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Clinico-Epidemiological Profile and Therapeutic Response of Alopecia Areata in A Tertiary Care Teaching Hospital

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Abstract

Background: Alopecia areata is a common disorder of hair occurring on any hair bearing site of the body and having a great psychosocial impact.

Aims: To assess the demographic pattern, clinical aspects, associations and therapeutic response of AA in a tertiary care teaching hospital.

Materials & methods: All patients diagnosed with alopecia areata were included in the study. With due consent, they were subjected to detailed history taking, physical examination, routine blood and radiological investigations and histopathological examination in doubtful cases. After confirmation of the diagnosis, they were subjected to two modes of therapy, oral mini pulse (OMP) steroid therapy in patients having diffuse involvement and intralesional steroid therapy in patients having localized involvement.**Results:** Age of onset was < 40 years in 97% of patients with a male predominance. Alopecia areata was the commonest (88%) variant noted, while alopecia totalis (1%), alopecia universalis (6%) and ophiasis pattern (5%) were also identified. Scalp (58.3%) was the commonest site involved followed by face (24%) and multiple sites (16%). Other associated autoimmune diseases were atopy (10%), lichen planus (3%) and vitiligo (2%). 72.2% of patients treated with intralesional steroid and 60% of patients treated with oral pulse steroid showed marked improvement.

Conclusion: Alopecia areata is a disease of the young. Poor prognostic factors are extensive involvement, early age of onset, and positive family history in first degree relatives. Mild localized disease can be managed with intralesional steroid alone. Pulsed oral steroid, is a well tolerated mode of therapy in extensive disease.

Key words: Alopecia areata, Alopecia totalis, Alopecia universalis, Nail affection, Ophiasis.

INTRODUCTION:

Alopecia areata (AA) is a common, unpredictable, non scarring form of hair loss without any visible signs of inflammation or skin symptoms. It is recurrent type of hair loss that can affect any hair bearing area. Limited scalp involvement is most common presentation, but more severe forms of the disorder, involving the entire scalp or body also exist. Onset may be at any age & sex.[(1)] Highest prevalence is found between 30-59 years of age.[(2)] At any given time, approximately 0.2% of population has alopecia areata & approximately 1.7% of the population will experience episodes of alopecia areata during their life time.[(3,4)] It is a T cell mediated autoimmune disorder, most likely to occur in genetically predisposed individuals.

Course of the disease is unpredictable with spontaneous remission and relapses. 34-50% of patients will recover within one year although almost all will experience more than one episode of the disease.[(3)] Clinically it may present with many patterns such as AA monolocularis (Fig. 1a), AA multilocularis (Fig. 1b), AA universalis, alopecia totalis, ophiasis, reticularis, sisaipho etc. A new variant recently described is 'Acute diffuse and total alopecia'.[(5)] It is characterized by a generalized thinning, rapid progression, tissue eosinophilia, brief clinical course and favorable prognosis.[(6)] Scarring is characteristically absent.[(7)] There are lack of adequate efficacy of medications available for the treatment of alopecia areata. The various modalities of treatment are immunosuppressants such as steroids, cyclosporine A, methotrexate, azathioprine, sulfasalazine and biological. Phototherapy has been tried in the form of psoralen plus UVA (PUVA) and narrow band UVB. Topical modalities include, topical immunotherapy (1-chloro, 2,4-dinitrobenzene [DNCB], Diphencyprone [DPCP], squaric acid dibutylester [SABDE]), topical steroids, calcineurin inhibitors, capsaicin, mesotherapy, topical irritant (anthralin) and vasodilator minoxidil. Treatment goal is hair re-growth that is cosmetically acceptable to the patient, but hair loss is not prevented by these treatment modalities.[(3,8)] The unpredictable severity and course of disorder exerts high emotional disturbances and may result in psychiatric co morbidity.[(9)]

Our study was a clinic-epidemiological study of alopecia areata in a tertiary care teaching hospital to note the varying patterns, demographic profile, associated diseases and therapeutic response to oral mini pulse (OMP) steroid therapy and intralesional steroids.

MATERIALS AND METHODS:

This study was undertaken in the department of dermatology, IMS & SUM Hospital, Bhubaneswar in collaboration with the department of Pharmacology, during a period of two years from November 2011 to October 2013.

Selection criteria:

During this period those patients attending the dermatology OPD having hair loss either patchy or diffuse with smooth bald surface and having no features of scarring, scaling or inflammation on the bald area were included in the study.

