ALOPECIA AREATA-A CLINICAL ANALYSIS

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ABSTRACT: Alopecia areata is one of the most emotionally devastating, dermatologic conditions. Alopecia areata is a unique, idiopathic disease in which there is patchy hair loss that is usually confined on the scalp but may occur on beard region, moustache, eyelashes, eyebrows, axilla, genitalia & general body surface. Alopecia totalis a condition if all the hair on the scalp is lost and Alopecia universalis is a condition if in addition to scalp, there is complete loss of body hair.[1]

It occurs equally in both males and females and onset can be at any age, but most often in children & young adults.[2]

The etiology of alopecia areata is not known with certainty. Factors implicated are-autoimmune theory, genetic factors, atopic state & emotional stress.

Alopecia areata progresses as a wave of follicles which enter telogen phase prematurely. It is characterized by non-scarring round and/or oval patches of hair loss. The diagnostic hallmark of alopecia areata is an exclamation mark hair at the active hair margin. The lesions are largely asymptomatic, which may manifest either as alopecia areata classic, reticulate alopecia areata, alopecia totalis/universalis, or ophiasis&ophiasis inverse.[3]

Associated clinical changes are-nail involvement, cataract, vitiligo etc.

Historical Aspects: The distinctive but variable syndrome or syndromes commonly known as alopecia areata (AA) (Pelade in France) have suffered many changes in nomenclature which have added to diagnostic confusion and a bewildering multiplicity of etiological hypothesis.

Cornelius Celsus, who flourished in Rome around 1437, has been credited with the first description of alopecia areata, which even now is sometimes referred to as 'area celsi'. In 1851 Hebra clearly separated alopecia areata from herpes tonsurans, but at first still accepted that AA was of fungal origin. He later revised his opinion (Hebra and Kaposi, 1874). By 1920 most dermatologists had abandoned the parasitic theory of alopecia areata and put forward hypothesis which blended the trophoneurotic and the endocrine theories appropriate to each national temperament. Then came the ogre of focal sepsis, which found favor in many countries since it offered and justified a vigorous therapeutic approach.

In the past 20 years the growing volume of reliable clinical observations has been reviewed in the light of new immunological data and theories. New hypothesis have been propounded, but the historically minded cynic cannot fail to wonder whether their factual basis is any more solid than that of Von Bavensprung'strophoneurotic theory. The account of alopecia areata follows endeavors to separate fact from speculation.[4]

DEFINITION: AA is a relatively common condition characterized by patchy hair loss without atrophy. It may affect any hairy area of the body and is usually reversible. Current evidence indicates

that the hair follicle inflammation in AA is caused by T-cell mediated autoimmune mechanism occurring in genetically predisposed individuals. Environmental factors may be responsible for triggering the disease.

ETIOLOGY:AA is a chronic, organ specific autoimmune disease, probably mediated by auto reactive T cells, which affects hair follicles and sometimes the nails.[5]

Genetic factors: Evidence regarding genetic etiology comes in the form of high frequency of positive family history, that varies from 10%-20%. The genetic predisposition is polygenic in nature.

AA was observed to be associated with a particular HLA haplotype especially DQ3, DR4 and DR11. Earlier onset of AA has been observed with HLADRB*11, especially DRB*1104 allele.[6]

AA is common in Down syndrome,[7] autoimmune polyendocrinopathy-candidiasis-ectodermal dysplasia syndrome (also called autoimmune polyglandular syndrome-1).[8]

Atopy: Several studies have reported an association between AA and atopic disease [9-12], earlier age of onset and more severe than in non-atopic subjects.

Autoimmunity:AA is associated with other autoimmune diseases, such as myxoedema and pernicious anemia.[9, 13] Patients with AA have an increased frequency of circulating organ-specific and non-organ-specific auto antibodies compared with normal subjects, and a variety of non-specific abnormalities in peripheral T-cell numbers and function. Circulating hair follicle specific IgG auto antibodies have been found in patients with AA [14, 15] but their pathogenic role is uncertain. The presence of lymphocytes around and within the hair follicles and the ability to promote hair regrowth with the use of immunosuppressive agents is consistent with an autoimmune hypothesis.[16]

Environmental factors:A variety of environmental factors, including infections, drugs, trauma, and stress, have been suggested as triggering factors of AA, although most patients with AA are unaware of any obvious precipitating factor.

