“COMPARISON OF VARIOUS TREATMENT MODALITIES IN PATIENTS WITH ALOPECIA AREATA”

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(BRANCH XX)

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CERTIFICATE

This is to certify that the dissertation titled **"COMPARISON OF VARIOUS TREATMENT MODALITIES IN PATIENTS WITH ALOPECIA AREATA"** is a bonafide work done by **DR. FATHIMA.S ,** Post graduate student of the Department of Dermatology, Venereology and Leprosy, Madras Medical College, Chennai – 3, during the academic year 2016– 2019. This work has not previously formed the basis for the award of any degree.

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DECLARATION

The dissertation entitled **"COMPARISON OF VARIOUS TREATMENT MODALITIES IN PATIENTS WITH ALOPECIA AREATA"** is a bonafide work done by **Dr. FATHIMA . S,** Department of Dermatology, Venereology and Leprosy, Madras Medical College, Chennai – 3, during the academic year 2016 – 2019 under the guidance of **PROF Dr.U.R.DHANALAKSHMI,M.D,D.D,DNB(DERM).,**Professor and head of the depatment, Department of Dermatology, Madras Medical College, Chennai -3. This dissertation is submitted to The Tamil Nadu Dr. M.G.R. Medical University, Chennai towards partial fulfillment of the rules and regulations for the award of M.D Degree in Dermatology, Venereology and Leprosy (BRANCH – XX).

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DECLARATION

I, **Dr. FATHIMA . S ,** solemnly declare that this dissertation titled **"COMPARISON OF VARIOUS TREATMENT MODALITIES IN PATIENTS WITH ALOPECIA AREATA"** is a bonafide work done by me at Madras Medical College during 2016-2019 under the guidance and supervision of **Prof. U. R. DHANALAKSHMI, M.D., D.D, DNB (DERM).,** Professor and Head of Department, Department of Dermatology, Madras Medical College, Chennai-600003. This dissertation is submitted to The Tamil Nadu Dr. M.G.R. Medical University, Chennai towards partial fulfillment of the rules and regulations for the award of M.D Degree in Dermatology, Venereology and Leprology (BRANCH – XX).

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CONTENTS

Introduction

INTRODUCTION

Alopecia is a term used to denote the loss of hair. Alopecia areata (AA) is a common form of hair loss seen by dermatologists and accounts for 25% of all alopecia cases.

The factors that activate the onset of alopecia areata and the mechanisms of its development are not fully understood. Circumstantial evidence suggests that alopecia areata is an autoimmune disease with genetic substrate. Alopecia areata produces marked cosmetic disability and extensive psychological morbidity. Diagnosis is simple but treatment is challenging and time consuming.

Though various modalities are available for the treatment of alopecia areata, assessment of the efficacy of a treatment must be considered with care because the condition is highly unpredictable in presentation, evolution, and response to treatment. Little data exists regarding the natural evolution of the condition. Hence with this in mind the following study was carried out to see the treatment response for various modalities for this benign condition.

Review of Literature

REVIEW OF LITERATURE

INTRODUCTION

Hair is a cutaneous appendage typical of mammalian skin[1]. It has no vital function in humans, but its psychological functions are extremely important[2]. Hair is present all over the body except over the palmo plantar skin, the distal dorsal aspect of the digits and muco-cutaneous junction [1]. Five million hair follicles are there in skin and 2% of it is in scalp. In the scalp of adults aged 20-30 years the density of hair on an average is 615/cm^2 and this density gradually decreases to 435cm^2 by 80-90 years of age.[2]

HAIR CYCLE[2]:

Knowledge of hair cycle is important for understanding the hair problems. Hair growth depends on three phases of hair cycle which includes anagen, catagen and telogen. The anagen which is active growth phase lasts for two to six years for scalp hair. This is followed by the catagen (involution phase) which takes 1- 3weeks and the telogen (resting phase) lasts for about 3 months.

The type and length of hair depends on the anagen phase. In normal healthy individuals, hair sheds out after the resting phase when the new hair anagen growth starts (exogen). In alopecia, hair shedding occurs even before the anagen starts leaving the hair follicle empty (kenogen). Thus, alopecia areata is generally a disorder of hair cycling and is considered to be a state of kenogen.

ALOPECIA AREATA (AA)

Hippocrates first used the term alopecia (literally translated as "Fox's disease"). Alopecia areata was first described by Cornelius Celsus in 30 AD. Sauvages coined the term AA in 1760[4]. It accounts for 2-3% of dermatology cases in UK and USA, 3.8% in China, and 0.7% in India[4,5,6]. The prevalence in general population was around 0.1-0.2% with lifetime risk of 1.7%[6]. Both males and females are equally affected[7], but some studies reported male preponderance[4,6,8,9]. It can occur at any age. The youngest reported case was 4-months-old, and the oldest was in late seventies[11]. Twenty percent of cases were children, and 60% of AA patients had their first patch before the age of 20 years[7]. Highest prevalence was between 30-59 yrs of age[10]. Family history of AA were reported in 8.7-20% of cases[4,11].

SYNONYMS OF AA[12]

Fox's disease	Porrigo decalvans
Alopecia circumscripto	Tinea decalvans
Area celsi	Area tonstonii
Jonstons alopecia	Cazanave's vitiligo
Vitiligo decalvans	Celsus vitiligo

AETIOLOGY:

AA accounts for about 2% of new dermatological outpatient attendance[2,13]. At present it is not possible to attribute it to any single cause. Various theories

such as infections, genetic, stress, trauma (physical), immunologic, endocrine dysfunction and focal sepsis have been proposed by earlier workers.

The etiopathogenesis of AA will be discussed under following headings.

Genetic factors

Immunological factors

Atopy

Stress

Infection

GENETIC FACTORS

Genetic factors play an important role in the origin of AA. A positive family history has been found in about 8.7-20% of patients[4,11]. There was a significantly higher incidence of family history of AA in patients with early onset of AA. The mode of inheritance is thought to be an autosomal dominant with variable penetrance. Some studies have shown a concurrence in the onset among twins[14]. AA is most likely a polygenic disease where several potentially identifiable major genes affect disease susceptibility and minor modifying genes may further affect the phenotype[15]. There has been increasing evidence of association between AA and HLA class II genes.

Studies have revealed an association with HLA-A1, HLA-B62, HLA-DR4, HLA-DR5, HLA-DQ1, HLA-DQ3, HLA-DR11 and HLA-DQ7[16]. One of the studies has shown that 80% of all AA patients were positive for HLA-DQ3, suggesting that this antigen is a marker for general susceptibility to AA[16,17]. HLA-

DR16 was significantly less common in patients than in control group and this allele probably has a protective role for AA.

Juvenile onset and severe involvement were related with CW7 and DR1[16,17]. HLA-DR5 is linked to early onset form of AA and more extensive hair loss. The alleles DR4 and DQ7 are markers for more severe long standing alopecia totalis and universalis respectively[18].

Recently, genome wide association studies (GWAS) identified specific genetic markers for AA. GWAS can recognize specific individual genes, which may increase the risk for AA. Petukhova et al. surveyed the entire genome and identified 139 single nucleotide polymorphisms (SNPs) for AA, clustered in 8 regions of the genome. GWAS studies had found key genes in AA related to T-cells (IL2/IL21, IL2RA, CTLA4, IKZF4, HLA) and hair follicle (NK-activating ligands-ULBP3, ULBP6, STX17, PRDX5)[19,20]. All these evidences point towards the genetic predisposition in the development of AA.

PSEUDOHERIDITY IN AA[21]

In some familes there is an unusually high incidence of AA which may be due to psychological component at the background perpetuating a stressful environment which takes toll generation after generation. The term pseudo heridity describes the situation which derives from environmental rather than genetic factors[21]. There is up to 8.8 % increased frequency in polyglandular syndrome[2,13]. The increased frequency of AA in Downs syndrome and polyglandular syndrome is due to mutation of autosomal regulator gene on

chromosome 21[21]. Several studies have confirmed the association between AA and atopic state[3].

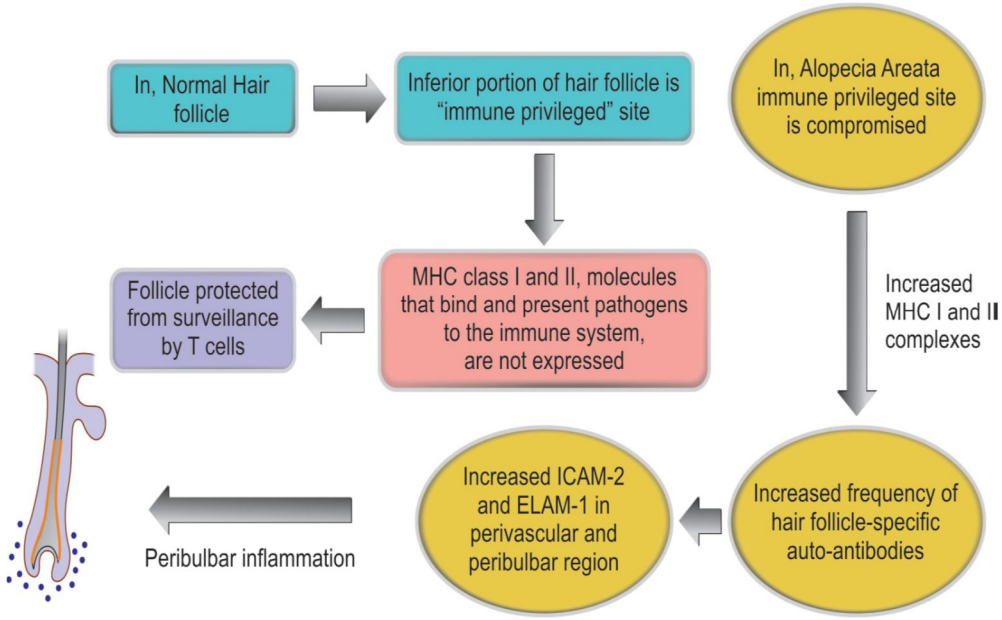
IMMUNOLOGICAL FACTORS:

The idea that AA is an autoimmune disease was suggested first by Rothman . The association of AA with other autoimmune diseases like thyroid disease, anemia, diabetes mellitus, vitiligo, and psoriasis may be one of the causes to believe AA is an autoimmune disease[22,23]. Increased level of Hair follicle-specific antibodies, especially to keratin 16 and trichohyalin were found in peripheral blood of AA patients[24].

Hair follicles being an immune-privileged site, major histocompatibility complex (MHC) class I and II molecules are not expressed. TGF- β, IGF-1, and α-MSH are more expressed.[25] This immune privilege is collapsed in AA leading to increased MHC I and II complexes, decreased immunosuppressive molecules and higher expression of adhesion molecules (ICAM-2 and ELAM-1) in perivascular and peribulbar hair follicular epithelium. This leads to perifollicular inflammation[26] and thus the activity of hair follicle is adversely affected, resulting in thin dystrophic hair with miniaturization[27].

Increased prevalence of antithyroid antibodies, thyroid microsomal antibodies, of gastric parietal cell antibodies as well as antinuclear and antismooth muscle antibodies have been reported. There have been reports of association of AA with pernicious anaemia, myasthenia gravis, diabetes, vitiligo,lupus erythromatosus, rheumatoid arthritis, ulcerative colitis, psoriasis and lichen

planus. Muller and Winkleman have reported an incidence of 4% of vitiligo in AA patients. Milgraum et. al. have found 24% of 45 children 16 years of age with AA to have abnormal thyroid function test or elevation of thyroid microsomal antibodies.



