Histopathological Diagnosis of Alopecia Clinically Relevant to Alopecia Areata

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ABSTRACT

Objective: To study the histopathological diagnosis of alopecia clinically relevant to AA and to compare the histopathology between acute and chronic AA divided by time to onset at three and six months.

Materials and Methods: We conducted a cross-sectional study of 113 patients with typical manifestation of AA. Two scalp biopsies were done horizontally and vertically to confirm diagnosis. Histological findings of AA in the acute group were subsequently compared with the chronic group.

Results: Of the 113 eligible patients, 109 (96.5%) were pathologically diagnosed with AA. Other diagnoses included lichen planopilaris, lupus panniculitis, and unspecified scarring alopecia. The percentage of terminal telogen hairs in the acute group was significantly higher than the chronic group (mean % anagen: % telogen ratio = 21.2%:78.8% vs. 36.0%:64.0%; $p = 0.016$), while the chronic group had a significantly higher number of follicular streamers (mean ± SD; 2.5 ± 2.2 vs. 3.7 ± 2.6; $p = 0.023$). The number of vellus hairs significantly increased in the acute group at the six-month onset (p = 0.006). The number of nanogen hairs also increased significantly in the chronic group at both the three- and six-month onset ($p = 0.020$ and $p = 0.007$).

Conclusion: Typical manifestations of AA are not always diagnosed as AA. Acute AA has more terminal telogens and vellus hairs, while chronic AA has more follicular streamers and nanogen hairs.

Keywords: Alopecia areata; scarring alopecia; lichen planopilaris; lupus erythematosus panniculitis; hair disorders; histopathology (Siriraj Med J 2023; 75: 138-144)

INTRODUCTION

Alopecia areata (AA) is a common hair loss condition that affects both men and women between the ages of 15 and 29. The incidence of AA is close to 2% of patients who visit dermatology clinics each year, with an estimated prevalence of 0.2%.[1,2] A previous study showed that the incidence rate of AA was higher in Asians at 3.8%, and that 85% of AA patients in Asia developed the disease before the age of 40.[3] The pathogenesis of AA is believed to be an autoimmune disorder caused by an abnormal immune response to hair follicle correlated antigens and genetic factors.[4-6] Interferon-gamma (IFN-γ) is an important inflammatory cytokine that tampers with the normal immune privilege of anagen hair bulbs and causes it to collapse and damage the hair follicles.[4,5] However, an association between AA and atopic diseases,

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metabolic syndrome, *Helicobacter pylori* infection, lupus erythematosus, iron deficiency anemia, thyroid diseases, vitamin D deficiency, and audiologic and ophthalmic abnormalities, and psychological problems have also been reported.[7-11]

The diagnosis of AA is usually based on typical clinical presentations, or well-defined round or ovoid non-scarring alopecia in the hair-baring area of the body, usually on the scalp.[12] The presence of exclamation point hairs provides sufficient discriminatory value to make a proper diagnosis.[12] However, diseases that can cause clinical patches of non-scarring alopecia and broken hairs, such as trichotillomania, secondary syphilis, and tinea capitis, may be confused with AA.[13] Several studies have shown clinical similarity between AA and early lesions of scarring alopecia, such as lupus erythematosus panniculitis and frontal fibrosing alopecia.[14,15] In addition, trichotillomania, telogen effluvium, or androgenetic alopecia can also mimic the diffuse variant of AA.[16,17] In those cases, the clinical presentation can be misleading, resulting in improper diagnosis and treatments. Therefore, a histopathological examination is essential for diagnosing AA, especially in ambiguous cases.

The objective of our study was to determine the diagnosis of alopecia that clinically mimicked AA and to compare the histopathology of AA between the acute stage (disease onset ≤ 3 months) and the chronic stage (disease onset >3 months).