PATHOGENESIS: Injury is severest in the center, leading to sudden precipitation of a group of follicles into telogen. Impaired keratinization and shaft breakage occur towards the periphery in anagen follicles, followed by precipitation into telogen. This results in a broken shaft that apparently continues to grow out because of the upward movement of the follicles in telogen. Such a hair is termed an exclamation mark hair because of the gradual thinning of the broken stub toward the base, which consists of an unpigmentedtelogen club. In the case of less severe injury, the follicles enter telogen prematurely and is shed as a normal club hair, where as the least severely affected follicles continue to produce dystrophic anagen hair. The near normal proportion of anagen hair follicles in the skin biopsies of AA indicates that reentry into anagen stage does occur after a premature telogen. Van Scott showed that the growth of these follicles was arrested in early anagen (anagen IV), following which they return prematurely to telogen. This truncated anagen-telogen cycle is termed as nanogen.[17, 18]

Proposed model of pathogenesis: The group of keratinocytes undergoing early cortical differentiation 2-3 cell layers above the papilla is called the presumptive cortex, and the matrix cells around the upper half of the dermal papilla that that go to form a presumptive cortex is termed the precortical matrix. Degenerative changes in these cells have been observed under light and electron microscope. Therefore Messanger and colleagues proposed that AA is a disease of differentiating cortical keratinocytes leading to defective keratinization, shaft fracture and precipitation of telogen.[19]Telogen is a safe phase since there are no differentiating keratinocytes. After a variable interval the follicle enters anagen but as soon as cortical differentiation starts, anagen is halted and the follicle reenters telogen. The anagentelogen ratio remains constant irrespective of the duration of disease. However, the proportion of vellus hair increases.[18, 20]

PATHOLOGY: Anagen follicles at the margins of expanding patches of AA characteristically show a perifollicular and intrafollicular inflammatory cell infiltrate, concentrated in and around the hair bulb. The inflammatory infiltrate is composed mainly of activated T lymphocytes, with a preponderance of CD4 cells, along with macrophages and Langerhans' cells.[21, 22] Little or none of the inflammatory infiltrate is seen around the isthmus of the hair follicle, the site of hair follicle stem cells.[23] Follicles are smaller than normal and anagen follicles do not develop beyond the Anagen III-IV stage, when the hair shaft starts to be formed.[24] AA causes a disturbance in the normal dynamics of the hair cycle. Anagen follicles are precipitated into telogen. This may occur as a centrifugal wave, reminiscent of a moult wave.[25] Follicles are able to re-enter anagen but, whilst the disease is active, are unable to progress beyond the Anagen III-IV stage.[24] It has been suggested that they then return prematurely to telogen and that these truncated cycles continue until disease activity wanes.[26] The inflammatory infiltrate in AA is concentrated in and around the bulbar region of anagen hair follicles in a "swarm of bees" pattern. The sparing of white hair sometimes seen in AA has also raised the possibility that AA is primarily a disease of hair bulb melanocytes.

CLASSIFICATION OF AA:AA can be classified either based on Ikeda's types or based on the pattern of hair loss.

Ikeda classified AA patients based on associated conditions and course of the disease into following types.[15]

1. Common
2. Atopic
3. Pre hypertensive
4. Autoimmune

Based on the pattern of alopecia, AA can be divided into

A. Restricted to scalp

- Patchy
- Ophiasis
- Sisaphio
- Reticulate
- Diffuse
- Subtotal

- Alopecia totalis
  B. Generalized
- Alopecia universalis

CLINICAL FEATURES:AA can occur on virtually any hair bearing area, but it affects the scalp in approximately 90% of the cases seen in dermatology clinics.[27]The disease can be classified based on the extent or pattern of the hair loss.