All age groups & both sexes of patients who had not received any treatment for last 3 months were included.

Exclusion criteria:

The following groups were excluded from our study

- Patients having Diabetes mellitus
- Pregnant women
- Those patients having other chronic illnesses such as tuberculosis

Methods:

With due consent, the patients were subjected to detailed history taking including demographic data, drug history, personal history, family history, present and past medical history and history of emotional stress, exposure to STD and drug intake. Other causes of hair loss were diagnosed and excluded from our study.

A thorough clinical evaluation was done to assess the clinical pattern, extent and severity of alopecia areata. Nail changes were documented in all patients.

Baseline investigations included complete hemogram, blood sugar, serum electrolytes, renal and liver function tests, chest X-ray, urine analysis and examination of stool especially for occult blood. All the patients underwent an ophthalmological evaluation before starting the therapy. Blood pressure and body weight were recorded initially and monitored every month.

In doubtful cases, KOH mount for fungal study was done to rule out tinea capitis. Similarly VDRL was done in some cases to rule out syphilitic alopecia. Biopsy was done in few cases. The diagnosis of alopecia areata was done from the history and clinical examination.

Two treatment modalities were tried on the patients as follows:

1. The patients having extensive alopecia areata (AA), active disease, alopecia universalis (AU), alopecia totalis (AT) were given steroid oral mini pulse therapy. We took active disease as those patients who were developing new patches or the existing patches were increasing in size. The OMP consisted of betamethasone 1mg tablets. The dose was given according to the age group.
    - 0 – 5yr = 1tab (1mg)
    - 5 – 10yrs = 2 tab (2mg)
    - 10 – 15yrs = 3 tab (3mg)
    - >15 yrs = 4 tab (4mg)

   It was given on two consecutive days of a week usually Saturday and Sunday after breakfast.
2. Those patients having one or two patches of small size < 4cm each were given intralesional triamcinolone injection.

Triamcinolone acetonide was injected into the deep dermis using a 0.5 inch long 30 gauze needle at multiple sites, 1cm apart and 0.1 ml into each site. This was repeated every 4-6 weeks. The triamcinolone concentration used was 10mg/ml for scalp and 2.5mg/ml for the face.

With both the above treatments the patients were followed up at an interval of 1 month for a period of 6months. During the follow up, the percentage of improvement in the form of re-growth of hair was recorded through serial clinical photographs. Any side effect of the treatment was recorded. Hair growth was classified as excellent with 76-100% growth, good with 51- 75% and unsatisfactory with <50% growth. It was considered as a treatment failure when there was no growth of hair even after 6 months of treatment.

Statistical methods: The data was analyzed statistically using descriptive statistics namely Mean and Proportion.

OBSERVATIONS AND RESULTS:

A total number of 72 cases were included in our study. Out of 72 patients included in our study maximum number (62 [88%]) of cases was AA classic type (Table 1). The other variants were AU (Fig. 2) (5, 7%), ophiasis (Fig. 3) (4, 5%) and AT (Fig. 4) (1, 2%). One patient had both the ophiasis and diffuse AA

Table 1: Clinical variants of Alopecia areata:

S:N:	Clinical variant	No of cases	Percentage (%)
1	Alopecia areata (AA)	62	88
2	Alopecia universalis (AU)	5	6
3	Alopecia totalis (AT)	1	1
4	Ophiasis	4	5

In our study (Table 2) most of the patients belonged to 3rd decade of life (21-30 years) i,e around 44% of total study population followed by 4th decade of life which is the second most common age for alopecia areata (29%). The others are being 11% in 1st decade, 13% in 2nd decade & 3% in 5th decade of life.

Table 2: Age wise distribution of cases of alopecia areata

Age in years	No of patients	Percentage (%)
0-10	8	11
11-20	9	13
21-30	32	44
31-40	21	29
41-50	2	3

The total number of male cases were 52 (72%) and female 20(28%) (Table 3). The sex ratio is being almost equal in first two decade but male predominance in subsequent decades.

Table 3: Table showing sex wise distribution of cases

Age groups	Total no of patients	No of males with %	No of females with %
0-10	8	4(50%)	4(50%)
11-20	9	5(55%)	4(45%)
21-30	32	24(75%)	8(25%)
31-40	21	17(80%)	4(20%)
41-50	2	2(100%)	0(0)
Total	72	52(72%)	20(28%)

In our study (Table 4) majority of patients had lesion on the scalp (58.3%). Out of these, 16 had single lesion and 26 had multiple lesions. Face was involved in 17 patients, 15 having single lesion & 2 having multiple lesion. Both scalp and body was involved in 11 and only body in 2 patients.

