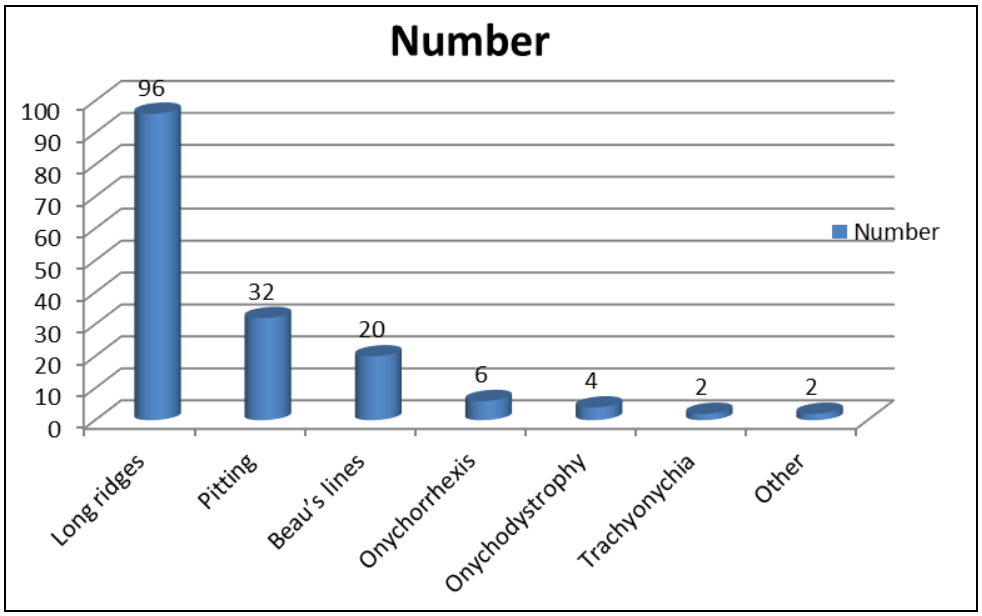
Pattern	Number	P value
Patchy	142	0.01
Sub totalis	14	
Ophiasis	6	

Table II shows that pattern was patchy seen in 142, Sub totalis in 14 and ophiasis pattern in 6 patients. The difference was significant ($P < 0.05$).

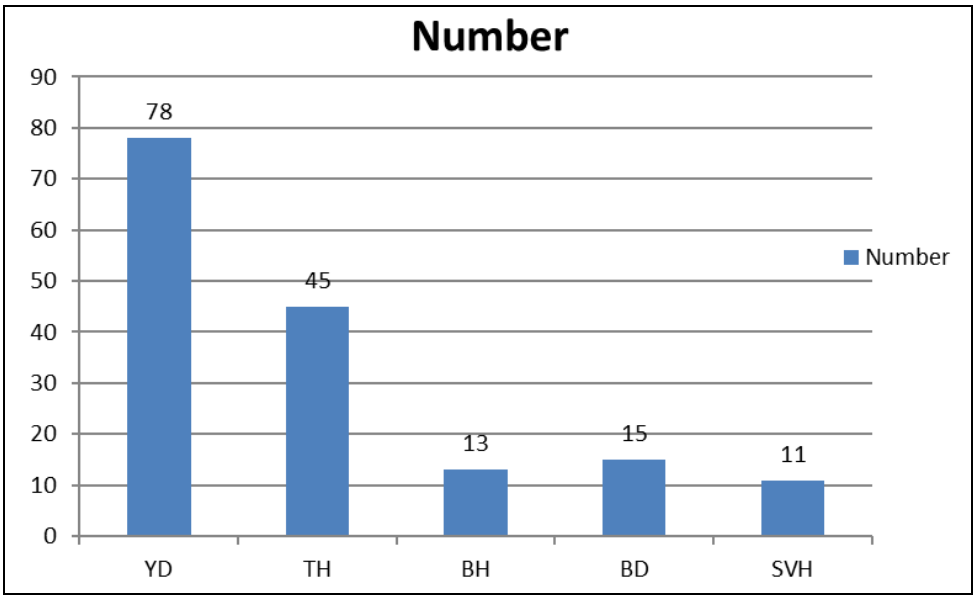
Table III: Nail findings in patients

Nail findings	Number	P value
Long ridges	96	0.01
Pitting	32	
Beau's lines	20	
Onychorrhexis	6	
Onychodystrophy	4	
Trachyonychia	2	
Other	2	

Table III, graph I shows that long ridges were seen in 96, pitting in 32, Beau's lines in 20, Onychorrhexis in 6, Onychodystrophy in 6, Trachyonychia in 2 and other pattern in 2 patients. The difference was significant ($P < 0.05$).



Graph I: Nail findings in patients



Graph II: Assessment of dermoscopic features in patients

Graph II shows that YD was seen in 78, TH in 45, BH in 13, BD in 15 and SVH in 11 patients.

Discussion

The disease manifests as patchy alopecia, reticulate alopecia, ophiasis, ophiasis inversus (sisaphio), alopecia totalis or alopecia universalis. A new subtype of AA is acute diffuse and total alopecia of the female scalp characterized by rapid progression of diffuse alopecia of the female scalp, marked female predominance and a favorable prognosis [7].