The hair loss can present as single delimited patches of hair loss (most common), multiple patches, or extensive hair loss, the disease is classified as follows: patchy AA, in which there is partial loss of scalp hair; alopecia totalis (AT), in which 100% of scalp hair is lost; or alopecia universalis (AU), in which there is 100% loss of all the body hair. Approximately 5% of cases will progress to AT/AU.[28]

The pattern of hair loss observed in AA can vary considerably, and less common presentations can be observed in minority of the cases, including reticular patches of hair loss; ophiasis type, band-like hair loss in parieto-temporp-occipital area; ophiasisinversus (sisaphio), very rare band-like hair loss in the fronto-parieto-temporal area; and diffuse thinning over part or the entire scalp.

Classic AA lesions are well demarcated, round or oval, completely bald, smooth-surfaced patches. The skin within the patch is usually normal on the first examination. At times it can be slightly peachy or reddened[29]color. A characteristic finding that is frequently seen in (or at the border of) the patches is 'exclamation mark hairs'.[27] These are short hair that are tapered proximally and wider distally. In active disease, a hair pull test may be positive at the periphery of lesion. The disease is frequently asymptomatic, although, a few patients report pruritus, burning sensations, or pain before hair loss begins. Close examination of the hair shafts at the edge of the lesions, particularly exclamation mark hair, may reveal subtle defects in the structure and cuticle.[30]

ASSOCIATED ABNORMALITIES: Involvement of the nails in the form of nail pitting can also be seen.[31] Other nail features in AA are trachyonychia, Beaus lines, onychorhexis, thinning or thickening, onychomadesis, koilonychia, punctuate or transverse leukonychia and red spotted lunula.[31, 32]

Autoimmune disease- Thyroid autoimmunity, Vitiligo, Atopy Down's syndrome, autosomal recessive polyglandular syndrome, pernicious anemia, psoriasis, lupus, celiac disease, ulcerative colitis and multiple sclerosis.

There may be high psychiatric morbidity in AA, especially anxiety and mood disturbances.[33]

DIFFERENTIAL DIAGNOSIS: Tineacapitis, Trichotillomania, Telogen effluvium, Lupus and secondary syphilis, Congenitalatrichia

PROGNOSIS: The course of AA is unpredictable. Upto 50% of the patients will recover within 1 year even without treatment. The most important factors indicating a poor prognosis are the extent of hair loss present (extensive AA/AT/AU)[34] or an ophiasis pattern of hair loss, [35]atopy, a positive family history, the presence of other autoimmune diseases, nail involvement, and young age of first onset. In children, the disease may have a tendency towards worsening with time, even if the initial

presentation was mild.[34] In AT/AU, the chance of full recovery is less than 10%.[36]

MATERIAL AND METHODS: 100 Patients attending skin outpatient department were included in the study.

- **Inclusion criteria:**
  1. All patients with circumscribed, bald patch without any signs of inflammation or scarring.
  2. Patients with short, easily extractable broken hair at the margin of a bald patch.
  3. Skin within the bald patch being normal.
  4. Patients above the age of 12 years will be subjected for treatment comparison.

- **Exclusion criteria:**
  1. Patients with re-growing hair.
  2. Patients with secondary infection
  3. After application of some medication.
  4. Patients having scar over the bald patch.

It was a randomized, single blind, study. An informed consent was obtained. Relevant history taken and clinical examination including general, systemic and local examinations were made.
The total number of patches and their measurements were noted in all quadrants of scalp. Presence of exclamatory hairs was noted.