Table4: Table Showing Location (site) wise Distribution of Cases

Location	No of patients with single lesion with %	No of patients with multiple lesions with %	Total
Scalp	16(38%)	26(62%)	42(58.3%)
Face	15(88.2%)	2(11.8%)	17(24%)
Scalp + Body	0(0%)	11(100%)	11(16%)
Body	0(0%)	2(100%)	2(3%)
			Total = 72

Out of 62 patients (Table 5), most (34) of the patients had oval lesion (54%). Fourteen (23%) had circular and 14 (23%) had reticular lesions.

Table 5: Shape of lesion (excluding AU,AT and Ophiasis)

Shape	No of patients	Percentage (%)
Oval	34	54
Circular	14	23
Reticular	14	23
Total	62	100

Out of 72 patients 7 had nail involvement (Table 6). Most common nail involvement was pitting (57%) followed by longitudinal ridging (29%) and dystrophy (14%).

Table 6: Nail involvement

Nail change	No of patients with %
Pitting	4(57)
Longitudinal ridging	2(29)
Dystrophy	1(13)
total	7

Majority (Table 7) of patients (72%) presented within 6 months of disease onset.

Table 7: Duration of disease:

Duration	No of patients	Percentage (%)
0-6months	52	72
6 m- 1 yr	7	10
>1yr	13	18

Out of 72 patients (Table 8), 7 (10%) had family history of AA. It was further noticed that those patients having positive family history had extensive disease. Three patients (43%) had extensive AA, 3 (43%) had AU and 1 (14%) had ophiasis pattern of involvement.

Table 8: Associated Family history of AA according to clinical variants:

Variants	No of patients with positive F/H	Percentage (%)
Extensive AA	3	43
AU	3	43
Ophiasis	1	14
Total	7	

Out of 72 patients (Table 9), 10 had associated diseases such as atopy in 7, followed by LP in 2 and vitiligo in 1 patient.

Table 9: Associated disease:

Associated disease	No of patients	Percentage (%)
Atopy	7	10
Lichen Planus	2	3
Vitiligo	1	1.5

Out of 42 patients tried with intralesional triamcinolone acetonide (Table 10), 6 patients didn't report for follow up. Thus out of 36 patients, 26 (72.2%) responded to our trial. Maximum number of patients (20) had excellent result by the end of 6 month. As regards to side effects, only 3 patients had folliculitis at the site of administration. One patient had local irritation which subsided with regular follow up. One patient had transient atrophy at the site of application.

Table 10: Response to intralesional steroid at 6 months (n=36)

Response	No. of patients
Excellent	20
Good	6
Unsatisfactory	2
Non responsive	8

Out of 30 patients with OMP therapy (Table 11), 5 patients didn't report for follow up. Thus out of 25 patients, 15 (60%) showed good response. Side effects noted were acneiform eruptions, gastric upset and cushingoid facies.

Table no 11: Response to OMP steroid at 6 months (n=25)