MATERIALS AND METHODS

This cross-sectional study was conducted at the Institute of Dermatology, Bangkok, Thailand, from February 2012 to November 2013. The study protocol was approved by the Institutional Review Board of the Institute of Dermatology and the Department of Medical Services, Ministry of Public Health, Thailand (certification of approval number 011/2012). Patients with clinical presentation of AA on the scalp were eligible for the study if they were 18 or older and allowed two scalp biopsies (horizontal and vertical sections). Patients were excluded if they had androgenetic alopecia, trichotillomania, telogen effluvium, anagen effluvium, tinea capitis, secondary syphilis, and scarring alopecia.

Tissue histopathology

According to the institute's standard of care, all patients with clinical diagnosis of AA underwent a horizontal and vertical section biopsy of the scalp with four millimeters (mm) punch at the advancing edge of an area with active hair loss, recent hair loss, or hair regrowth. Biopsy specimens were embedded in 10% formalin and processed routinely. At least 15 horizontal and 10 vertical sections were cut and stained with hematoxylin and eosin and periodic acid-Schiff (PAS) to exclude fungal elements.

The horizontal sections of all cases were independently examined by two dermatopathologists (PP and PS), and the vertical sections individually also by two dermatopathologists (SJ and PS). For the diagnosis of alopecia areata; all dermatopathologists followed the combination of characteristic histopathologic findings for the diagnosis of alopecia areata as followed;

1. Non-scarring alopecia with normal or nearly normal number of hair follicles
2. Peribulbar lymphocytic infiltrate (with occasional eosinophils)
3. Increased number of terminal catagen and telogen hairs equal or greater than 50% of total hairs (or anagen-to-telogen ratio equal or less than 1:1)
4. Presence of miniaturized (nanogen) hairs, pigmented hair casts, melanin pigment deposits in fibrous tracts

Any disagreements were resolved via discussion among the authors. The histopathological findings of each case were recorded whether AA was present in the vertical or horizontal section. Other histologic features in the horizontal sections were recorded as follows: 1) the number of follicular units, 2) presence of terminal anagen follicles, 3) presence of terminal catagen/telogen follicles, 4) presence of total terminal follicles, 5) presence of vellus follicles, 6) presence of total follicles, 7) presence of nanogen follicles, 8) presence of follicular stelae (streamers), 9) presence of pigmented hair casts, 10) lymphocytes around follicular papillae and stelae, and 11) eosinophils around follicular papillae and stelae. Histological findings of patients with AA in the acute group were subsequently compared with those in the chronic group. For the presence of inflammatory infiltrate, fibrosis, and pigmented hair cast; the histologic results were gathered from both vertical and horizontal sections.

Statistical analysis

Categorical variables were expressed as numbers and percentages, while continuous variables were expressed as mean ± SD. The histologic characteristics between the acute and chronic groups were compared using the two-sample t-test for continuous variables and the Fisher's exact test for categorical variables. A two-tailed test with a p-value of <0.05 was considered statistically significant. All statistical calculations were done using STATA version 14.0 (STATA Corp, College Station, TX).

RESULTS

A total of 113 patients were enrolled in the study. Of these, 109 patients (96.5%) were pathologically diagnosed with AA, and four patients (3.5%) were identified as having scarring alopecia. Of these four patients with scarring alopecia, two had lichen planopilaris (1.7%), one of them had lupus erythematosus panniculitis (0.9%), and one had unspecified scarring alopecia (0.9%). Figs 1, 2, 3 and 4 show the clinical presentation and histopathologic findings of the four patients with scarring alopecia.