HUMORAL IMMUNITY

Toben et. al. have found high level of antibodies to multiple structure of anagen hair follicle in AA patients using indirect immunoflorescence[28]. The most common target structures were the outer root sheath, followed by the matrix, inner root sheath and hair shaft.

CELL MEDIATED IMMUNITY

Studies of cell-mediated immunity in AA have given conflicting results. Circulating total T lymphocytes are reduced or normal[3,29]. A slight increase in helper cells (CD4) and decrease in number of suppressor T cells (CD4) resulting

in an increase in the ratio of helper to suppressor cells correlates with the amount of hair loss[29].

Successful treatment of AA with immunomodulatory agents such as oral cyclosporine and systemic steroids also supports the immune mediated pathogenesis in AA[30,31]. Biopsies of patients treated with contact allergen diphencyprone and minoxidil have shown reduction in peribulbar T cell, in regrowing AA[32].

Increased frequency of organ specific antibodies, antibodies to pigmentary hair follicles, high level of auto antibodies to multiple structures of anagen hair follicles in AA patients, an increase in ratio of helper to suppressor cells, are the evidences to support the hypothesis that alopecia areata is a hair follicle-specific autoimmune disease, triggered by environmental factor in genetically susceptible individuals.

ATOPY

The association between alopecia and atopy is observed in many studies. In atopic indivuduals, AA occurs at an earlier age and then to be more severe than in non-atopics. Statistical analysis in Indians with AA showed no such links[33].

STRESS[34]

The influence of psychological factor in development, evolution and therapeutic management is well documented[35]. Psychological trauma is often suggested as a factor involved in precipitating attacks of AA. Koo et al.have

reported 74% of patients of AA had one or more lifetime psychiatric illnesses. Major depression, generalized anxiety, social phobia and paranoid disorders were all present in patients with AA at rates significantly higher than in general population[34].

INFECTION

Focal infection , particulary dental disease has been reported as a precipitant in AA, but never been shown ,conclusively to be important[36]. Skinner et. al. have shown convincing positive association with CMV[33,37]. Subsequent studies have not confirmed these findings[38].

Besides these , hormones, diet, vaccinations and many others were incriminated in the pathogenesis of AA[27,39]. Iron deficiency was noted in 24-71% of females with AA[40]. AA was less frequently observed in people, taking diet rich in soy oil. Some studies found reduced zinc levels in AA patients[9], but others reported conflicting results[41]. An outbreak of AA in workers of water treatment plant in paper factory, linked to long-term exposure to chemical acrylamide was reported by Roselino et al[42].

CLINICAL FEATURES[1,2,3]

AA occurs all over the world. Both sexes are almost equally affected .The onset of disease occurs at any age with the peak incidence probably between the age of 20 and 50 yrs. In an Indian study the incidence among children was higher, around 31%.

The characteristic lesion of AA is commonly a round or oval, totally bald, smooth patch involving the scalp or any hair bearing area on body. The hair loss is usually asymptomatic in most cases, but some patients complain of parasthesias with mild to moderate pruritis , tenderness, burning sensation or pain before the appearance of patches.

Clinical classification of AA

1.Based on pattern	2. Based on extent	3. Based on area
Patchy type	AA circumscripta	AA of the scalp
Reticular type	AA subtotalis	AA of eyelashes
Ophiasis type	AA totalis	AA of eyebrows
Sisaipho type	AA universalis	AA of beard
Diffuse type	Diffuse AA	AA of body hair
Perinevoid type	AA incognito	AA of nail

- Alopecia areata – The most commonly used term covers all forms of the disease
- Alopecia partialis – Defines specific patchy hair loss
- Alopecia totalis – Involves100 % loss of scalp hair.
- Alopecia universalis – When all body and scalp hairs are lost
- Alopecia areata barbae – AA lesion of beard
- Alopecia areata ophiasis – A special band like pattern of AA , which winds along the occipital hair line extending toward the temples (oophiasis – Greek for snake)

- Diffuse AA –A term occasionally used for an alopecia areata where there is generalized thinning of hair of scalp.
- Reticular alopecia areata- Multiple active, stable or re growing patches, which may merge to form mosaic or reticular pattern.
- Sisaipho AA- Opposite of ophiasis where hairs are lost centrally and spared at the margins of scalp. It may mimic androgenetic alopecia.
- AA Incognito – First described by Rebora in 1987. Extremely acute onset with subsequent diffuse hair loss that occurs within few weeks.
- Perinevoid AA – An unusual and rare form with patches around a nevus.

Ikeda classification of AA

Based on the associated conditions and on the course of the disease

1. Atopic type (10%): Begins in early life and mostly (30-75%) progresses to AT.
2. Autoimmune type (5%): Seen in middle-aged , runs prolonged course. Associated with autoimmune diseases, diabetes mellitus, peptic ulcer and progresses to AT in 10-50%.
3. Pre hypertensive type(4%): Occurs in young adults whose one or both parents were hypertensive and progress fastly to AT in 40% of cases.
4. Common type(81%): Mostly affects adults aged 20-40 years and AT develops in 5-15% of cases.

A frequent feature of an AA patch is ‘exclamation mark’ hair that may be present at its margin[12]. Exclamation mark hair denotes the broken short hairs that taper proximally. At the margins the normal looking hair may be easily extracted. This pull test when positive may indicate very active disease.

A hand lens shows that at the margin, the free ends are splayed, giving frayed appearance. Even the apparently normal terminal hair, found within the patches, may show one or more constrictions in their shaft.

Shuster has described the coudablity sign to (differentiate diffuse AA from other diffuse alopecia) in AA, a normal looking hair kinks when forced inwards or bents, the kink being situated 5-10mms above the surface[43].

An intriguing feature of AA is sparing of white hairs[44]. It appears preferentially to affect pigmented hair, so that non pigmented hair is spared. The white hairs although less susceptible are not immune to the disease.

The re growth of hair may be

- Fine and un pigmented hair initially
- Gradually gains in caliber and color
- Heterochromia of hair[45]
- Hair growth starts in center, extends peripherally
- May remain un pigmented for a long time- leading to poliosis
- Re growth and extension may occur simultaneously in different regions of the scalp

ASSOCIATED CLINICAL SIGNS [1,2,3]

NAIL INVOLVEMENT

Nail involvement may be associated with AA. The reported incidence of nail changes range from 10 to 66%. Changes may be seen in one ,several or all nails. The dystrophy may precede or coincide with AA and may also occur after resolution of AA , some authors speculate the possibility of 'Twenty nail dystrophy sine alopecia'

The commonest nail change is superficial,uniform minute pits arrange regularly along and across the nail giving a 'Scotch Plaid' effect or coalescing into ripples.

Other changes include ,

Trachyonychia or rough nails(longitudinal striation resulting in sand appearance)

- Red or mottled lunula
- Nail thinning and ridging
- Beau's lines (grooves through the nails matching that of the lunula margin)
- Onychorrhexis (Superficial splitting of nail extending to the free edge)
- Onychomeadesis (Onycholysis with loss of nails),
- Koilonychia punctate or transverse leuconychia
- Dystrophy
- splitting

EYE CHANGES[2,46]

Keratoconus and cataract are the most common eye changes reported in association with AA. Other reported eye changes include Horner's syndrome, ectopia of the pupil, iris atrophy or tortuosity of the fundal vessels.

ASSOCIATIONS

- Thyroid autoimmunity
- Vitiligo
- Atopy
- Down syndrome
- Addison disease
- Autosomal recessive autoimmune polyglandular syndrome(APS- I)
- Pernicious anemia
- Lupus
- Celiac disease
- Ulcerative colitis
- Multiple sclerosis
- Psychological disturbances

INVESTIGATIONS :

Diagnosis is based mainly on the clinical appearances. AA is characterized by non scarring smooth bald patches. Histology can be used to corroborate the diagnosis. Trichogram reveals a mixed telogen dystrophic pattern. Telogen hairs

predominate in the slowly progressing patches, whereas dystrophic anagen hairs form the majority in rapidly progressing disease.

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 dermoscopy (black dots,yellow dots, brokenhair, and tapering hair) suggest active disease.

TRICHOSCOPY

Trichoscopy is a non-invasive method of hair and scalp evaluation. The test may be performed with the use of a handheld dermoscope or a videodermoscope. Trichoscopy findings in

- Alopecia Areata
    - Yellow dots (hyperkeratotic plugs),
    - Micro-exclamation mark hairs,
    - Black dots (destroyed hairs in the hair follicle opening)
    - Miniaturized nanogen hairs
- Androgenetic alopecia
    - Greater than 20% diversity in the hair diameter
    - A brown, depressed halo at the follicular opening – Early cases
    - Yellow dots – Advanced cases
    - Honeycomb-pigmented appearance is seen in sun-exposed regions of the scalp.

- Tinea capitis
    - comma hairs, corkscrew hairs , fractured hair shafts
- Cicatricial alopecia
    - Reveals decreased hair density and loss of follicular openings
- Lichen planopilaris
    - Hyperkeratotic, perifollicular white scales with variable perifollicular erythema and peripilar white dots along with blue-gray dots with a target distribution around the follicle.
- Discoid lupus erythematosus of the scalp
    - Mottled dyschromia, follicular plugs, telangiectasias, white central plaques, and irregularly distributed blue-gray dots in a speckled pattern between the hair follicles may be seen.
    - Red dots appear as erythematous, polycyclic, concentric structures, regularly distributed around the follicular opening.
- Folliculitis decalvans
    - Tufted hairs
    - Perifollicular pustules

HAIR PULL TEST.

A group of approximately 60 hairs is hold between the thumb and index finger of the non dominant hand. With the dominant hand the strands of hair are loosely twisted to remove tray hairs and then the hairs are grasped between the dominant thumb and forefinger near the scalp. Gentle traction is applied in a

smooth, gradual manner, away from the scalp. The number and type of hairs extracted may give clues to the underlying diagnosis.

Grasping a few hairs from the edge of a focal area of hair loss may give an indication of disease activity.

Telogen effluvium	-	Increase in telogen hairs extracted from all areas
Alopecia areata	-	Increase in telogen hairs or dystrophic anagen hairs from affected areas
Primary cicatricial alopecias	-	Increase in anagen hairs extracted (pigmented bulb, ensheathed within root sheath)
Loose anagen syndrome	-	Painless extraction of dysplastic anagen hairs, Often lacking root sheath

TRICHOGRAM.

A trichogram is a forced pluck of hair that includes the hair roots. A group of 60–80 hairs is clasped between rubber armed forceps or needle holders, close to the scalp surface. The examiner holds the needle holders firmly and rapidly tugs the needle holders away from the scalp in a perpendicular direction to extract all the hairs in the sample. The hairs can then be analysed under a microscope to calculate the percentage of hairs in telogen and anagen phases.

HISTOPATHOLOGY

The histopathological findings varies with duration of the disease. Ideal biopsy should include two 4 mm punch including the subcutaneous fat. One specimen is processed with vertical sectioning and other with horizontal sectioning. If only a single specimen is planned, horizontal sections will give a better . A horizontally-sectioned scalp biopsy in addition confirming the diagnosis of AA also provides information about the possible regrowth[47]. The ideal site for biopsy is at the advancing border of hair loss. This helps to view the follicles at different levels in dermis to quantify the hair follicle density, diameter, and to assess the proportion of hair follicles in various stages. A mean count of less than 1follicle/mm ^{2} usually indicates less chances of regrowth[47].