Selected investigations were done only in doubtful cases of AA,

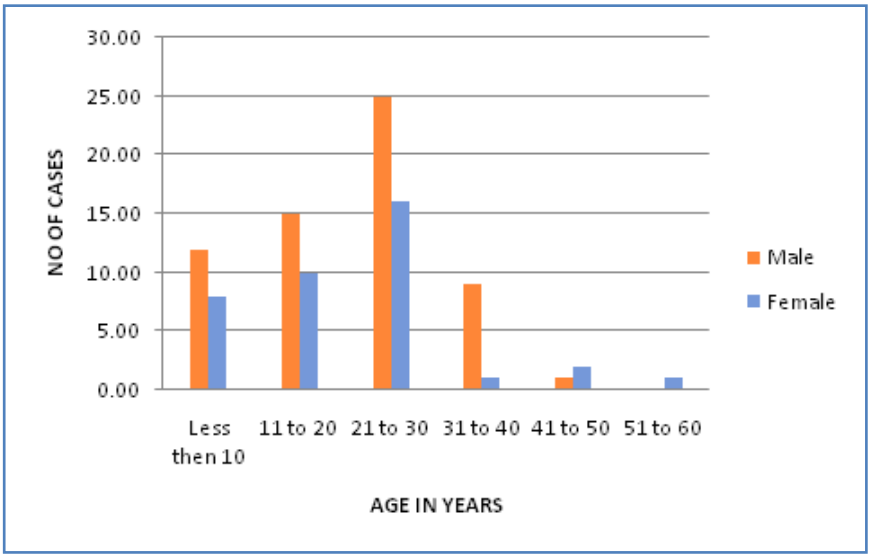
- KOH preparation and fungal culture
- Hair microscopy
- Skin biopsy
- Serology for lupus erythematosus
- Serology for syphilis

RESULTS:

DEMOGRAPHIC DATA:

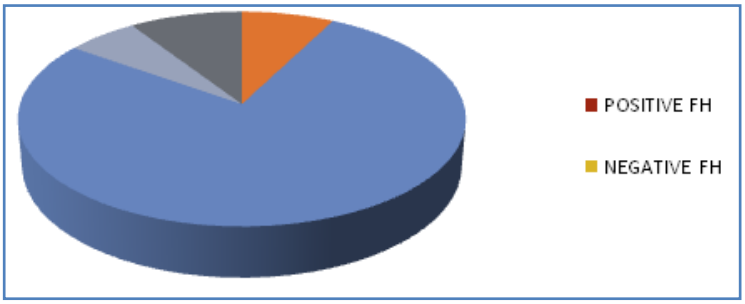
Age(years)	Sex		Total
	Male	Female	
Less than 10	12	8	20
11-20	15	10	25
21-30	25	16	41
31-40	9	1	10
41-50	1	2	3
51-60	0	1	1
Total	**62**	**38**	**100**

Table 1: Age and Sex Distribution



Graph showing age and sex distribution

FAMILY HISTORY OF AA

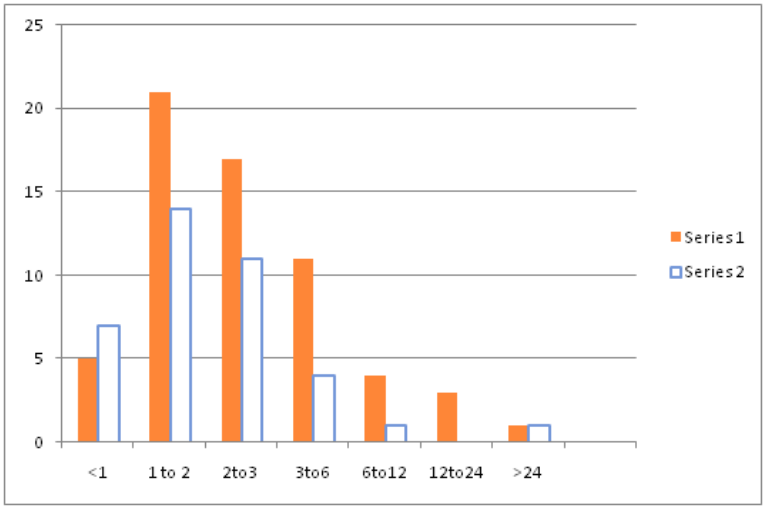


Graph Showing Positive Family History

DURATION OF AA AT PRESENTATION

Duration(months)	Sex		Total
	Male	Female	
<1	5	7	12
1-2	21	14	35
2-3	17	11	28
3-6	11	4	15
6-12	4	1	5
12-24	3	0	3
>24	1	1	2
Total	**62**	**38**	**100**