Response	No of patients
Excellent	9
Good	6
Unsatisfactory	4
Non responsive	6

Figure 1a : AA MONOLOCULARIS and 1b : AA MULTILOCULARIS

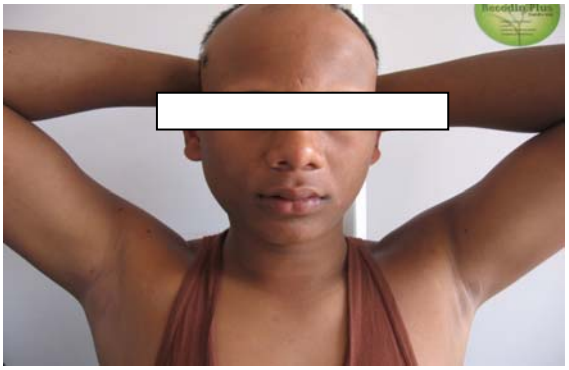


Figure 2: ALOPECIA UNIVERSALIS

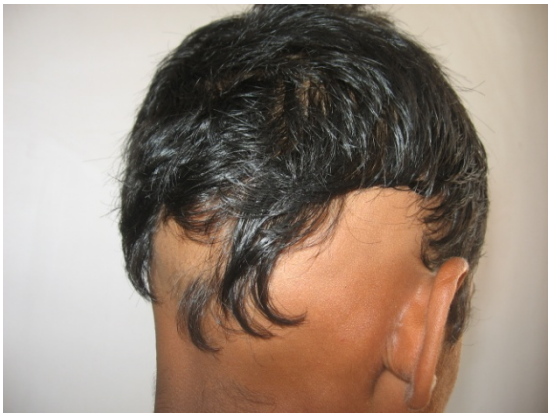


Figure 3: OPHIASIS

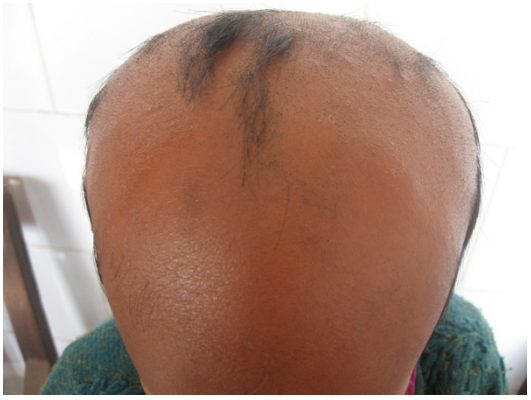


Figure 4: ALOPECIA TOTALIS

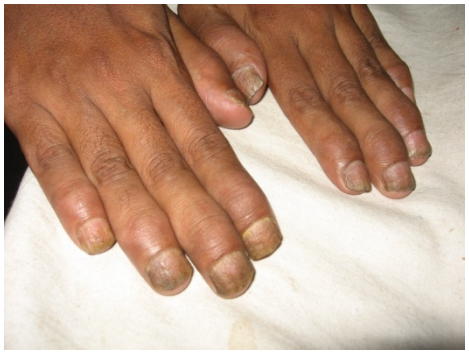
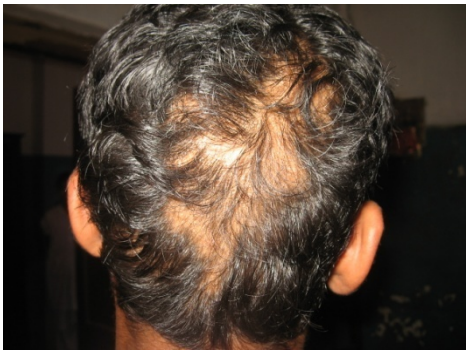


Figure 5: PITTING AND LONGITUDINAL RIDGING IN SEVERE ALOPECIA AREATA



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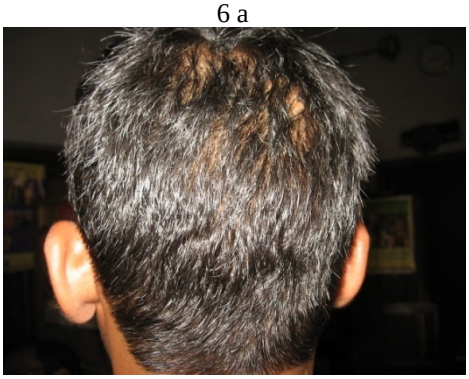

6b

Figure 6a : PRETREATMENT PHOTO OF ALOPECIA AREATA, 6b : HAIR GROWTH POST 8 WEEKS OF OMP THERAPY

DISCUSSION:

The etiology of AA is still an enigma. The most accepted hypothesis is a hair follicle specific T-cell mediated autoimmune disorder occurring in genetically predisposed individuals with a suggested polygenic inheritance.[5] It is linked with certain HLA class II alleles. The HLA-DQB1*03 allele may be an important marker for susceptibility to the disease.[10,11]

Diagnosis is mostly clinical. A skin biopsy specimen is confirmatory for AA. In acute cases peribulbar and intrabulbar lymphocytic inflammatory infiltrate around anagen follicles, resembling 'swarm of bees' is characteristic. Dermoscopic findings include yellow dots, black dots, broken hairs, tapering hair (exclamation mark) and short vellus hairs.