Acute stage - Peribulbar and intra bulbar lymphocytic inflammatory infiltrate around anagen follicles, resembling 'swarm of bees,' is characteristic. The lymphocytic infiltrate are mainly found around the hair matrix and dermal papilla sparing the bulge area, causing follicular edema, cellular necrosis, micro vesiculation, and pigment incontinence. A dense lymphocytic inflammation can cause weakening of the hair shaft resulting in a trichorrhexis nodosa-like fracture, leading to the exclamation mark hairs[48].

Sub acute stage - High proportion of catagen/telogen hair follicles are seen.

Chronic stage - Follicular miniaturization with variable inflammatory infiltrate are seen in papillary dermis. The terminal to vellus hair ratio is decreased

to 1:1 in contrast to 7:1 in normal population. Androgenetic alopecia also shows follicular miniaturization, but more number of telogen hairs with decreased anagen to telogen ratio may be a clue towards AA[47]. Immunofluorescence studies shows deposits of C3, IgG and IgM on the basement membrane in the inferior part of hair follicle.

Recovery stage - The number of terminal anagen hairs increase and there is a lack of inflammation.

HISTOPATHOLOGICAL DIFFERENTIAL DIAGNOSIS:

In androgenic alopecia, dermal infiltrates when present remains superficial, perivascular. Alopecia syphilitica may produce follicular infiltrates that can closely mimic those of acute alopecia areata. Syphilitic alopecia areata may demonstrate a superficial and deep perivascular and perifollicular infiltrate that penetrates the outer root epithelium with a concomitant perifollicular fibrosing reaction.

In telogen effluvium normal number of hair follicles with no miniaturization of follicles and a slight decrease in the anagen to the telogen may be noted[3].

Optical coherence tomography (OCT) is a recently evaluated non-invasive technique to detect the hair shaft abnormalities in AA. Bartles et al. demonstrated that the cross section of hairs from an AA patch was significantly lower compared with hairs of an unaffected area by this OCT[49]. Thus, OCT may be an useful non-

invasive technique to differentiate AA from other causes of patchy alopecia, such as trichotillomania.

Complete hemogram, ear nose throat and dental examination are done rule out any foci of sepsis. In doubtful cases, Potassium hydroxide wet mount of hair root and fungal culture is done to rule out fungal infection. Serological test is done to rule out syphilis and lupus. Serum levels of thyroid hormones may be of use and may be done in suspected cases of thyroid disorders. Thyroid screening may be of use in long-standing cases, females with persistent patches, patients with suggestive symptoms of thyroid disease and severe AA (AT/AU).

SALT SCORE[50]

Severity of AA can be measured by SALT((severity of alopecia tool) score, developed by the National Alopecia Areata Foundation working committee.

SALT score is useful to find out the quantitative assessment of scalp hair loss. The entire scalp was divided into 4 parts based on the surface area, top (40% - 0.4), posterior (24% - 0.24), right side (18% - 0.18), and left side of scalp (18% - 0.18). Percentage of hair loss in each area is determined independently and is multiplied by the percentage of scalp covered in that area of the scalp, and summing the products of each area will give the SALT score. For example, the hair loss is 40%, 30%, 20% and 10% in top, back and right and left side respectively, then the SALT score can be calculated as- (40 × 0.4) + (30 × 0.24) + (20 × 0.18) + (10 × 0.18) =16 + 7.2 + 3.6 + 1.8 = 28.6. SALT score is easily

reproducible and validated. However, it does not include hair pigmentation, body hair, and nail involvement.

DIFFERNTIAL DIAGNOSIS[1,3]

AA has to be differentiated from the from the following conditions

1. Telogen effluvium - Here the hair loss is generalised over the entire scalp, where as in AA it is usually patchy .The hairs that are shed are either in telogen or dystrophic anagen in AA, but are predominantly in telogen in telogen effluvium.
2. Androgenetic alopecia-It typically involves the fronto temporal region and the vertex
3. Trichotillomania-Patches of alopecia with twisted broken hairs are noticed.
4. Secondary syphilis –It is a moth eaten alopecia with positive serological investigation
5. Alopecia neoplastica[51]-There may be history of malignancies like carcinoma breast or others .The affected skin is slightly indurated and erythematous. A biopsy clinches the diagnosis.
6. Congenital triangular alopecia- Characterised by a triangular area overlying the frontotemporal suture just inside the anterior hair line with its base directed forward which is completely bald or covered or by sparse vellus hair.

7. Tinea capitis- The patches of baldness of have irregular borders and broken bits may be seen within the patch. Examination of hair root with 40%KOH reveals fungal elements.

8. Others – Congenital hypotrichosis, Ectodermal dysplasis, Loose anagen hair , Cicatricial alopecia, Tractional alopecia..

COURSE OF AA

The progress of AA is unpredictable. The initial patch may regrow within a few months or further patches may appear after interval of 3 to 6 weeks and further patches may appear in a cyclical fashion at varying intervals. A succession of discrete patches may rapidly become confluent by the diffuse loss of remaining hair. In some cases the initial hair loss is diffuse and total denudation of scalp has been reported. Diffuse hair loss also may occur over a part or whole of the scalp without the development of bald patches. Kling miller has showed that white hairs are spared initially by the disease process. Patients with sudden diffuse onset of AA would appear to ‘go white” over the course of a few days. Re-growth is often at first fine and unpigmented but usually the hair gradually resume its normal calibre and colour. Persistent depigmented hair regrowth from the site of AA has also been reported[52].

PROGNOSIS

The prognosis is good in the common simple form of AA in which the hair loss is confined to the scalp and has got a high natural remission rate.

The following are indicators of poor prognosis.

- Early age of onset
- Child with atopy
- Patients with Down's syndrome
- Family history of AA
- Alopecia totalis or Alopecia Universalis
- Involvement of more than 50% of the scalp
- Ophiasic and reticular pattern
- Bilateral loss of eyebrow and eyelashes
- Severe nail changes
- Patchy regrowth of terminal hairs within the patch
- Recurrent episode

TREATMENT[1,2]

Early recognition and intervention involving topical and or intralesional therapy can provide patient with comforting reassurance about eventual recovery. An unpredicatable course and heterogeneity of the disease leads to varying results being obtained with the same therapy. The various modalities can be classified as follows,

1. Topical therapy
2. Systemic therapy
3. Others

TOPICAL THERAPY INCLUDES

1. Immunosuppresants- Topical corticosteroids with or without occlusion, intralesional steroids, nitrogen mustard.
2. Non specific irritants- Anthralin, Phenol, Salicylic acid, sulphur , oil of cade, cantharidin, croton oil, tretinoin
3. Contact sensitizer- Dinitrocholrobenzene ,Squaric acid dibutylester (SADBE) and diphencyprone
4. Photochemotherapy
5. Cryotherapy
6. Lasertherapy - excimer laser(308nm), pulsed infrared diode laser(904nm)

SYSTEMIC THERAPY

1. Immunosuppresants - corticosteroids, cyclosporine
2. Immunomodulators - alefacept
3. Photochemotherapy -whole body PUVA
4. Miscellaneous - Sulfasalazine, IVIg

OTHERS

- Psychotherapy – hypnotherapy, systemic desensitization
- Supportive therapy – Dermatography(tattooing) , hair pieces or wigs
- Investigational therapy – Recombinanthuman bone morphognic protein , intralesional candida antigen

TOPICAL THERAPY

The mode of action and effects of various modalities are as follows.

TOPICAL STEROIDS[2,53]:

Corticosteroids because of their anti-inflammatory action have been used widely in the management of alopecia areata. Topical steroids of class I to V are effective in Alopecia areata, but take several months for elicitation of hair growth. Most studies have used topical flucinolone and halcinonide. Side effects include acne, hypertrichosis and folliculitis ,due to inadvertent transfer from scalp. Local epidermal atrophy can occur when potent steroids are used.

INTRALESIONAL STERIODS[3,54,55]

Intralesional steroids are considered the first line therapy for adult patients with less than 50% of the scalp involvement. Concentrations ranging from 2.5-10mg/ml of Injection triamcinolone acetonide may be used. Preferred dose is 5mg/ml (maximum volume, 3ml). It is injected intradermally with a 0.5 in long 30gauge needle as multiple 0.ml injections at 1 cm intervals. Treatment is repeated every 4 to 6 weeks. Initial regrowth is often seen in 4-8 weeks. Response

rates up to 86% have reported. This mode of treatment is well tolerated by adults and the predominant side effect noticed was transient atrophy.

TOPICAL IRRITANTS

These agents rely on the induction and maintenance of skin inflammation by repeated application of an irritant.

ANTHRALIN[3,56] :

Anthralin has been used in treatment of AA in concentration of 0.25% to 1.0%.It may be have a non specific immunomodulating effect in eliciting hair growth. Clinical irritation is not necessary for its efficacy and short contact therapy is effective and cosmetically acceptable regrowth has been reported to vary from 20 to25%. Anthralin 0.5% to 1% cream may be applied overnight or as a short contact therapy initially for 30 minutes and gradually increased to 1 hour with 1% antharlin cream. When therapy is effective new hair growth is seen usually within 3 months. It may take 24 or more weeks for cosmetically acceptable response. Adverse effects include irritation, scaling, folliculitis and regional lymphadenopathy. Patients should be advised to protect treated area from sunlight, avoid getting anthralin into the eye and be warned about staining of the treated skin and clothes that come in contact with anthralin.

PHENOL[57,58] :

Phenol is well known as an antiseptic agent. It is also attributed to have antipruritic, antibacterial (>1%), antifungal (>1.3%), and a local anaesthetic property. At a concentrations over 80%, phenol coagulates proteins. Pertaining to

its easy availability and affordability, it is being used in many office procedures in dermatology clinics. The mechanism of action is based on its irritating property that leads to tissue necrosis. A study done by siddhi chikhalkar et al on fifty patients treated with 88% phenol at three weekly interval showed 78% clinical response.

CONTACT SENSITIZERS[2,3,59-62]

Topical immunotherapy is the most effective and an accepted modality in the treatment of AA. The mechanism of action of local sensitizers is by immunomodulation. There is a decrease in the peribulbar CD4/CD8 Lymphocyte ratio, a shift in the position of T lymphocytes away from perifollicular areas to the interfollicular areas and dermis. Two concepts of local immunomodulation have been advanvced. First being that effector T cells are attracted in that area ,there is probably a localised antigen competition ocuring. The second being that repeated application activates non specific suppressor responsible for AA. Three contact sensitisers have been used in AA namely dinitrocholrobenzene(DNCB), Squaric acid dibutyl ester (SADBE)and diphenyl cyclo propanone (DPCP).The use of DNCB has been discontinued because of its mutagenetic effect.

SADBE is an ideal immunogen in that it is a strong topical sensitiser ,it is not found in the natural environment ,does not cross react with other chemicals and it not mutagenic . 29% to 87% of the patients report successful regrowth of hair after the use of SADBE. The disadvantage of SADBE is that is not as stable.