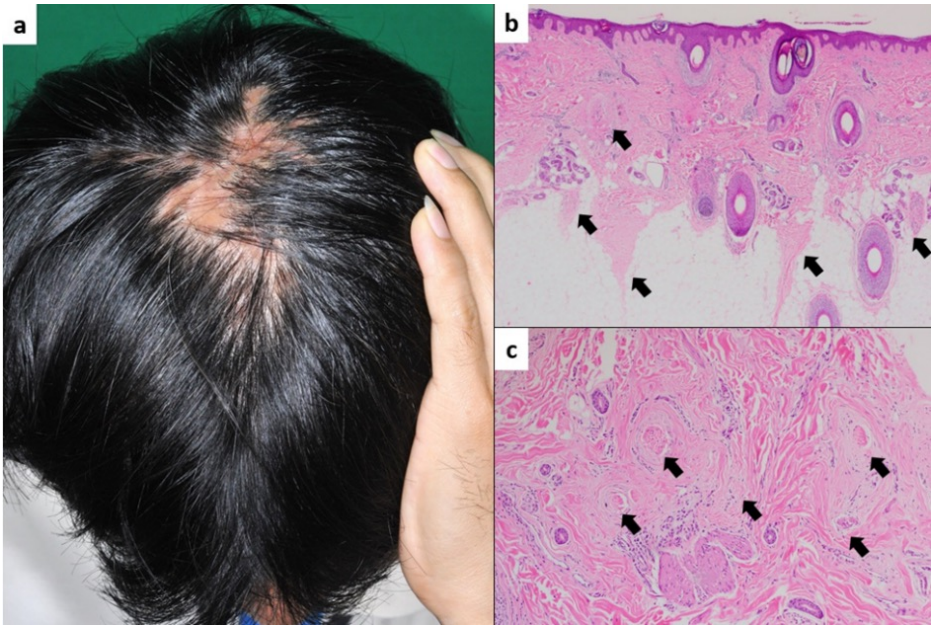

Fig 1. Lichen planopilaris; **(a)** Male who had circumscribed area of hair loss for one year, **(b)** Fibrotic tracts (arrows) in the vertical section, HE x40 magnification, **(c)** Fibrotic tracts (arrows) in the transverse section, HE x100 magnification.

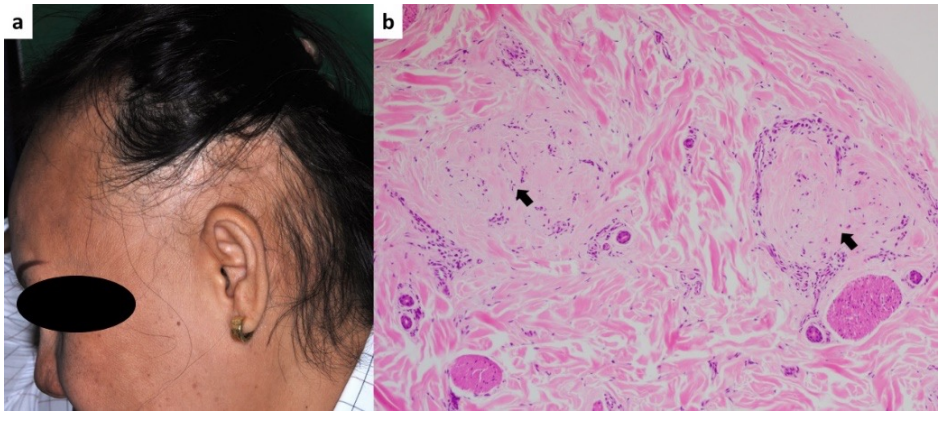

Fig 2. Lichen planopilaris; **(a)** Female who had ophiasis-like alopecia for four years, **(b)**, Fibrotic tracts (arrows), HE x200 magnification

