

Study on Clinical and Histopathological Changes of Alopecia Areata

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Abstract

Alopecia areata is a common, unpredictable, non-scarring form of hair loss. It is characterized by rapid and complete loss of hair in one or more round or oval patches, usually on the scalp, bearded area, eyebrows, eyelashes and less commonly on other hairy areas of the body. The present study was undertaken in the department of Dermatology and Venereology of Ibn Sina Medical College, Dhaka from July 2014 to June 2015 to observe the histopathological changes in different stages of alopecia areata. For this purpose 30 patients with age ranged from 18 to 45 years were enrolled. Of them 17 were males and 13 were females. A 4 mm punch biopsy from the involved scalp taken from each patient and histopathological changes were examined. The study revealed that anagen hairs decreased but catagen and telogen hair increased in all stages of alopecia areata.

Telogen hairs increased in acute and chronic stage and catagen hair increased markedly in subacute stage. Although miniaturized (atrophic) follicle was frequently found in chronic stage. It was absent in acute and subacute stages. Moderate to dense peribulbar infiltration of lymphocytes were observed in acute stage and mild to moderate infiltration in subacute stage. In chronic stage either no or mild infiltrations were observed. Peribulbar infiltration of eosinophils and macrophages was seen in all stages of alopecia areata. Thus we can conclude that alopecia areata can be diagnosed with some confidence, even when inflammatory infiltrate is absent, based on increasing numbers of telogen hairs in the acute and subacute stages and increasing miniaturized hairs in chronic stage.

Keywords: Alopecia Areata, Anagen, Catagen, Telogen, Histopathology.

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Introduction

Alopecia areata is a common, unpredictable, non-scarring form of hair loss¹. It is characterized by rapid and complete loss of hair in one or more round or oval patches, usually on the scalp, bearded area, eyebrows, eyelashes and less commonly on other hairy areas of the body².

The incidence of alopecia areata in the United States (Minnesota) is 20.2/100,000 person-years and the lifetime risk is estimated to be approximately 1.7%³. It accounts for about 2 or 3% of new patients presenting at dermatology clinics in the U.K. and U.S.A⁴. It occurs in both sexes in all racial groups. Alopecia areata can occur at any age from birth to the late decades of life. Congenital cases have been reported. Peak incidence appears to 15-29 years. As many as 44% of people with alopecia areata have onset at younger than 20 years. Onset in patients with older than 40 years is seen in fewer than 30% of patients with alopecia areata⁵. The pathophysiology of alopecia areata remains unknown. The most widely accepted hypothesis is that alopecia areata is a T-cell mediated autoimmune condition that is most likely to occur in genetically predisposed individuals⁵.

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In the histological assessment of scalp biopsy specimens from alopecia areata, the diagnostic pathologic feature is peribulbar lymphocytic inflammation ("Swarm of bees") affecting anagen follicles or follicles in early catagen⁶.

The histopathologic features of alopecia areata vary with the stages of the lesion. In the acute stage, a dense or moderate infiltrate develops around the anagen hair bulb. The infiltrate comprises lymphocytes and Langerhans cells, with eosinophils and plasma cells occasionally⁷. The inflammatory assault on anagen follicles induces a premature conversion of catagen. Consequently the number of catagen and telogen follicles found may be marked, approaching 100%. One may also see nanogen hairs, that combine characteristics of both catagen and telogen follicles¹³.

In the subacute stage, an increasingly large numbers of catagen hairs are seen and some telogen hairs will appear after a few weeks as the hair bulb retracts upward in its journey to telogen level, the inflammatory infiltrate may persist in or around follicular stela⁷.

In the chronic stage, there is a reversal of the terminal-vellus ratio and many more miniaturized hairs will be seen at the expense of terminal hairs. The terminal vellus ratio is likely to be 1:1 rather than the usual 7:1. A variable amount of inflammation will be seen and if present, is more likely to be in the papillary dermis around miniaturized hair bulbs than in the reticular dermis or subcutaneous tissue because many terminal hairs have miniaturized and ascend to the upper dermis⁷. The purpose of this cross sectional study is to observe the histopathologic changes of alopecia areata at different stages by means of vertically sectioned scalp biopsy specimens.

Materials and Methods

It was a cross sectional study carried out in Department of Dermatology and Venereology of Ibn Sina Medical College and Department of Pathology of Bangubandhu Sheikh Mujib Medical University from alopecia areata of scalp with or without involvement of other sites. Total 30 samples were included in this study. Data were collected by structured questionnaire. A careful history was taken from each patient regarding the presence of atopy or other autoimmune diseases. A family history of alopecia areata, atopy and autoimmune diseases were also recorded. At the initial visit, one 4-mm punch scalp biopsy specimen was taken from an area of hair loss by application of local anesthetics with aseptic precaution. None has received any treatment for at least 3 months before the biopsy. Biopsy specimens were send in a test tube filled with formalin to the department of Pathology, BSMMU where tissue were processed, section cutting vertically by microtome (2-5 μ m). The haematoxylin and eosin stain are used. This allowed examining the hairs

from the isthmus to the bulbar portion and thereby differentiating anagen, catagen and telogen hairs. Statistical analysis was performed with SPSS (Statistical package of social science) win 16 software packages.