Efficacy of DPCP in the treatment of AA range from 4% to 85%. However during long term follow up it has been noticed that, a 3rd of the responders with cosmetically accepatable results eventually stop responding to the immunogen. Initial responses are seen 12 weeks after therapy and cosmetically acceptable results at 24 weeks. If there is no response by 12 weeks, DPCP topical immunotherapy is discontinued. DPCP is applied as 92% solution in acetone with a wooden applicator to a 4x4 cm area on one side of the scalp. If after 1 week there is no eczematous response, 0.001% solution is applied to the same half of the scalp. If the initial eczematous reaction is severe, treatment is delayed by 1 week.

The aim of the treatment is to maintain a low grade tolerable erythema and pruritus on the treated side for 24 to 36 hours after application. The applications are repeated weekly and the concentrations are titrated according to the severity of previous weeks reactions. Concentrations used vary from 0.0001%to 2%.The DPCP is applied and left for 24 hours. As DPCP is degraded by light, the scalp should be protected from light. Once hair growth is established on one side the next side is treated. Untoward effects includes eczematous reactions with or without blistering, or extension of allergic contact dermatitis to other areas, itching and edema of the face and scalp, regional lymphadenopathy and post inflammatory hyper or hypopigmentation. Contact urticaria, vitiligo and erythema multiforme have also been reported. DPCP is contraindicated during pregnancy.

PHOTOTHERAPY[63,64]

Orally administered PUVA therapy has been one among the numerous available modalities in the treatment of alopecia areata. The use of Oral PUVA is often limited by side effects. Hence PUVA has been used topically. It has also been used in the form of turban, where towels soaked in 0.00001% of 8 methoxypsoralen are wrapped around the patients head in a turban fashion,for a period of 20 minutes and then it was followed by PUVA radiation.

PUVA therapy results for alopecia areata are highly varied. The major problem with PUVA therapy is the high relapse rate. Circumscirbed lesions respond better than total alopecia and total body PUVA is probably more effective than local irradiation. Follow up studies of large group of patients have shown that PUVA in general is not an effective treatment option.

Phototherapy with NB UVB have been tried in various studies. It is given initially thrice a week. Initial treatment dose was determined according to the skin phototype: 0.2J/cm2 for skin phototype II and 0.3J/cm2 for phototype III. Treatment continued with 20% dose increases at each subsequent session to a maximum of 1.8J/cm2.

The dose and frequency of treatments were gradually reduced once satisfactory clinical responses were achieved. The frequency was reduced to twice per week when terminal hair regrowth was seen with no further abnormal hair fall, then once per week, and finally terminated when regrowth was maintained.

CRYOTHERAPY[65]:

Lei et .al used a cotton swab dipped in liquid nitrogen to the patches of AA, for 2 freeze thaw cycles of 15 seconds each repeated once in two weeks. Mechanism of action of liquid nitrogen may be due to improved blood circulation to the hair follicles thus promoting topical nutrition and acceleration of hair growth.

After initial vasoconstriction with cryotherapy, there is a significant local vasodilatation during the thaw period .Thus, cryotherapy is speculated to dilate the vessels around the affected hair follicles, with an increase in the blood flow leading to follicular hair regrowth. Moreover, local edema and inflammation occurring after cryotherapy may play a role in inducing vasodilatation. Cryotherapy also causes partial damage to keratinocytes, especially the antigenic components of hair folliclekeratin16 and trichohyalin, which are targeted by antibodies and thus decreases perifollicular infiltrate. Cryotherapy may also alter tissue Langerhans cells, which in turn could alter the process of antigen presentation with further decrease in T cell infiltration.

The adverse effects noted were insignificant and included mild swelling, hyperesthesia and mild itching in the treatment area for a few hours following treatment. Liquid nitrogen therefore was suggested as an efficient, simple, cheap modality of therapy without any troublesome side effects.

LASER THERAPY :

Encouraging results have been obtained with excimer laser (308 nm) and pulsed infrared diode laser(904 nm).

OTHER TOPICAL THERAPIES

Minoxidil[66]

Minoxidil is a powerful vasodilator.Used mainly in andrgenetic alopecia topically as 2%–10% solution, this drug has also been found useful in AA. Cosmetically acceptable regrowth was induced in 20%–60% of cases in various studies, higher concentrations being more effective.

Tacrolimus

Topical calcineurin inhibitors have been tried in the treatment of AA. The peculiarity of tacrolimus was that the induction of anagen . However, there has not been a positive result with tacrolimus 0.1% applied twice daily even after 24 weeks in patients with AA.

Topical retinoids

Among topical retinoids, tretinoin and bexarotene have been tried in alopecia areata with mixed results. Irritation of the skin is a very common side effect and the efficacy is doubtful in the absence of double-blind randomized trials.

Prostaglandin analogs[67]:

The adverse effect of certain prostaglandin analogues used as anti-glaucoma eye drops to cause hypertrichosis has been employed in treatment of alopecia areata. These prostaglandin analogues include Latanoprost and Bimatoprost and they are used in the treatment of alopecia areata involving the eyelashes. However, the results have not been really encouraging.

SYSTEMIC THERAPY:

SYTSEMIC STEROIDS[1,2,3,68]:

Systemic steroids will restore normal hair growth in many cases of AA. Systemic steroids have probably a place in controlling the spread of rapidly progressing Alopecia areta. They are frequently effective but their use is limited because of their adverse effects and high relapse rate after reduction of the dose. Systemic steroids have been used in the following regimens.

A) One treatment regimen is to use 40 to 60 mg of prednisolone and to taper by 5mg every week.
B) Oralmini pulse therapy with betamthasone 5mg on two consecutive days for 3-6months
C) Dexamethasone has been used as oral pulse on two consecutive days every week. The dose being 5mg in patient patient above 12 years and 2.5 to 3.5 mg in children less than 12 years.
D) Pulse therapy with intravenous methyl prednisolone (500mg) for 3 successive days have also been used.

PUVA[63]

Oral PUVA has been used in the treatment of alopecia areata. In one study PUVA was administered to the whole body 3 times a week in patients suffering from extensive AA. This study concluded that PUVA could be used as an alternative therapy with extensive AA. However the long term utility of PUVA therapy is in doubt, as only 10% patient had cosmetically acceptable regrowth.

SULFASALAZINE[69]

Sulfasalazine via the inhibition of inflammatory cells chemotaxis and cytokine antibody production to produce satisfactory results. Recommended dose is 0.5g twice daily for first month, then 1g twice daily for second month, 1.5 g for another three months. Complete regrowth was found in 27.3% and partial regrowth was foundin 40.9%.

CYCLOSPORINE[70]

Cyclosporine is an immunosuppressant drug isolated from Norwegian fungus Tolypocladium inflatum. It inhibits helper t cells activation and suppresses interferon gamma production. it also prolongs the anagen phase of hair growth causing hypertrichosis as a cutaneous side effect.It is used either alone or in combination with systemic steroids.

MISCELLANEOUS

Methotrexate[71] had shown hair growth in 57% AA patients and when combined with prednisone, the efficacy was 63-64%

Azathioprine[72] was tried in an open label study by Farshi et al. at a dose of 2 mg/kg/day for 6 months and reported 52.3% mean regrowth, using SALT score. Supplementation of anti-histamines like fexofenadine and ebastine have been found useful in AA, associated with atopic dermatitis.

Biologics[73] like infliximab, etanercept, adalimumab and efalizumab have been tried, and all o them were unsuccessful. Some showed worsening or occurring of AA while on treatment with biologics. Abatacept has been found effective to prevent AA by blocking T-cell activation in experimental mouse models and was proposed for further clinical tests in AA. Capsaicin was tried in a recent randomized non-blinded study in 50 patients with patchy AA in comparison with 0.05% clobetasol ointment for 6 weeks, and 9.5%showed cosmetically acceptable regrowth at week 12 in both groups.

A combination of topical 5% garlic gel and betamethasone 1% cream for 3 months showed statistically significant hair growth in more number of patients when compared to betamethasone and placebo.

Tincture iodine has been suggested as a non-specific irritant in patchy AA patients with less than 10 years of age. Topical bexarotene 1% gel was evaluated in single-blinded half head study and found 50% or more partial regrowth in 12% of patients on the treated side and in 14% responded on both sides.

Fractional Er: Glass laser has shown complete hair regrowth in a single patient who was not responding to conventional therapies.

Topical tretinoin 0.05%, topical azelaic acid, inosiplex, topical onion juice, and intralesional candida antigen injections have been tried with some efficacy and need to be confirmed in large-scale, double-blind, placebo-controlled trials.

New drugs are being tried based on the GWAS against T-cell mechanisms and NK-cell activating ligands. Many drugs like anti-CD25, anti-CTLA-4, anti-IL-6, anti-IL-15, and Syk inhibitor, which are being evaluated in other autoimmune diseases which affect these mechanisms,can be the potential therapies for clinical trials in AA.

CAMOUFLAGE[74]

At times, when there was no regrowth after various treatments, Camouflage techniques like hairpieces and hair additions may be tried. Hairpieces could be in the form of wigs, toupees, cascades and wiglets. Human hair wigs are most expensive, needs regular shampooing and lasts only 2-3 years. Synthetic hair fibers are less expensive, needs less maintenance and lasts for3-5 years.

Future Therapeutic Options

Recombinant human bone morphogenetic protein is involved in hair morphogenesis. A patient of alopecia universalis who has put on this medication to hasten bone fracture healing showed dramatic hair growth all over the body. JAK (Janus kinase) inhibitors – oral Tofacitinib (JAK 1and 3 inhibitor) and ruxolitib (JAK 1 and 2 inhibitor) are the promising emerging drugs.

Aims & Objectives

AIM OF THE STUDY

- To study the efficacy of various modalities of treatment used in alopecia areata and to compare them with one another
- The various modalities of treatment used in our present study are
    - Intralesional steroid with triamcinalone acetonide (10mg/ml)
    - Cryotherapy with liquid nitrogen
    - Topical phenol (88%)
    - Spot phototherapy with NBUVB

Materials and Methods

MATERIALS AND METHODS

Study Design:

Prospective observation study

Study approval :

Prior to commencement of this study - Thesis & Ethical Committee of Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai had approved the thesis protocol.

Place of study :

Department of Dermatology, Madras Medical College &

Rajiv Gandhi Govt. General Hospital ,Chennai – 600003

Period of study :

Duration starting from August 2017 to July 2018.

Patients presenting with alopecia areata to dermatology department of our hospital during the study period from August 2017 to July 2018 were recruited in the study. A diagnosis of Alopecia areata (AA) was made on clinical grounds, based on the following criteria.

Asymptomatic, circumcised, smooth, non scaly, non scarring patch of hair loss on the scalp. In doubtful cases a mycology opinion as obtained and fungal scraping from the patch was done to rule out tinea capitis. The age and sex of the

patients recruited were noted. A thorough history was elicited with regards to duration of symptoms, progression of disease, history of past episodes, history of any treatment , personal and family history of atopy, diabetes mellitus and hypertension and history suggestive of any other autoimmune diseases were also elicited.

A meticulous general and systemic examination was performed. Complete dermatological examination was performed taking care to note any other dermatoses in the patient. Nail changes and mucosal changes were noted.

The location of the patches of AA, with reference to the size, presence of hair in the patches was also made. The presence of exclamation mark hair , coudability sign and pluckablity of hair at the margins was also recorded.

A dental and ENT opinion was sought to rule out septic foci. A complete haemogram was carried out in all patients and a random blood sugar in adults was done. The individuals with the history of exposure to the risk of sexually transmitted disease were subjected to a blood VDRL examination.

After completing the examination 100 patients were taken in the therapeutic study based on the following criteria.