Table 2: showing duration of AA



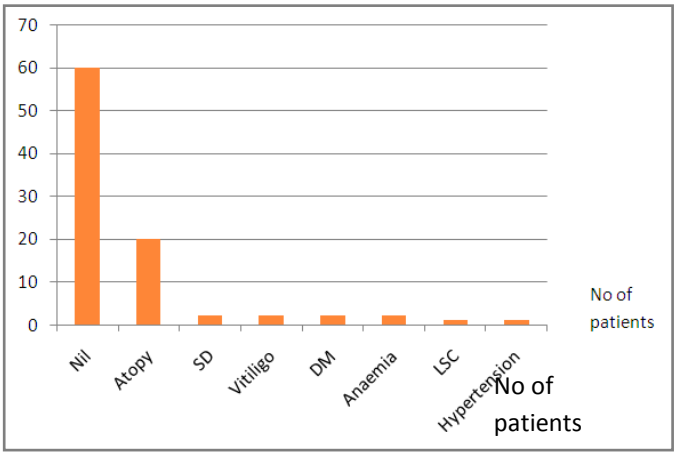
Graph Showing Duration of Alopecia Areata

TYPES OF AA:

Duration(months)	No. of patients		Total
	Male	Female	
Single lesion	37	19	56
Multiple lesions	24	16	40
Alopecia Totalis	1	0	1
Alopecia Universalis	0	2	2
Ophiasis	0	1	1
Total	**62**	**38**	**100**

Table 3 Showing types of AA

ASSOCIATED DISEASES:



Graph showing associated diseases with AA

REGIONAL DISTRIBUTION OF AA

Distribution	No. of cases
Frontal	21
Parietal	41
Vertex	18
Temporal	14
Occipital	45

Table 4 Showing Regional Distribution of AA in Scalp

Distribution	No. of Cases
Face	36
Extremity	07
Trunk	05
Pubic Hair	04
Axilla	02

Table 5 Showing Regional Distribution of AA

Nail Changes	No of Cases
Pitting	10
Longitudinal Ridges	5
Leukonychia	3
Lusterless Nails	3
Total	**21**

Table 6 Nail Changes in AA

DISCUSSION: AA is a unique idiopathic disorder in which there is patchy loss of hair they usually begins on scalp and can affect any hair bearing area of the body.

In the present study, 100 patients attending skin out patient department were included.

In the present study, 86 patients were under the age of 30 years. The maximum no. of patient was in the age group of 21-30 years. Amongst the 100 patients studied, 47 cases were under the age of 20 years.

In the present study the youngest patient was 11 months old and the oldest was 56 years old. In the present study 62 cases were males and 38 females with male to female ratio of 1. 6:1.

Family History of AA: In our study, 9% of patients had a positive family history of AA

In the present study, evidence of atopy was seen in family in 7% of patients of AA. Family history of diabetes mellitus was observed in 8% of the patients, vitiligo in 2%, hypertension in 1% of patient.

History of consanguineous marriage among parents was seen in 33 out of 100 patients.

Personal history: In the present study, personal history of atopy was seen in 4 patients.