Extrafollicular involvement comprises of ungual and ocular alterations. Nail changes are seen in 29% of adults and 50% of children with AA. Nail changes (Fig. 5) mainly include superficial geometric pitting, punctuate leukonychia and trachyonychia.[12] Punctate lens opacities, early cataracts and fundal abnormalities may occur in 40% to 50% of patients with AA.[13]

The presence of exclamatory mark hairs at periphery, positive hair pull test (>6 hairs), daily hair count (>100 hairs), hair pluck test (more telogen hairs) and positive dermoscopic findings suggest active disease.[14] Predictors of poor prognosis include, younger age of onset, family history of AA, atopy, severe disease like alopecia totalis and alopecia universalis, ophiasis, duration more than 1 year & associated nail and autoimmune disease.[15,16]

In our study out of 72 patients, AA classic variant having patchy lesions constituted the maximum percentage (88%)

of all cases followed by 7% AU, 5% Ophiasis and 3% AT. In various studies conducted in the past it was seen that AA classic variant constituted the major proportion of cases in comparison to other variants.[17,18] Age range of the patients of AA in our study was 1 and ½ years to 48 years among which maximum cases were seen in 3rd decade of life (44%) followed by 4th decade (29%). The peak incidence of manifestation in all clinical variants of AA if grouped together was in between the age of 20 and 50 years and onset to occur is at any age. S Manzoor et. al.[19] reported that maximum number of patients were in the age group of third decade and 4th decade, which was consistent with our findings. Out of 72 patients 72% (52 patients were male and 28% (20 patients) were female. Thus the sex ratio in our study is 2.6:1. The reported sex incidence has varied widely from males out numbering females by 3:1[20], through equality[21], to twice as common in females.[22] Manzoor S et al[19], in an Indian study had found the male to female ratio to be 2.63:1. Male preponderance could be due to more number of male patients attending the hospital.

Majority of patients had lesion on the scalp (58.3%) followed by face i,e beard, moustache and eyebrows (24%), scalp and body (16%) and body alone (3%). In an Indian study by Jain S et. al. 2003[18], maximum number of cases were seen in scalp. Lesion of AA can have various shapes. In our study maximum number of patients excluding AU, AT and Ophiasis had oval and circular lesions (77%). Only 14 patients had reticular lesion. This is consistent with the finding of Sadollah S et.al.[23] who in their study of 956 patients of AA reported that their commonest clinical presentations were round and oval configuration. In our study, out of 72 patients 10% (7 patients) had nail involvement among which the commonest presentation was pitting (57%) followed by longitudinal ridging (29%) and dystrophy (14%). S. Jain et al 2003 [18] in their study of 150 patients reported nail changes in 13% of patients, the commonest being pitting.

In our study the minimum duration of the disease was 15 days and maximum was 6 years. However, majority of patients presented within 6 months of onset (72%). This is consistent with the findings of Jain S etal 2003 [18] who reported that 78% of patients presented within 6 months of onset. Familial incidence of AA varies from 10 to 20% in different studies.[21,24] In our study 10% of patients were having positive family history. This is to be more frequent with severe forms of AA. Some cases of AA are associated with autoimmune diseases. In our study 10 patients had associated diseases among which 10% had atopy, 3% with Lichen planus and 1.5% vitiligo. Jain S et.al.[18] had observed atopic manifestation in 11.37% of their patients.

Out of 30 patients tried with OMP, 5 patients didn't turn up for follow up. Of the remaining 25 patients, 60% (15 patients) responded to our trial. Ahu Birol et. al.[25] had reported response rate of 80% by giving pulse steroid for a period of 6months. Patients had acneiform eruptions, gastric upsets & cushingoid facies at the end of 6 months. Binod K Khaitan et. al.[26] have reported these side effects in few patients. Treatment with intralesional steroid showed good result in 72.2% of patients in our study which was consistent with results (65-70%) found in other studies.[27]

CONCLUSION:

Alopecia areata is a relatively benign non scarring form of hair follicle specific autoimmune disease, triggered by environmental factor in genetically susceptible individuals. Treatment is still an enigma and large number of treatment modalities speaks of their lack of adequate efficacy.

We conclude that both intralesional steroid and OMP steroid are effective modalities in the treatment of alopecia areata with limited side effects. But as our study was done in a limited number of cases and was an uncontrolled one we recommend a large scale controlled study which is needed to be done to conclude that both are really effective modality of treatment in AA. This condition has a definite psychological impact and affects the quality of life of many patients. Proper counseling and appropriate treatment helps to attain cosmetically acceptable hair regrowth and improves quality of life.

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