INCLUSION CRITERIA :

- Patients having only scalp involvement.
- Patients who had no treatment either topical or systemic in the past 3 months.

- Before starting the therapy appropriate treatment of focal sepsis if any were given to the patient.

EXCLUSION CRITERIA :

- Patient not willing for study
- Patient with alopecia universalis , totalis
- Patients with oophiasis and sisaipho pattern
- Patients with other systemic illness – cardiac , renal ,hepatic illness, severely Immunocompromised

Pregnancy

- Patients who have cold intolerance
- Patients taking photosensitive drugs
- Patients with history of photosensitivity

A total number of 100 patients were included in the study. A written informed consent was taken from each patient before starting the study.The patients were divided into 4 groups randomly , comprising 25 in each group and the following therapy was initiated..

GROUP I : PATIENTS TREATED WITH INTRALESIONAL STEROID

Corticosteroids, because of their anti-inflammatory activity, have been the mainstay of therapy for AA. They have been used topically, orally, and parenterally. Intralesional corticosteroids have been used since 1958 in the

treatment of AA, and it is the treatment of choice for adults in patchy AA, with approximate success rates of 60-75%

Twenty five patients comprising the first group were treated with intralesional steroid (triamcinalone acetonide). Injection triamcinalone acetonide at concentration of 10mg/ml was given deep into the dermis using a 0.5-inch long 30-gauge needle at multiple sites, 1 cm apart and 0.1 ml into each site. The maximum dose should not exceed 20 mg for each visit. The treatment is repeated once in 4 weeks. The patients were followed up once in 2weeks till the end of study period.

GROUP II : PATIENTS TREATED WITH CYOTHERAPY

The second group of twenty five patients was given cryotherapy with liquid nitrogen. It was applied over the patches of AA with a cotton wool liquid applicator producing 2 – 3 freeze thaw cycles at an interval of 2 – 3 seconds until an erythema developed over the patches. The treatment was repeated once in two weeks.

GROUP III : PATIENTS TREATED WITH LIQUID PHENOL

Full strength liquefied phenol was prepared by keeping the bottle containing phenol crystals in hot water until dissolution of crystals. It was further diluted with water to make 88% phenol solution (88 ml liquefied pure phenol in 100 ml water).

The patch was degreased by scrubbing with savlon , followed by spirit and acetone. In a small glass container, 0.5-1 ml 88% phenol was taken which was then applied gently with thin cotton tipped applicator until an ivory white uniform frosting was seen. Neutralization is not required. Treatment was repeated at 3 weekly intervals.

GROUP IV : PATIENTS ON PHOTOTHERAPY

For the last group of twenty five patients phototherapy was given with hand held NB-UVB device directly over the AA patch. Patch was exposed to NBUVB with initial dose of 200 mJ/cm ^{2}, thrice weekly with increments of 10% at every week, till minimal erythema developed.. After that, follow-ups were done fortnightly completing a total period of 12 weeks.

The patients were reviewed once in 15days. All the selected patches were assessed clinically as well as photographically at the baseline, at each visit, and at each follow-up visit. The following changes were noted and recorded.

Increase in size of the lesions.

Appearance of new lesions

Response to therapy was noted as follows

- No regrowth of hair
- Appearance of vellus hair
- Growth of pigmented normal hair
- Extent of regrowth

Complication of therapy like

- Burning and irritation
- Erythema
- Vesiculation
- Pigmentary changes
- Folliculitis
- Atrophy

Patient's response to the treatment was graded as follows :

0-25%	A	Hair regrowth covering < 25% of the patch. Very poor response
25-50%	B	Hair regrowth occurs, but is not cosmetically acceptable to the patients. Poor response
50-75%	C	Hair regrowth is cosmetically acceptable and cover ¾ of the patch. Moderate response
75-90%	D	Hair regrowth covered almost all of the patch, but the hair was not of similar density to the surrounding hair. Good response
90-100%	E	Complete regrowth of hair, which is indistinguishable from the surrounding area. Excellent response

Results were analyzed photographically as well as statistically (using Chi-square test) after a follow-up period of 12 weeks.

For the purpose of statistical analysis, comparison & interpretation patients with excellent, good & moderate response were considered to have cosmetically acceptable regrowth and those patients with very poor and poor response were considered to have unacceptable regrowth.

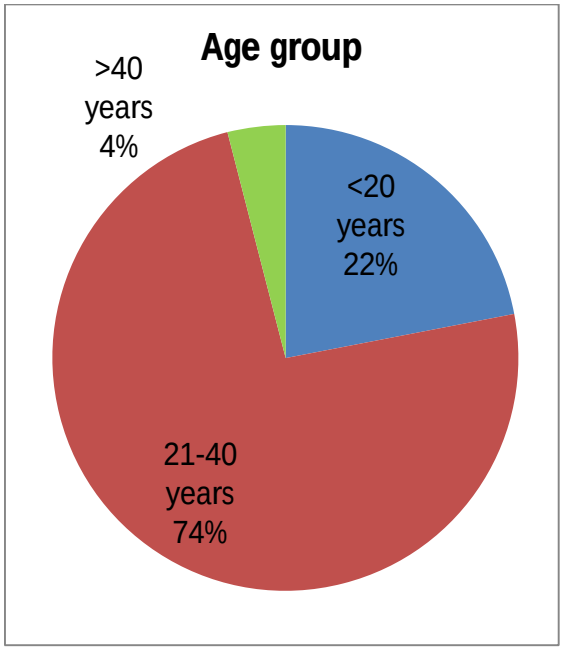
Observations & Results

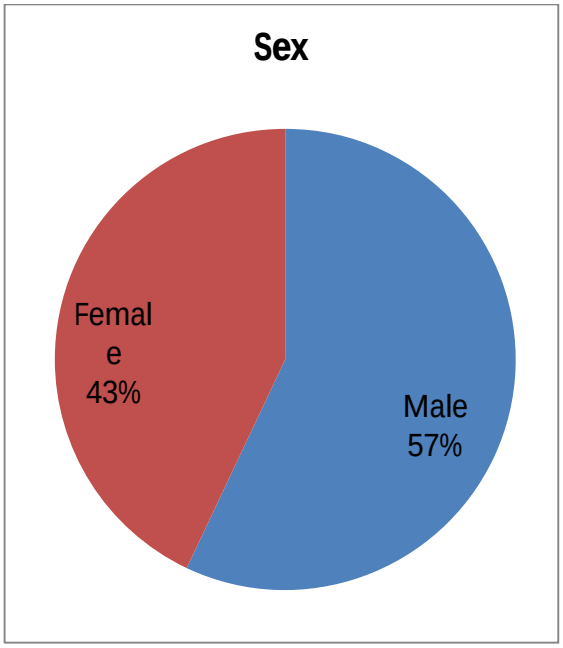
OBSERVATIONS

An analysis of the clinical profile of the patients recruited for the therapeutic response revealed the following data.

There were 43(43%) females & 57(57%) males in the study groups.

The age of the patient ranged from 7 to 49 years and the mean age is 27.48years (SD - 7.87years).



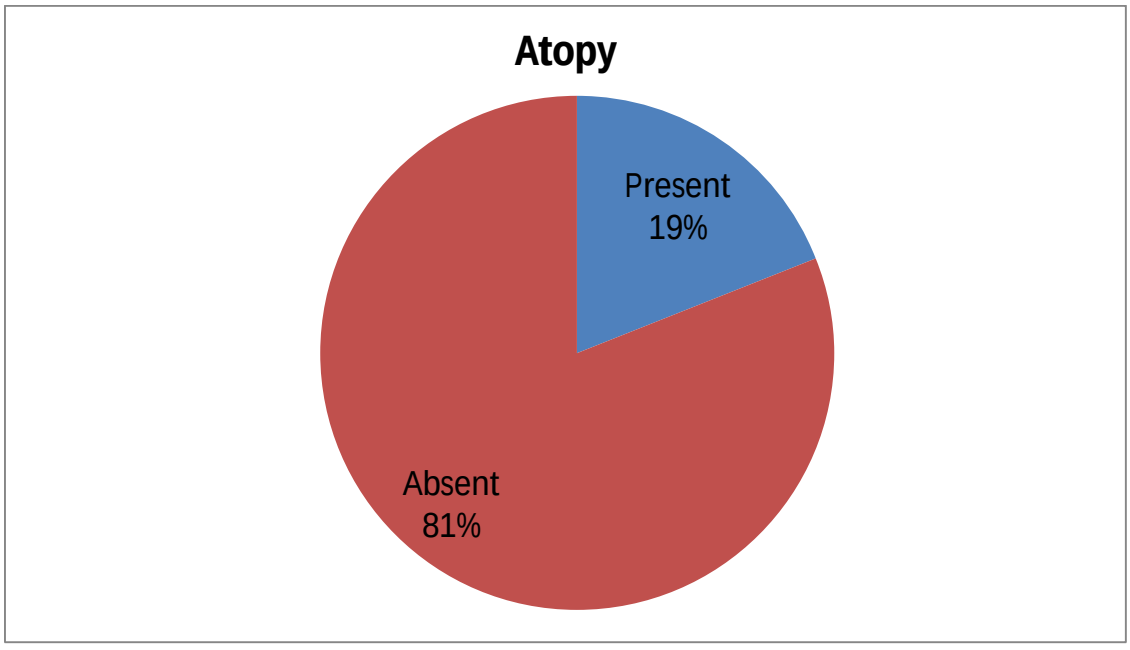


The duration of the disease ranged from two weeks to 2 years, average duration being 5.65 months (SD - 4.75).

Variables		Intralesional steroid	Cryotherapy	Topical phenol	Spot phototherapy
Duration (months)	Mean	4.90	6.34	7.34	4.02
	SD	3.77	5.11	5.76	3.58

11 patients had a family history of atopy. There was a personal history of atopy in 19 patients in the form of chronic urticaria (8), allergic rhinitis (6), bronchial asthma (3), pityriasis alba (1) atopic dermatitis in one patient.