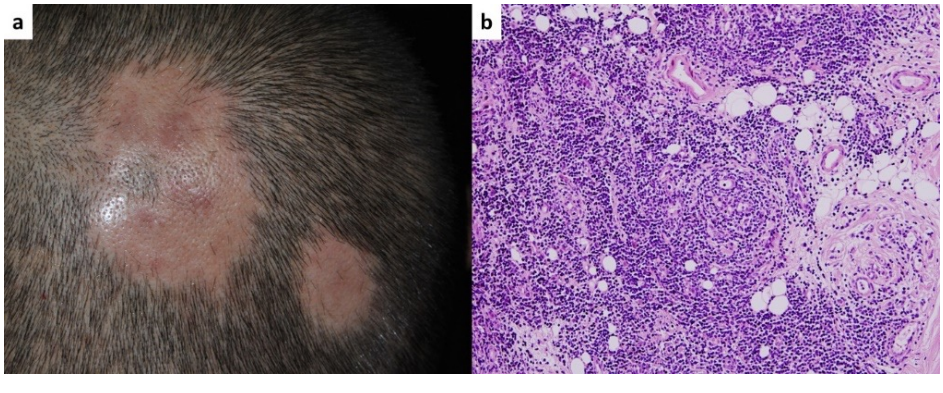

Fig 3. Lupus erythematosus panniculitis; **(a)** Male with multiple patches of alopecia over three months, **(b)** Dense lymphoplasmacytic infiltrates in the subcutaneous fat lobules, HE x200 magnification

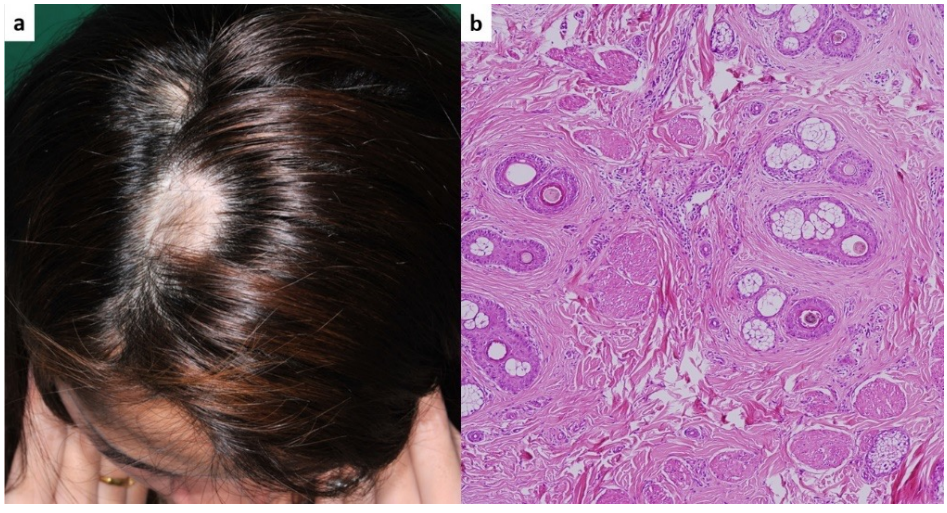

Fig 4. Unspecified scarring alopecia; **(a)** Female who had circumscribed patches of alopecia for two months, **(b)** Intense perifollicular fibrosis; HE x100 magnification

Baseline characteristics of patients with acute AA vs. patients with chronic AA

Patients' demographic data for acute and chronic AA is compared in Table 1. Among the 109 AA patients, 102 had adequate horizontal section tissue for histopathology assessment. There were 54 males and 59 females, with a mean (SD) age of 36 (±11.7) years. Clinical subtypes of AA included patchy AA (95%), alopecia totalis/universalis (3%), and acute diffuse and total AA (2%). All patients were classified by clinical onset of AA. Seventy-six patients (74.5%) were classified as having acute AA as the duration from disease onset was equal to or less than three months. Twenty-six patients (25.5%) were classified as having chronic AA as the duration of disease onset was greater than three months. There were no significant differences between patients with acute and chronic AA for mean age, gender, and alopecia clinical subtypes.

Histologic features of the patients with acute AA vs. chronic AA

Table 2 shows histologic features from horizontal sections of patients with acute AA and patients with chronic AA. The percentage of terminal telogen hairs in the acute group was significantly higher than those in the chronic group ($p = 0.016$). The median anagen to telogen ratio (%) was significantly different between the acute and chronic group (21.2%:78.8% vs. 36.0%:64.0%; $p = 0.016$). However, the median number of follicular stelae or streamers was significantly higher in the chronic group than acute group (3.7 ± 2.6 vs. 2.5 ± 2.2.; $p = 0.023$). Other parameters included the number of terminal anagen hairs, terminal telogen hairs, total terminal hairs, vellus hairs, total hairs, terminal to vellus ratio, total follicular units, lymphocyte, and eosinophilic infiltration, fibrosis, and pigmented hair casts that were not substantially

TABLE 1. Population characteristics of patients with AA.