Results

Table -I: Demographic characteristics of the patients.

Characteristics	Frequency	Percent
Age in years		
10-19	4	13.3
20-29	16	53.3
30-39	7	23.3
40-49	3	10.0
Mean \pm SD	27.00 \pm 7.92	
Sex		
Male	17	56.7
Female	13	43.3

Table-II: Distribution of respondents by sites of hair loss.

Site of hair loss	Frequency	Percent
Scalp	30	100.0
Eyebrows	6	20.0
Eye lashes	3	10.0
Other area	0	0.0

Table-III: Exclamation point hair and stages of alopecia areata.

Stages of alopecia areata	Exclamation point hair		Total	P value
	Present	Absent		
Acute	3 (75)#	1 (25)	4 (100)	0.962
Subacute	5 (71.4)	2 (28.6)	7 (100)	
Chronic	13 (68.4)	6 (31.6)	19 (100)	
Total	21 (70)	9 (30)	30 (100)	

Table-IV: Percentage of different types of hair in different stages of alopecia areata.

Stages of AA	Anagen	Catagen	Telogen
	Mean \pm SD	Mean \pm SD	Mean \pm SD
Acute	17.5 (\pm 11.9)	32.5 (\pm 8.7)	50 (\pm 8.2)
Subacute	26.2 (\pm 7.7)	46.7 (\pm 10)	27.14 (\pm 14.7)
Chronic	24.7 (\pm 17.8)	34.2 (\pm 25.3)	41.1 (\pm 30.5)

Table-V: Miniaturization of hair follicles.

Stages of alopecia areata	Miniaturization of hair follicles		Total	P value
	Present	Absent		
	0 (0)#	4 (100)		
Acute	0 (0)	7 (100)	4 (100)	0.001
Subacute	18 (94.7)	1 (5.3)	7 (100)	
Chronic	18 (60)	12 (40)	19 (100)	
Total			30 (100)	

Table-VI: Peribulbar lymphocytic infiltration.

Stages of alopecia areata	Peribulbar lymphocytic infiltration				Total	P value*
	Nil	Mild	Moderate	Dense		
Acute	0 (.0)	0 (.0)	2 (50)	2 (50)	4 (100)	0.001
Subacute	0 (.0)	2 (28.6)	5 (71.4)	0 (.0)	7 (100)	
Chronic	4 (21.1)	13 (68.4)	2 (10.5)	0 (.0)	19 (100)	
Total	4 (13.3)	15 (50)	9 (30)	2 (6.7)	30 (100)	

Discussion

This study was conducted with a view to see the variations of histopathologic changes in different stages of alopecia areata in the scalp. Thirty patients were enrolled in this study. Among them 17 were male and 13 were female. Mean age of this study participant was 27 years with a standard deviation of 7.92 years with a range of 18 to 45 years.

From the present study it was found that alopecia areata is more common among the age group of 20-29 years. Among respondents 16 (53.3%) were within this group. This finding is comparable with Bolduc⁵. He showed that peak incidence appears to occur from age 15 to 29 years. But differ from Sharma et al⁸. They observed the peak age of onset was 20 to 29 years constituting 32.3% of the total cases.

It was revealed that males (56.7%) were predominant than that of females (43.3%). This result is supported by Sharma et al⁸. They showed that 61.2% patients were male and 38.8% were female in their study.

From this study it was also found that among the respondents students were predominant. Total 53.3% respondents were student. Due to lack of relevant literature it could not be compared with other study findings. Probably it is due to awareness to seek medical care and also due to the fact that they are usually in the age group 20-29 years.

Although alopecia is commonly seen on the scalp it may occur on other body sites. In the present study it was found eyebrows (20%) and eyelashes (10%) were the site of affected area of alopecia areata along with scalp. Sharma et al (1988) reported eyebrows alopecia in 5.2% of their patients along with scalp alopecia. This result is not consistent with the present study.

Present study revealed that most of the patients, 19 (63.3%) were in the chronic stage. This finding is consistent with the work of Whiting,⁷ they studied 50 patients among them 31 (62%) patients were in chronic stage of alopecia areata.

From the present study it was revealed that 6.7% respondent had autoimmune disease. This finding is not consistent with Whiting⁷. He found that in 20% to 30% of patients were associated with other autoimmune disease.