Precipitating factors: precipitating factors were observed in 22 patients out of 100 patients (22%).
Duration of AA at Presentation: duration of AA varied from 8 days to 10 years. The duration was less than 1 month in 12 cases, between 1-2 months in 35 cases, 2-3 months in 28 cases, between 3-6 months in 15 cases, between 1-2 years in 3 and above 2 years in 2 patients.
Number of Patches at presentation: In the present study, single lesion was seen in 56 patients, multiple lesions in 40 patients, alopeciatotalis in 1 and alopecia universalis in 2 patients.
Site of affection: Preponderance of the scalp affection was seen in 87% of patients. In the present study face was involved simultaneously on different areas in 25 patients, amongst which beard in 18 patients, moustache in 9 and eyebrows in 8 and eyelashes in 1 patient. In the present study extremity was involved in 8 patients, trunk in 5 and pubic hair in 4 and axilla in 2 patients.
Associated Diseases: Majority of the patients had no associated dermatological or systemic Disease (60%). Atopy was the commonest dermatological disease associated (20%). In the present study 15 patients showed associated conditions. Seborrheic dermatitis was seen in 3 patients. vitiligo, diabetes mellitus, anemia, and pyoderma was observed in 2 patients, lichen simplex chronicus, hypertension, T. corporis was observed in 1 patient each.
Nail Changes: Nail changes were noted in 21% of patients. The commonest nail change being pitting seen in 10 patients.Others like longitudinal ridges in 5 patients, leukonychia and lusterless nails in 3 patients each.
Exclamatory mark hairs: A characteristic finding that is frequently seen in (or at the border of) the patches are “exclamation mark hairs.” These are short hairs that are tapered proximally and wider distally.

In the present study, 12 patients had exclamatory mark hairs with equal sex incidence.

Hair pull test: In active disease, where alopecia patches are expanding, a hair pull test may be positive at the periphery of lesions.

In our study, a positive hair pull test was noted in 12 patients.

CONCLUSION: In a clinical study of alopecia areata including 100 cases the following conclusions were made after analysis of the data.

1. Alopecia areata is a common cause for hair loss in which causes considerable parental anxiety.
2. It is more common in age group 21-30, with male to female ratio being 1. 6:1. In 87% of the patients the first attack was in the patients less than 30 years.
3. Family history of AA was present in 9% of the subjects.
4. The most commonly affected site was scalp being 87%, followed by beard 18, moustache in 9 and eyebrows in 8 and eyelashes in 1 patient, extremity was involved in 8 patient, trunk in 5 and pubic hair in 4 and axilla in 2 patient.
5. Single and multiple lesions accounted for majority of the patients being 96%, alopecia totalis being 1%, alopecia universalis in 2% and ophiasis in 1%.
6. Duration of AA varied from 8 days to 10 years. In majority of the patients the duration was less than 3 months being 75%.
7. Past history of alopecia areata was seen in 9% and atopic dermatitis in 14% cases. Family history of alopecia areata was seen in 9 patients, atopy in 7 and associated conditions in 11 patients, the commonest being diabetes mellitus in 8 patients, vitiligo in 2 patients and hypertension in 1 patient.

8. Nail changes were noted in 21% of patients. The commonest nail change being pitting seen in 10 patient. Others like longitudinal ridges in 5 patient, leukonychia and lusterless nails in 3 patients each.