Characteristics	All patients N = 102	Acute AA N = 76	Chronic AA N = 26	*P* value
Age, year,				
median (range)	35 (18-67)	35 (18-67)	34.5 (22-52)	0.94
mean ± SD	36.3 ± 11.7	36.3 ± 12.5	36.2 ± 9.4	
Sex, N (%)				
Female	54 (52.9)	41 (54)	13 (50)	0.82
Male	48 (47.1)	35 (46)	13 (50)	0.82
Type of AA, N (%)				
Patchy AA	97 (95.1)	73 (96.1)	24 (92.3)	0.07
Alopecia totalis, universalis	3 (2.9)	1 (1.3)	2 (7.7)	0.07
Acute, diffuse, total AA	2 (2.0)	2 (2.6)	0 (0)	0.07

TABLE 2. Comparison of histologic features between acute and chronic AA.

Histologic features	Patients (n = 102)	Acute ≤ 3 months (n = 76)	Chronic > 3 months (n = 26)	*P*- value
Age, years, mean ± SD	36.3 ± 11.7	36.3 ± 12.5	36.2 ± 9.4	0.972
Terminal anagen hairs, mean ± SD	2.0 ± 2.3	1.7 ± 2.1	2.8 ± 2.7	0.027
Terminal telogen hairs, mean ± SD	6.4 ± 4.9	6.5 ± 4.8	6.2 ± 5.3	0.814
Total Terminal hairs, mean ± SD	8.4 ± 5.2	8.2 ± 5.1	9.1 ± 5.7	0.466
Vellus hairs, mean ± SD	9.0 ± 6.1	9.3 ± 6.1	8.3 ± 6.0	0.451
Total hairs, mean ± SD	17.5 ± 5.0	17.6 ± 5.1	17.1 ± 4.7	0.617
Terminal: Vellus ratio, mean ± SD	2.3:1 ± 3.4:1	2.0:1 ± 3.0:1	3.0:1 ± 4.2:1	0.167
%Anagen: %Telogen ratio, mean ± SD	24.9:75.1 ± 26.9:26.9	21.2:78.8 ± 25.8:25.8	36.0:64.0 ± 27.4:27.4	0.016
Follicular units, mean ± SD	8.1 ± 2.0	8.1 ± 1.9	8.3 ± 2.3	0.631
Follicular stelae, mean ± SD	2.8 ± 2.3	2.5 ± 2.2	3.7 ± 2.6	0.023
Eosinophilic infiltration at stelae, upper, mean ± SD	1.5 ± 3.5	1.2 ± 2.0	2.3 ± 6.1	0.160
Nanogen hairs, lower, mean ± SD	0.4 ± 1.4	0.2 ± 0.6	0.9 ± 2.5	0.020

different between the two groups. Although the acute and chronic groups had a similar median number of vellus hairs when categorized at three months (8 and 6.5, respectively; $p = 0.27$), there was an increase of vellus hairs in the group with disease onset before six months and the group with onset after six months ($p = 0.02$) (Table 3). Nanogen hairs increased significantly in the chronic group compared to the acute group at three and six month of disease duration ($p = 0.020$ and $p = 0.007$, respectively) (Tables 2 and 3).

Hair follicles were surrounded by lymphocytes (64.7% at three-month onset and 74.8% at six-month onset) and eosinophils (20.6% at three-month onset and 41.7% at six-month onset) in the lower dermis, but there was no statistically significant difference between the acute and chronic groups ($p > 0.05$).