Variables		Frequency N (%)
Atopy	Present	19 (19%)
	Absent	81 (81%)



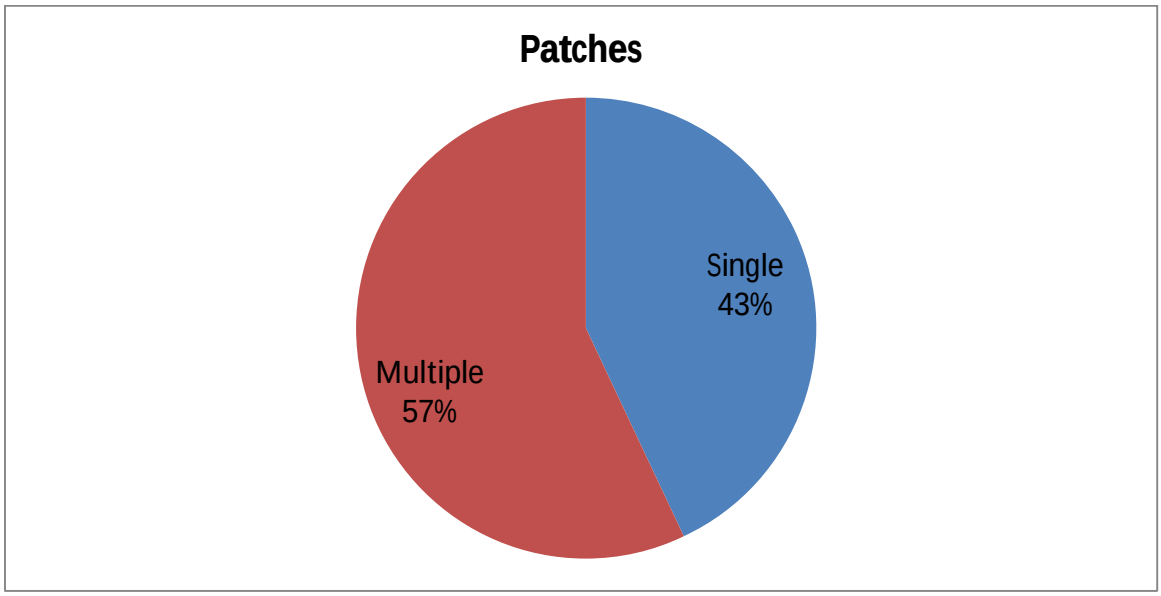
Nine patients had association of other autoimmune disorders which includes multinodular goitre in three patients, vitiligo in three patients, hypothyroidism in two patients and lichen planus in one patient. One patient with multinodular goitre was in addition on antipsychotic medication

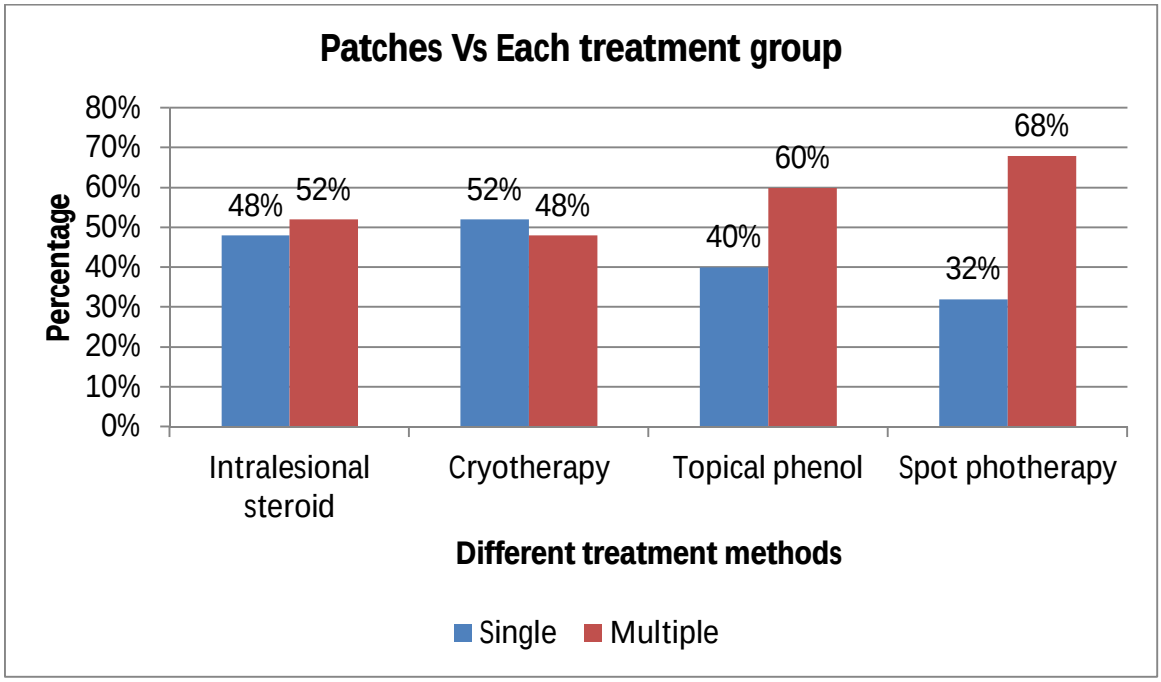
10 patients had past history of alopecia areata . In addition to the family history of atopy recorded earlier, 22 & 15 patients had family history of DM and hypertension respectively.

Examination of the patches of AA revealed the following:

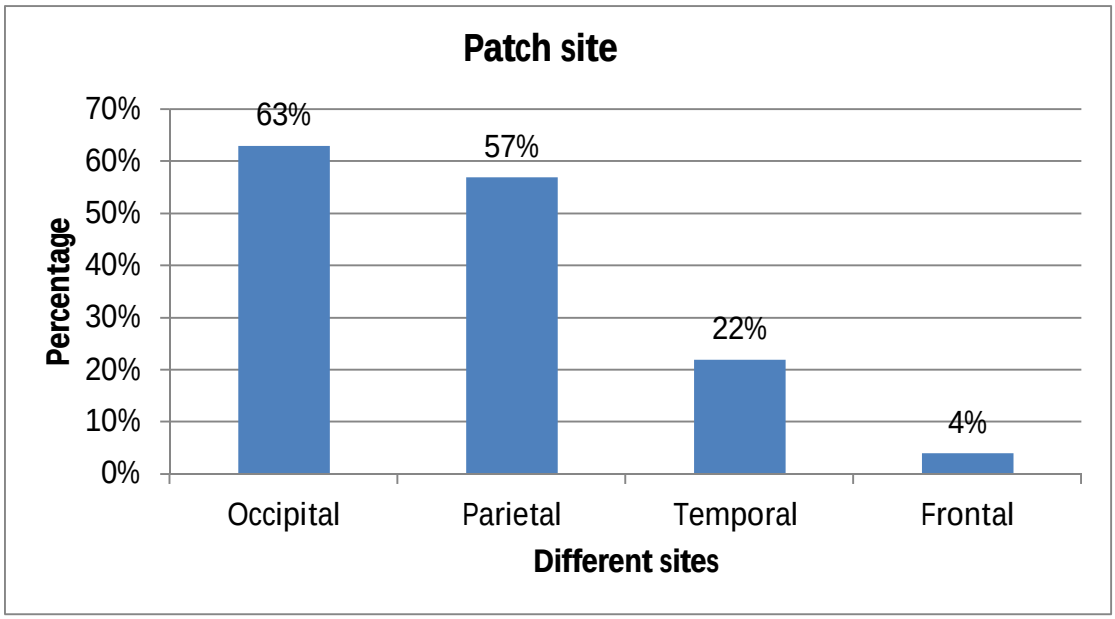
The number of patches varied from 1 to 8, the average being 2 (SD-1). The size of the smallest patch was 1x1cm and the size of the largest patch was 5 X 4 cm. The coudability sign and exclamation mark hair were positive in 69 and 81 patients respectively.

Out of 100 patients, 43 patients (43%) had single patch and remaining 57 patients (57%) had multiple patches.





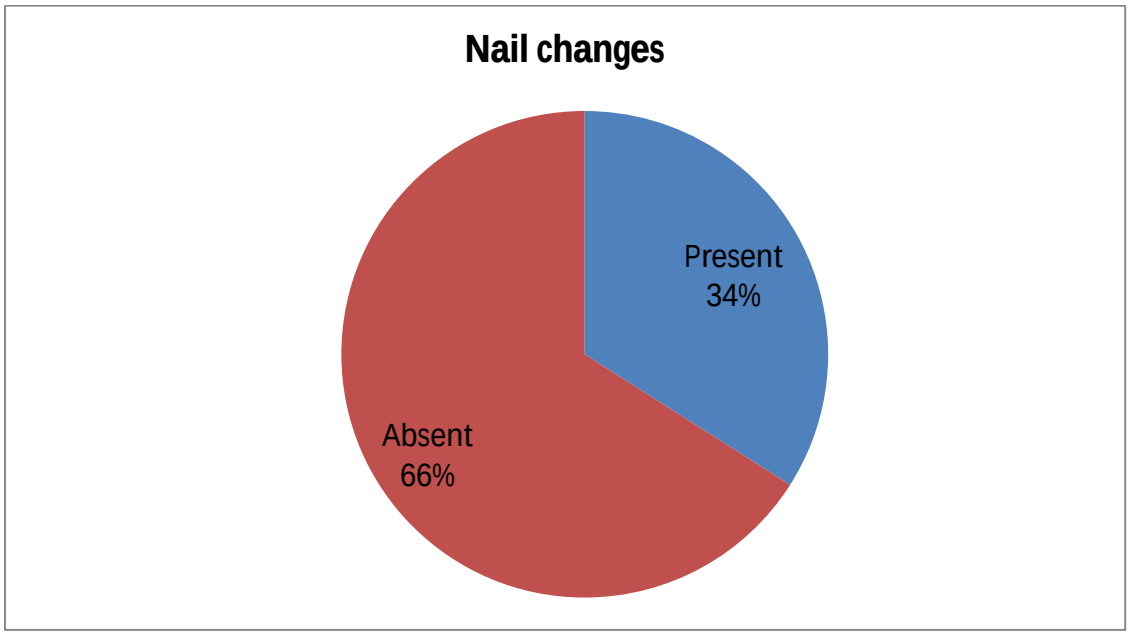
The region wise distributions of the patches were occipital (63), parietal (57), frontal (4) and temporal (22).



The nail changes were noticed in 34 patients which includes – pitting in 18 patients, leuconychia in 7, longitudinal ridges in 6, bues line in 1, melanonychia in 1, onycholysis in 1 patient. One patient had pitting associated with leuconychia,

1patient had pitting and onycholysis and 2 patients had pitting and trachyonychia. Two patients had hyper pigmentation of oral mucosa.

Variables		Frequency N (%)
Nails changes	Present	34 (34%)
	Absent	66 (66%)



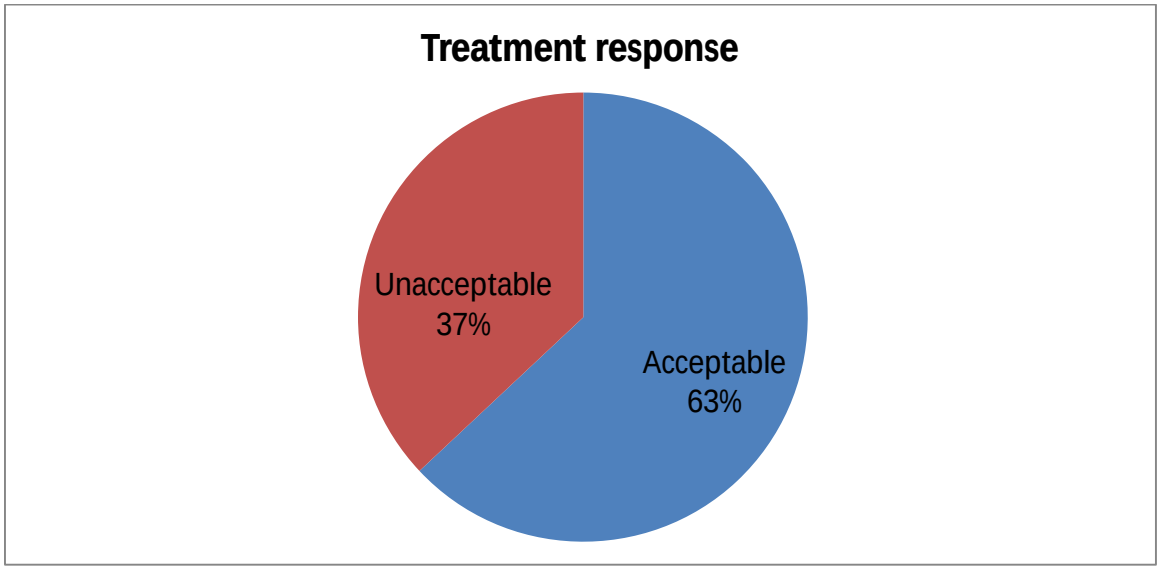
Other associated dermatological manifestations were dermatophytosis (2), psoriasis, verruca vulgaris, lichen simplex chronicus, intertrigo and seborrheic dermatitis each in 1 patient. Clinical examination of focal sepsis revealed chronic gingivitis in 1 patient, dental caries in 5 and pharyngitis in 1 patient.