The incidence of nail changes in AA is recorded as ranging from 7% to 66%. The nail changes observed include diffuse fine pitting, onychorrhexis, Beau's lines, longitudinal ridging, onychodystrophy and trachyonychia. Gross nail dystrophy is said to be proportional to the degree of hair loss [8]. Marked changes occur more commonly in alopecia totalis and universalis. Nail changes may either precede, accompany or follow the hair loss. They may persist even after hair re-growth. So the presence of nail changes in AA can be considered an indicator of severity of the disease and probably reflects a more refractory disease as compared to patients with absence of nail changes [9]. The present study was conducted to assess Alopecia areata (AA) cases in known population.

In present study, out of 162 patients, males were 92 and females were 70. Pattern was patchy seen in 142, Sub totalis in 14 and ophiasis pattern in 6 patients. Al-Refu *et al.* [10] found that Tinea capitis was the most common, and the trichoscopic features were comma‐shaped hairs, corkscrew hairs, short broken hairs, and interrupted hairs. While in alopecia areata patients, the most specific features were yellow dots and black dots, microexclamation mark, hair shafts with variable thickness, and vellus hairs, with uncommon features included: monilethrix, coiled, zigzag, and tulip hairs. Trichoscopy of trichotillomania showed hair with fraying of ends, breakage at different lengths, short and coiled hairs, and amorphous hair residues. The trichoscopic features of traction alopecia were similar to those of trichotillomania. However, flame hairs and coiled hairs were less common.

We found that long ridges were seen in 96, pitting in 32, Beau's lines in 20, Onychorrhexis in 6, Onychodystrophy in 6, Trachyonychia in 2 and other pattern in 2 patients. YD pattern was seen in 78, TH in 45, BH in 13, BD in 15 and SVH in 11 patients. The initial event in alopecia areata seemed to be a rapid progression of hair follicles from the anagen phase to the catagen and telogen phases. Follicles that were less severely affected remained in anagen but produced dystrophic hair shafts that eventually underwent progression to telogen. Biopsies from the margins of expanding lesions of alopecia areata contained large numbers of follicles in catagen or early telogen. Whether follicles attained telogen via normal catagen transition was not determined. The affected follicles do re-enter the anagen phase [11].

Madani [12] observed that the dermatoscopic features included yellow dots (YDs) in 43 (57.33%) patients, black dots (BDs) in 63 (84%) cases, broken hairs (BHs) in 28 (37.33%) cases, short vellus hair (SVH) in 51 (68%) patients and tapering hair (TH) in 14 (18.67%) cases. The most common dermatoscopic finding observed was BDs, followed by SVHs, YDs, BH and TH.

Conclusion

Alopecia areata is a common disease affecting hairs and nails. Most common pattern was patchy. Most common nail findings were long ridges.

References

1. Rakowska A, Slowinska M, Kowalska‐Oledzka E, Rudnicka L. Trichoscopy in genetic hair shaft abnormalities. J Dermatol Case Rep. 2008; 2:14‐20.
2. Elghblawi E. Tinea capitis in children and trichoscopic criteria. Int J Trichology 2017; 9:47‐9.
3. Bourezane Y, Bourezane Y. Analysis of trichoscopic signs observed in 24 patients presenting tinea capitis: Hypotheses based on physiopathology and proposed new classification. Ann Dermatol Venereol. 2017; 144:490‐6.
4. El‐Taweel AE, El‐Esawy F, Abdel‐Salam O. Different trichoscopic features of tinea capitis and alopecia areata in pediatric patients. Dermatol Res Pract 2014; 2014:848763.
5. Khunkhet S, Vachiramon V, Suchonwanit P. Trichoscopic clues for diagnosis of alopecia areata and trichotillomania in Asians. Int J Dermatol. 2017; 56:161‐5.
6. Gip L, Lodin A, Molin L. Alopecia areata. A follow-up investigation of outpatient material. Acta Derm Venereol. 1969; 49:180-188.
7. Ekiz O, Sen BB, Rifaioğlu EN, Balta I. Trichoscopy in paediatric patients with tinea capitis: A useful method to differentiate from alopecia areata. J Eur Acad Dermatol Venereol. 2014; 28:1255‐8.
8. Xiao FL, Yang S, Liu JB, He PP, Yang J *et al*. The epidemiology of childhood alopecia areata in China: a study of 226 patients. Pediatr Dermatol. 2006; 23:13-18.
9. Sharma VK, Dawn G, Muralidhar S, Kumar B. Nail changes in 100 Indian patients with alopecia areata. J Eur Acad Dermatol Venereol. 1998; 10:189-191.
10. Al-Refu K. Clinical significance of trichoscopy in common causes of hair loss in children: Analysis of 134 cases. Int J Trichol. 2018; 10:154-61.
11. Mane M, Nath AK, Thappa DM. Utility of dermoscopy in alopecia areata. Indian J Dermatol. 2001; 56:407-411.
12. Madani S, Shapiro J. Alopecia areata update. J Am Acad Dermatol. 2000; 42:549-566.