DISCUSSION

The diagnosis of AA is usually based on characteristics of clinical features. However, clinical assessment alone sometimes is not sufficient to attain accurate diagnosis. Therefore, histopathological assessment is essential in some specific cases. Our study objective was to determine whether clinical evaluation has sufficient specificity for diagnosis of AA. A histological examination was performed to distinguish between AA and other alopecia conditions. Both horizontal and vertical histopathological sections should provide a high diagnostic yield for alopecia.[18] We also assessed differences in histological features between acute and chronic AA.

In our study, 109 out of 113 patients (96.5%) with symptoms resembling AA were diagnosed for it by histological examination. The other four patients (3.5%) were diagnosed with scarring alopecia, such as lichen planopilaris, lupus erythematosus panniculitis, and unspecified scarring alopecia. Sometimes early stage of scarring alopecia is confused with AA when absence of follicular ostia is not noticeable. There were reported cases of lupus erythematosus panniculitis in the patient who presented non-scarring alopecic patches and linear alopecia with clinical features mimicking AA.[14,19,20]

Even though histological examinations can lead to proper diagnosis, it may be impractical to perform scalp biopsies in patients with clinically relevant AA. For clinicians, a thorough history and well-performed physical examination can increase the diagnostic accuracy of AA. In some cases, an absence of follicular ostium with any evidence of changes such as slight erythema, perifollicular scale, epidermal atrophy, and tenderness can lead to other diagnoses of alopecia rather than AA. Dermoscopy allows dermatologists to obtain additional information such as exclamation mark hairs, yellow

TABLE 3. Comparison of histologic features of AA at different onset (before and after six months).

Histologic features	Patients (n = 102)	Onset ≤ 6 months (n = 90)	Onset > 6 months (n = 12)	*P*- value
Age, years, mean ± SD	36.3 ± 11.7	35.8 ± 12.1	39.8 ± 8.0	0.264
Terminal anagen hairs, mean ± SD	2.0 ± 2.3	1.9 ± 2.2	2.7 ± 3.0	0.275
Terminal telogen hairs, mean ± SD	6.4 ± 4.9	6.3 ± 4.7	7.3 ± 6.1	0.538
Total Terminal hairs, mean ± SD	8.4 ± 5.2	8.2 ± 5.0	9.9 ± 6.7	0.296
Vellus hairs, mean ± SD	9.0 ± 6.1	9.4 ± 6.0	6.2 ± 5.9	0.079
Total hairs, mean ± SD	17.5 ± 5.0	17.7 ± 5.0	16.2 ± 5.5	0.328
Terminal: Vellus ratio, mean ± SD	2.3:1 ± 3.4:1	1.9:1 ± 2.9:1	4.7:1 ± 5.5:1	0.006
%Anagen: %Telogen ratio, mean ± SD	24.9:75.1 ± 26.9:26.9	24.3:75.7 ± 26.8:26.8	29.4:70.6 ± 28.1:28.1	0.536
Follicular units, mean ± SD	8.1 ± 2.0	8.2 ± 2.0	7.6 ± 1.6	0.332
Follicular stelae, mean ± SD	2.8 ± 2.3	2.8 ± 2.4	2.9 ± 2.1	0.898
Eosinophilic infiltration at stelae, upper, mean ± SD	1.5 ± 3.5	1.6 ± 3.7	1.1 ± 2.0	0.661
Nanogen hairs, lower, mean ± SD	0.4 ± 1.4	0.2 ± 0.7	1.3 ± 3.5	0.007

dots, black dots and circle hairs to aid in the diagnosis of AA.[21] In equivocal cases, histological examination is necessary for a definitive diagnosis.