Eight patients were found to be diabetic and 3 patients had systemic hypertension, all patients were on regular treatment.

OBSERVATION OF THERAPEUTIC RESPONSES

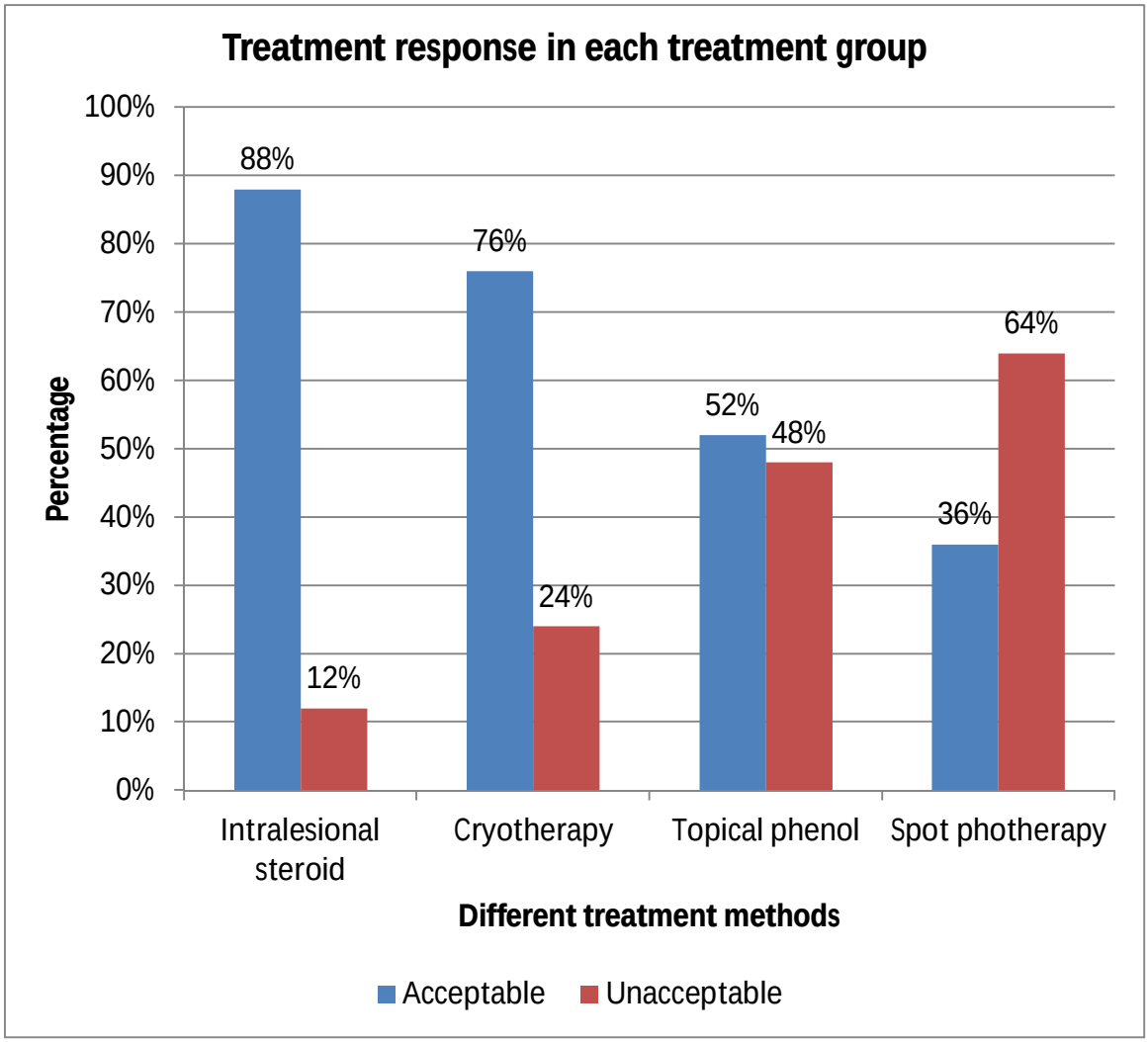
In our study group of 100 patients, there was no significant difference in the mean age and gender distribution among different study groups.

Acceptable regrowth (excellent, good and moderate) was found in 63 patients (63%) and remaining 37 patients (37%) had cosmetically unacceptable regrowth (poor and very poor response)

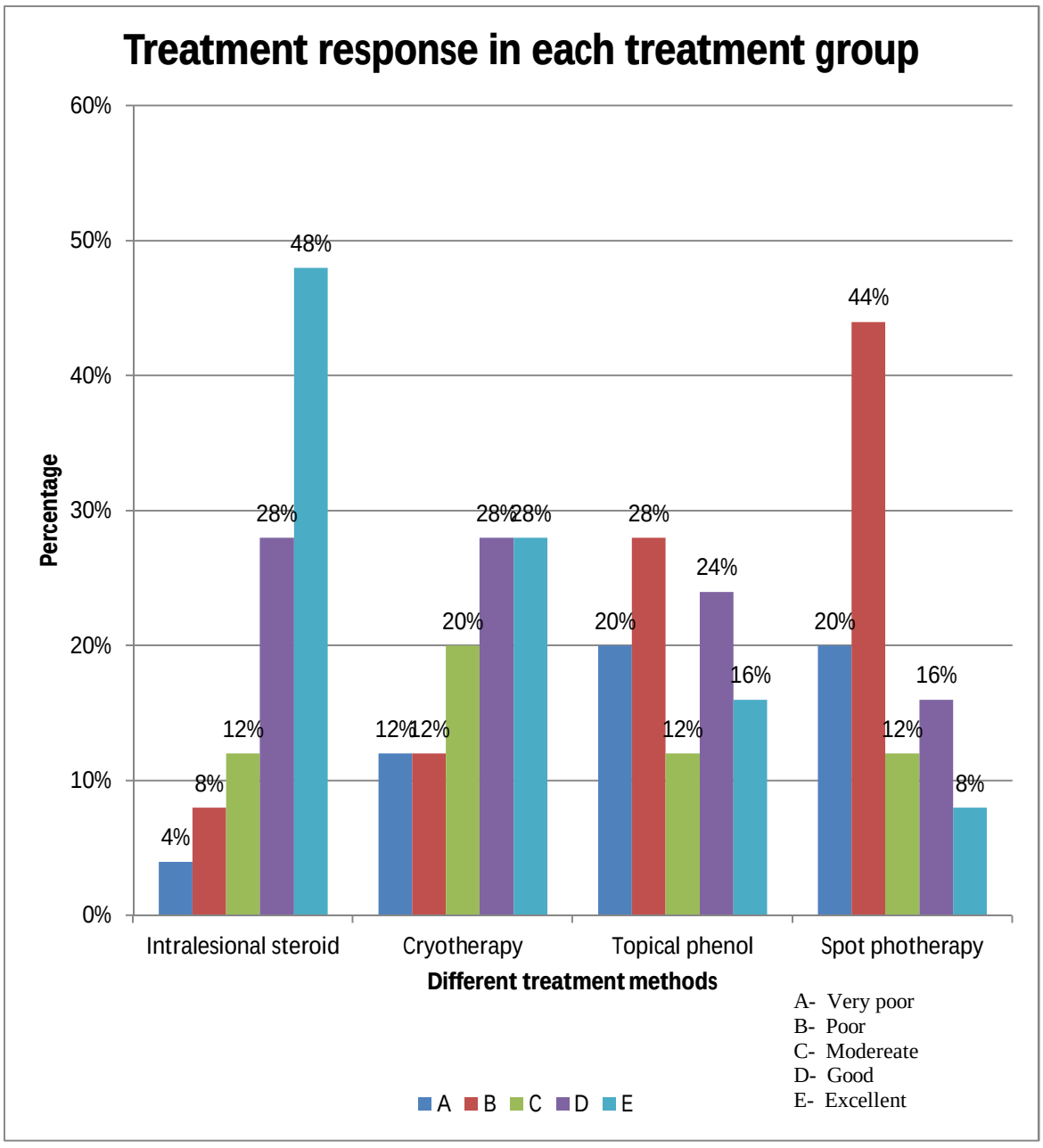


S No	Variables		Intralesional steroid	Cryo therapy	Topical phenol	Spot phototherapy
			N (%)			
1	Response	Acceptable	22 (88%)	19 (76%)	13 (52%)	9 (36%)
		Unacceptable	3 (12%)	6 (24%)	12 (48%)	16 64%)

Cosmetically acceptable regrowth of hair was seen in 88% of patients treated with intralesional steroids, 76% of patients with cryotherapy, 52 % in patients treated with phenol and 36%of patients treated with phototherapy with NBUVB.

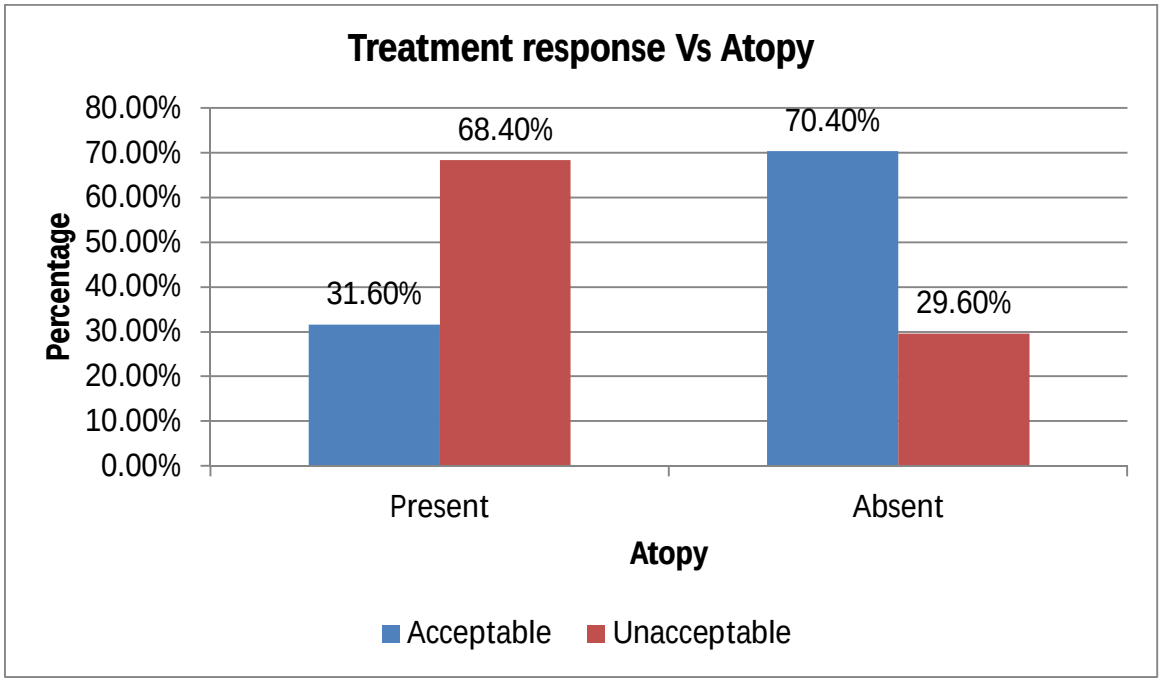


Excellent response occurred in 48% (12patients) treated with ILS, 28% (7patients) with cryotherapy,16% (4patients) with phenol , 8% (2 patients) with NBUVB.