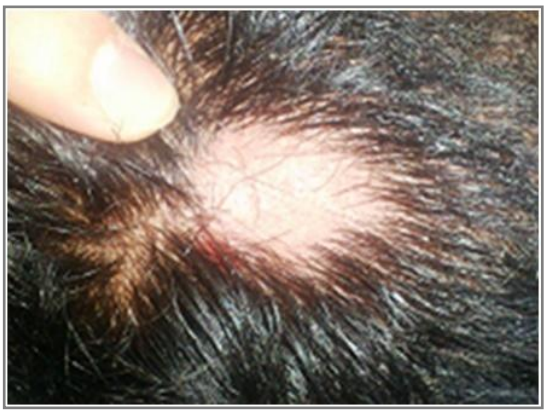


Fig. 16: Single patch of AA

Fig. 17: Multiple patches of AA

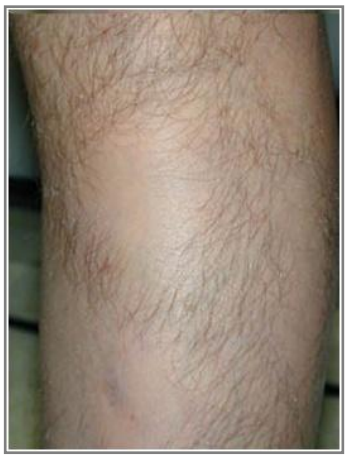


Fig. 20: Alopecia areata-arm

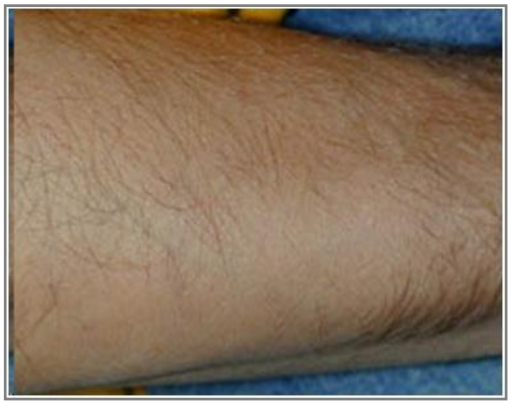


Fig. 21: Alopecia areata-leg

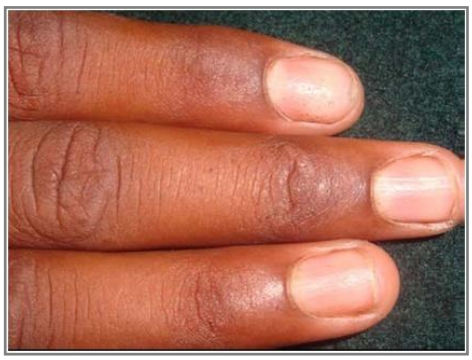


Fig. 23: Nail Pitting

BIBLIOGRAPHY:

1. Bergfled WF. Hair Disorders. In: Moschella S, Hurley H. editors. Dermatology. 3rd ed. Philadelphia:W. B. Saunders; 1992. p. 1545-46.
2. Schwartz RA et al, "ALOPECIA AREATA" cutis, 1997 may; 59(5):238-41.
3. Shegal VN, Shrivastava G, Aggarwal AK, Sheti G, Adhikari T. Alopecia areata in Indian subcontinent Skinmed, 2007;6:639.
4. Arthur Rook. "Diseases of hair and scalp". In :textbook of dermatology. 1st ed. Blackwell scientific; 1982. p. 272-305.
5. Gilhar A, Paus R, Kalish R: Lymphocytes, neuropeptides, and genes involved in alopecia areata. J Clin Invest 111:2019, 2007
6. Marques Da Costa C, Dupont E, Van der Cruys M, et al. Earlier occurrence of sever alopecia in HLA-DRB1*11-positive patients. Dermatology. 2006;213:12-14.
7. duVivier A, Munro DD: Alopecia areata, autoimmunity and Down's syndrome. Br Med J 1:191, 1975
8. Betterle C, Greggio NA, Volpato M: Autoimmune polyglandular syndrome type 1. J ClinEndocrinolMetab 83:1049, 1998
9. Muller SA, Winkelmann RK. Alopecia areata. Arch Dermatol 1963; 88: 290–7.
10. Ikeda T. A new classification of alopecia areata. Dermatologica 1965; 131: 421–45.
11. Penders AJM. Alopecia areata and atopy. Dermatologica 1968; 136: 395–9.
12. Young E, Bruns HM, Berrens L. Alopecia areata and atopy. Dermatologica 1978;156: 306–8.
13. Cunliffe WJ, Hall R, Stevenson CJ et al. Alopecia areata, thyroid disease and autoimmunity. Br J Dermatol 1969; 81: 877–81.
14. Tobin DJ, Orentreich N, Fenton DA et al. Antibodies to hair follicles in alopecia areata. J Invest Dermatol 1994; 102: 721–4.
15. Lu W, Shapiro J, Yu M, Barekatain A, Lo B, Finner A, et al. Alopecia areata: pathogenesis and potential for therapy. Expert review mol med 2006;8:1-19.
16. McElwee KJ, Tobin DJ, Bystryn JC, King LE Jr, Sundberg JP. Alopecia areata: an autoimmune disease? ExpDermatol:1999;8:371-9.
17. VAN scott EJ. Morphologic changes in pilosebaceous units and anagen hairs in alopecia areata. J Invest Dermatol. 1958;31:35-8.
18. Headington JT, Mitchell A, Swanson N. new histopathological finding in alopecia areata studied by transverse section. J Invest Dermatol. 1981;76:325.
19. Messanger AG, Slater DN, Bleehan SS. Alopecia areata:alterations in hair growth cycle and correlation with follicular pathology. Br J Dermatol. 1986;114:337-47.
20. Bleiker TO, Nicolaou N, Traulsen J, Hutchinson PE. "atrophictelogen effluvium" from cytotoxic drugs and a randomized controlled trial to investigate the possible protetive effect of pretreatment with topical vitamin D analogues in humans. Br J Dermatol. 2005;153:103-12.
21. Perret C, Wiesner-Menzel L, Happle R. Immunohistochemical analysis of T-cell subsets in the peribulbar and intrabulbarinfiltrates of alopecia areata. ActaDermVenereol (Stockh) 1984; 64: 26–30.
22. Wiesner-Menzel L, Happle R. Intrabulbar and peribulbar accumulation of dendritic OKT 6-positive cells in alopecia areata. Arch Dermatol Res 1984; 276:333–4.