The histological features of AA are characteristic of peribulbar infiltrations.[22] When peribulbar infiltrations are notably absent, the diagnosis of AA is difficult. Other histopathologic changes can be beneficial in the diagnosis of AA, such as an increase in catagen and telogen hair follicles, follicular miniaturization, pigment hair cast, nanogen hairs, lymphocytes, and eosinophils as well as melanin in the fibrous tract.[22-26]

A prior study confirmed that disease onset was associated with the number of follicles and degree of inflammation.[22] Peribulbar infiltrations are frequently associated with acute AA rather than chronic AA, whereas an increase of catagen and telogen hair follicles, and hair miniaturization, are associated with sub-acute and chronic AA, respectively.[22,26] This study reveals histopathologic changes of AA regarding acute and chronic stages similar to previous literature[22,26], but we evaluated the change over a specified period at three months and six months. At the three-month cut-off point, there was an increase in terminal telogen hairs in the acute group, which caused a significant decrease in the anagen to telogen ratio. On the other hand, a significant increase in the number of follicular stelae (streamers) and nanogen hairs was noticed in the chronic group, and although e vellus hairs also increased, they were not statistically significant. We can assume that the designated cut-off point at three months for acute and chronic AA may have been too early to detect differences in the number of vellus hairs since increase of vellus hairs in the chronic group was consequentially seen at the sixth-month cut-off point. When patients experience hair loss for a lengthy period of time, the anagen follicles reach the catagen phase, where hair bulbs retract upward toward the isthmus, causing the connective tissue sheath to collapse and become stelae or streamers. This explains why catagen and telogen follicles increase in acute AA, and why follicular stelae are associated with chronic AA. Furthermore, nanogen hairs and small dystrophic follicles, which are unique features of long-standing alopecia areata, also begins to appear more frequently. Peribulbar eosinophils are not seen significantly between the acute and chronic AA, similar to a previous study.[27]

Our study had several strengths. First, all patients in this study underwent both horizontal and vertical scalp biopsies. Second, the histopathological findings were carefully obtained by two independent board-certified dermatopathologists.

However, our study also had limitations. First, some of the histopathological slides of our patients were

inadequate and unreadable, and therefore, some study subjects were excluded. However, only seven patients were excluded due to missing histopathology results. Second, the study was conducted at a specialized dermatology institute in Thailand. Last but not least, generalizability may also have been an issue.

CONCLUSION

Dermatologists have relied on clinical presentation to diagnose AA, but diagnosis s from clinical signs and symptoms may not always rule out other alopecia conditions besides AA. A total of 96.5% of patients with clinical relevance for AA were diagnosed with it through histopathology. Scalp biopsies should be performed in clinically unclear cases. Patients with acute AA have more telogen hairs, whereas those with chronic AA have more follicular stelae, nanogen hairs, and hair miniaturization.