This study revealed that atopy was associated in 10% of respondents whereas family history of atopy was associated in 6.7%. This finding is consistent with Sharma et al⁹. They found 8.4% for personal atopy and 6.8% for family history of atopy.

From the present study it was revealed that 16.7% respondents had nail involvement. This finding is accord with Drake et al¹⁰. They reported 10% patients of alopecia areata had nail involvement.

A common finding of a patchy alopecia areata is 'exclamation point' hair present at the margin. Present study revealed that exclamation point hair was present in 3 (75%) respondents in acute stage, 5 (71.4%) in subacute stage and 13 (68.4%) in chronic stage. Difference was not found statistically significant ($P>0.05$). Due to scarcity of relevant literature it could not be compared with present study.

The present study revealed that in the acute stage percentage of anagen hair were decreased whereas catagen

and telogen hair were increased. In the subacute stage decreased anagen hairs and markedly increased catagen hair and slightly increased telogen hair. In chronic stage percentage of anagen hair also decreased and increased catagen and telogen hairs were found. These findings were quite consistent with Whiting,⁷ and Sperling & Lupton¹¹, Stefanato¹².

Though miniaturized hair follicles were absent in all acute and subacute cases. It was present in 18 (94.7%) of chronic cases. This observation was found statistically significant ($P<0.05$). This result is supported by Whiting⁷, Stefanato¹². They stated that miniaturized hairs were found in chronic stage of alopecia areata. Sperling and Lupton¹¹ also observed that in longstanding (chronic) stage of disease almost all hairs are miniaturized. The present study is totally accord with their study.

Dense lymphocytic infiltration was only seen in acute 2 (50%) stage whereas moderate infiltration was found in acute (50%), subacute (71.4%) and chronic stage (10.5%). This observation was found statistically significant ($P<0.05$). This result is supported by Whiting⁷ and Ioffreda⁶. They described in acute stage a dense or moderate infiltrate develops around the terminal hair bulb.

Conclusion

Thus we can conclude that alopecia areata can be clinically diagnosed with some confidence, even when inflammatory infiltrate is absent, based on increasing numbers of telogen hairs in the acute and subacute stages and increasing miniaturized hairs in chronic stage. It can be recommended that transverse histologic section of the biopsy specimen may be taken to see the alterations of terminal and vellus hair ratio which is also helpful to differentiate the chronic stage from other stages of alopecia areata.

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References

1. Papadopoulos, AJ., Schwartz, RA., Janniger, CK. Alopecia areata: emerging concepts. *Acta dermatovenerologica*. 2000; 9(3): 1-9.
2. James, WD., Berger, TG., Elston, DM. 'Disease of the skin appendages', *Andrews' Diseases of the skin*. Clinical dermatology. 10th ed. Canada: Saunders Erevier; 2004.
3. Hordinsky, Ericson. Autoimmunity: Alopecia areata. *J Investig Dermatol Symp Proc*. 2004; 9: 73-78.
4. Nutbrown, Hull, SPM., Baker, Cunliffe, Randall VA, et al. Ultrastructural abnormalities in the dermal papillae of both lesional and clinical normal follicles from alopecia areata scalps. *British Journal of Dermatology*. 1996; 135: 204-210.

5. Bolduc, C. 2006, 'Alopecia Areata', eMedicine.com, inc. (online) Available from: <http://www.emedicine.com/derm/htm> (Accessed: 3 November 2006).
6. Ioffreda. Inflammatory diseases of hair follicles, sweat glands and cartilage. In: Elder DE, Elenitsas R, Johnson BL, editors. *Lever's Histopathology of the skin*. 9th ed. Philadelphia: Lippincott Williams Wilkins; 2005: 480-485.
7. Whiting, DA. Histopathologic features of alopecia areata- A new look. *Arch dermatol*. 2003; 139: 1555-1559.
8. Sharma, VK., Kumar, Kaur. Alopecia areata- a clinical study of 250 patients. *Indian J Dermatol Venerol Leprol*. 1996; 54: 132-136.
9. Sharma, VK., Dawn, Kumar. Profile of alopecia areata in northern India. *International Journal of Dermatology*. 1996; 35(1): 22-27.
10. Drake, Ceilley, Cornelison, Dobes, Dorner, Goltz, et al. Guidelines of care for alopecia areata. *Journal of the American Academy of Dermatology*. 1992; 26: 247-250.
11. Sperling, LC., Lupton, GP. Histopathology of non-scarring alopecia. *J Cutan Pathol*. 1995; 22: 97-114.
12. Stefanato CM. Histopathology of alopecia: a clinicopathological approach to diagnosis. *Histopathology*. 2010; 56: 24-38.