23. Cotsarelis G, Sun TT, Lavker RM. Label-retaining cells reside in the bulge area of pilosebaceous unit: implications for follicular stem cells, hair cycle, and skin carcinogenesis. Cell 1990; 61: 1329–37.
24. Van Scott EJ. Morphologic changes in pilosebaceous units and anagen hairs in alopecia areata. J Invest Dermatol 1958; 31: 35–43.
25. Eckert J, Church RE, Ebling FJ. The pathogenesis of alopecia areata. Br J Dermatol 1968; 80: 203–10.
26. Messenger AG, Slater DN, Bleehen SS. Alopecia areata: alterations in the hair growth cycle and correlation with the follicular pathology. Br J Dermatol 1986; 114: 337–47.
27. Wasserman D, Gumanz-Sanchez DA, Scott K, McMichael A. alopecia areata. Int J Dermatol. 2007;46:121-31.
28. Price VH. Therapy of Alopecia areata:on the cusp and in future. J InvestigDermatolSympProc 2003;8:207-11.
29. deBerker DAR, Messenger AG, Sinclair RD. Disorders of hair. In; Burns Tony, Breathnach Stephen, Neil Cox, Griffiths Christopher. Editors. Textbook of dermatology. vol 4. 7th ed. Oxford: Blackwell scientific; 2004. p. 63. 1-63. 120.
30. Tobin DJ, Fenton DA, Kendall MD. Ultrastructural study of exclamation mark hair shafts in alopecia areata. J CutanPathol 1990;17:348-54.
31. Gandhi V, Barhuah MC, Bhattacharya SN. Nail changes in alopecia areata: incidence and pattern. Indian J of DermatolVenerolLeprol 2003;69:114-5.
32. Olsen EA, Dunlap FE, Funicella T, Koperski JA, Swinehart JM, Tschen EH, et al. A randomized clinical trial of 5% topical minoxidil versus 2% topical minoxidil and placebo in the treatment of androgenetic alopecia in men. J Am AcadDermatol 2002;47:377-85.
33. MacDonald Hull SP, Wood ML, Hutchinson PE, Sladden M, Messenger AG. Guidelines for the management of alopecia areata. Br J Dermatol 2003;149:692-9.
34. Price VH. Double-blind, placebo-controlled evaluation of topical minoxidil in extensive alopecia areata. J Am AcadDermatol 1987;16:730-6.
35. Lew BL, Shin MK, Sim YW. Acute diffuse and total alopecia. A new subtype of diffuse Alopecia areata with a favourable prognosis. J Am AcadDermatol 2009;60:85-93.
36. Fiedler-Weiss VC. Topical minoxidil solution (1% and 5%) in the treatment of alopecia areata. J Am AcadDermatol 1987;16:745-8.

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