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REFERENCES

1. Safavi K. Prevalence of alopecia areata in the First National Health and Nutrition Examination Survey. Arch Dermatol. 1992;128(5):702.
2. Mirzoyev SA, Schrum AG, Davis MDP, Torgerson RR. Lifetime incidence risk of alopecia areata estimated at 2.1% by Rochester Epidemiology Project, 1990-2009. J Invest Dermatol. 2014; 134(4):1141-2.
3. Tan E, Tay YK, Goh CL, Chin Giam Y. The pattern and profile of alopecia areata in Singapore--a study of 219 Asians. Int J Dermatol. 2002;41(11):748-53.
4. Rajabi F, Drake LA, Senna MM, Rezaei N. Alopecia areata: a review of disease pathogenesis. Br J Dermatol. 2018;179(5):1033-48.
5. Trueb RM, Dias M. Alopecia Areata: a Comprehensive Review of Pathogenesis and Management. Clin Rev Allergy Immunol. 2018;54(1):68-87.
6. McElwee K, Freyschmidt-Paul P, Ziegler A, Happle R, Hoffmann R. Genetic susceptibility and severity of alopecia areata in human and animal models. Eur J Dermatol. 2001;11(1):11-6.
7. Kantor J, Kessler LJ, Brooks DG, Cotsarelis G. Decreased serum ferritin is associated with alopecia in women. J Invest Dermatol. 2003;121(5):985-8.
8. Seyrafi H, Akhiani M, Abbasi H, Mirpour S, Gholamrezanezhad A. Evaluation of the profile of alopecia areata and the prevalence of thyroid function test abnormalities and serum autoantibodies in Iranian patients. BMC Dermatol. 2005;5:11.
9. Chu SY, Chen YJ, Tseng WC, Lin MW, Chen TJ, Hwang CY, et al. Comorbidity profiles among patients with alopecia areata: the importance of onset age, a nationwide population-based study. J Am Acad Dermatol. 2011;65(5):949-56.
10. Aksu Cerman A, Sarikaya Solak S, Kivanc Altunay I. Vitamin D deficiency in alopecia areata. Br J Dermatol. 2014;170(6):1299-304.
11. Lee S, Lee H, Lee CH, Lee WS. Comorbidities in alopecia areata: A systematic review and meta-analysis. J Am Acad Dermatol. 2019;80(2):466-77.e16.
12. Strazzulla LC, Wang EHC, Avila L, Lo Sicco K, Brinster N, Christiano AM, et al. Alopecia areata: Disease characteristics, clinical evaluation, and new perspectives on pathogenesis. J Am Acad Dermatol. 2018;78(1):1-12.
13. Messenger AG, McKillop J, Farrant P, McDonagh AJ, Sladden M. British Association of Dermatologists' guidelines for the management of alopecia areata 2012. Br J Dermatol. 2012;166(5): 916-26.
14. Kossard S. Lupus panniculitis clinically simulating alopecia areata. Australas J Dermatol. 2002;43(3):221-3.
15. Kwong RA, Kossard S. Alopecia areata masquerading as frontal fibrosing alopecia. Australas J Dermatol. 2006;47(1):63-6.
16. Hoss DM, Grant-Kels JM. Diagnosis: alopecia areata or not? Semin Cutan Med Surg. 1999;18(1):84-90.
17. Lew BL, Shin MK, Sim WY. Acute diffuse and total alopecia: A new subtype of alopecia areata with a favorable prognosis. J Am Acad Dermatol. 2009;60(1):85-93.
18. Elston DM. Vertical vs. transverse sections: both are valuable in the evaluation of alopecia. Am J Dermatopathol. 2005;27(4): 353-6.
19. Chen YA, Hsu CK, Lee JY, Yang CC. Linear lupus panniculitis of the scalp presenting as alopecia along Blaschko's lines: a distinct variant of lupus panniculitis in East Asians? J Dermatol. 2012; 39(4):385-8.
20. Chiesa-Fuxench ZC, Kim EJ, Schaffer A, Fett N. Linear lupus panniculitis of the scalp presenting as alopecia along Blaschko's lines: a variant of lupus panniculitis not unique to East Asians. J Dermatol. 2013;40(3):231-2.
21. Miteva M, Tosti A. Hair and scalp dermatoscopy. J Am Acad Dermatol. 2012;67(5):1040-8.
22. Whiting DA. Histopathologic features of alopecia areata: a new look. Arch Dermatol. 2003;139(12):1555-9.
23. Sperling LC, Lupton GP. Histopathology of non-scarring alopecia. J Cutan Pathol. 1995;22(2):97-114.
24. Sperling LC, Cowper SE, Knopp EA. An Atlas of Hair Pathology with Clinical Correlations. 2nd ed. Informa Healthcare; 2012.
25. Elston DM, McCollough ML, Bergfeld WF, Liranzo MO, Heibel M. Eosinophils in fibrous tracts and near hair bulbs: a helpful diagnostic feature of alopecia areata. J Am Acad Dermatol. 1997; 37(1):101-6.
26. Dy LC, Whiting DA. Histopathology of alopecia areata, acute and chronic: Why is it important to the clinician? Dermatol Ther. 2011;24(3):369-74.
27. Yoon TY, Lee DY, Kim YJ, Lee JY, Kim MK. Diagnostic usefulness of a peribulbar eosinophilic infiltrate in alopecia areata. JAMA Dermatol. 2014;150(9):952-6.