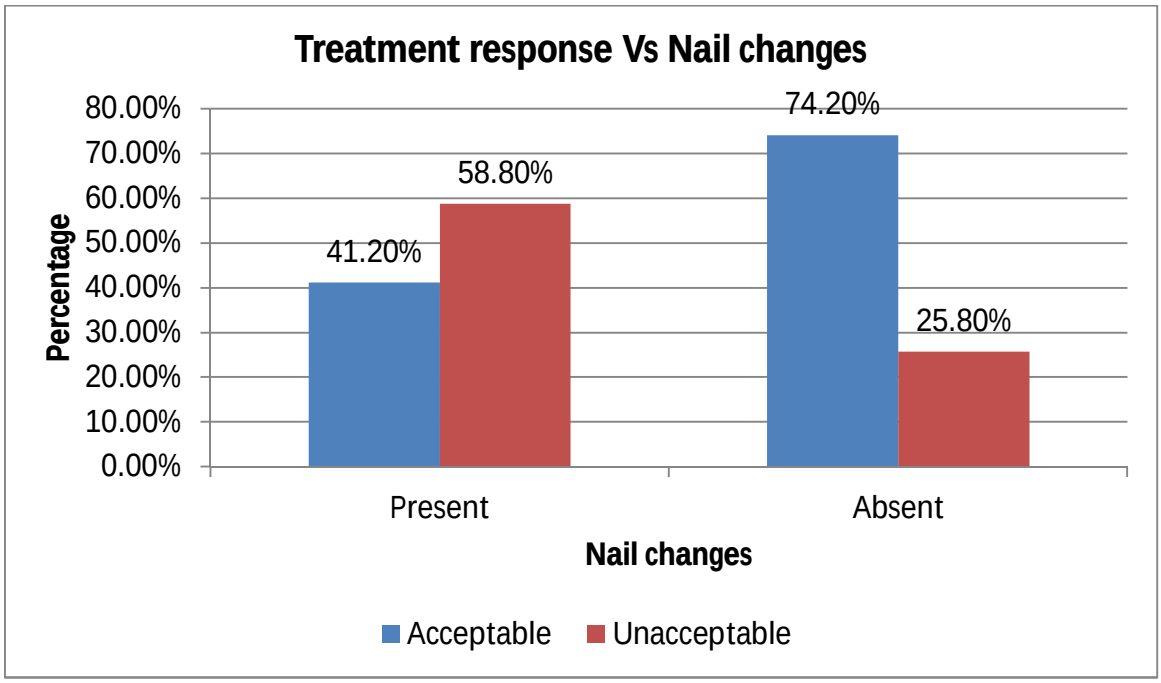
Atopic patients responded poor to the treatment comparing to non atopics and this difference is statistically significant.

Variables		Response	
		Acceptable	**Unacceptable**
Atopy	Present	6 (31.6%)	13 (68.4%)
	Absent	57 (70.4%)	24 (29.6%)
Chi- square value – 9.93 p value 0.002 (statistically significant)			



There is statistically significant difference in treatment response in patient with associated nail changes and in patients without nail changes.

Variables		Response	
		Acceptable	**Unacceptable**
Nails changes	Present	14 (41.2%)	20 (58.8%)
	Absent	49 (74.2%)	17 (25.8%)
Chi- square value – 10.52 p value 0.001 (statistically significant)			



The number of patches (single/ multiple) among different study groups doesn't influence the response outcome

Treatment type	Variable		Response	
			Acceptable	Unacceptable
Cryotherapy	Patch cat	Single	12 (92.3%)	1 (7.7%)
		Multiple	7 (58.3%)	5 (41.7%)
Chisquare (Fischer exact)- 3.94 p value – 0.07 (not statistically significant)				

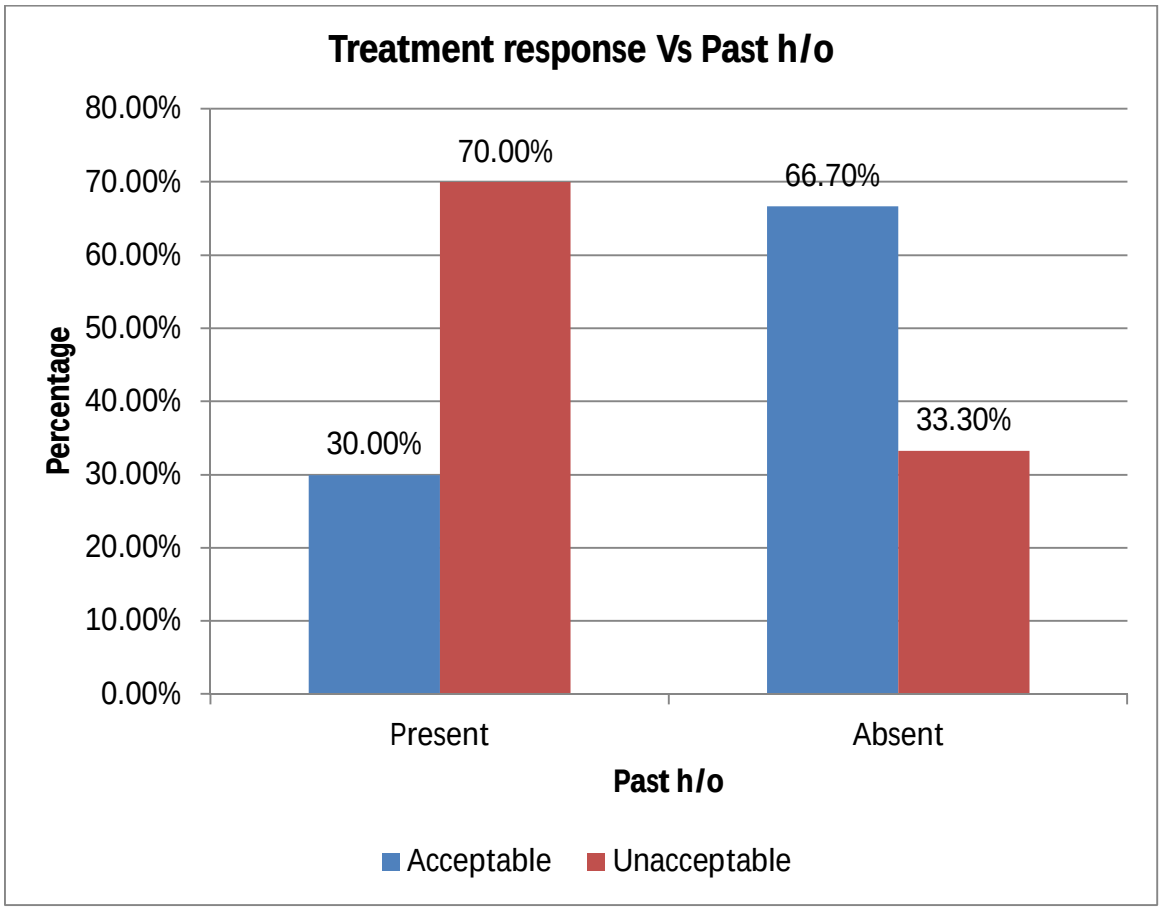
Treatment type	Variable		Response	
			Acceptable	Unacceptable
Intralesional steroid	Patch cat	Single	12 (100%)	0
		Multiple	10 (76.9%)	3 (23.1%)
Chisquare (Fischer exact)- 3.14 p value – 0.22 (not statistically significant)				

Treatment type	Variable		Response	
			Acceptable	Unacceptable
Spot phototherapy	Patch cat	Single	4 (50%)	4 (50%)
		Multiple	5 (29.4%)	12 (70.6%)
Chisquare (Fischer exact)- 1.001 p value –0.394 (not statistically significant)				

Treatment type	Variable		Response	
			Acceptable	Unacceptable
Topical phenol	Patch cat	Single	7 (70%)	3 (30%)
		Multiple	6 (40%)	9 (60%)
Chisquare (Fischer exact)- 2.16 p value – 0.226 (not statistically significant)				

Patients with past history of alopecia areata responded poor than patients with new onset of alopecia areata.

Variables		Response	
		Acceptable	Unacceptable
Pasth/oAA	Present	3(30%)	7(70%)
	Absent	60(66.7%)	30(33.3%)
Chi-square value(Fischer Exact)-5.19 pvalue0.022(statistically significant)			



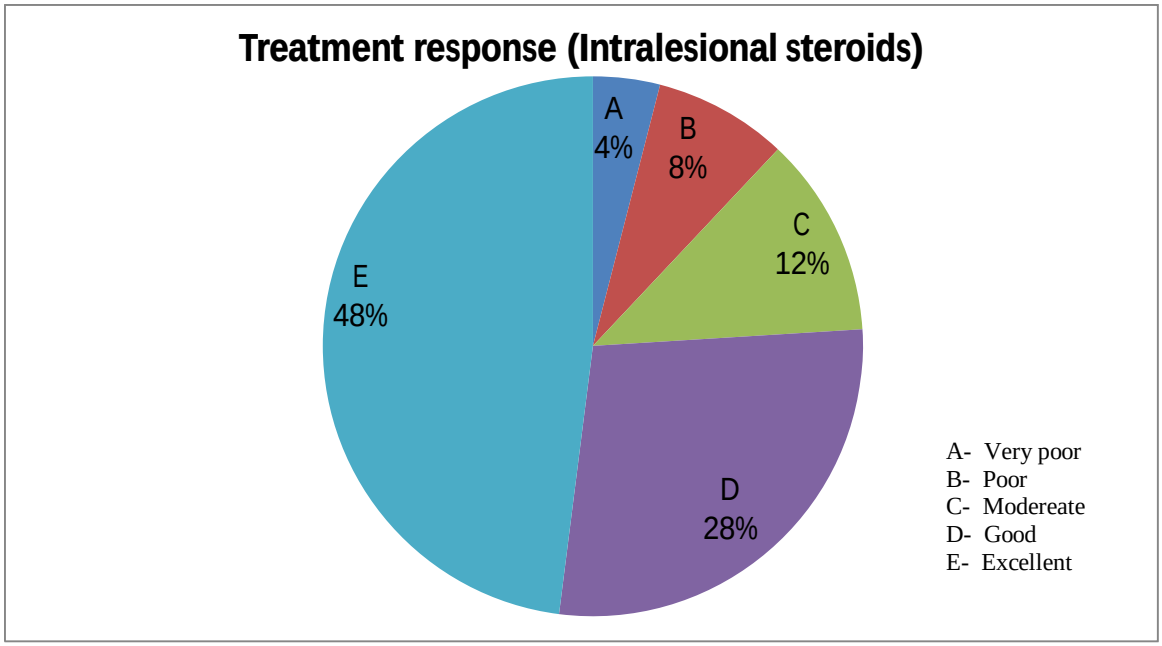
GROUP I: RESULTS OBTAINED WITH INTRALESIONAL STEROIDS.

The mean age of the patients included in this group was 28.36 years and the mean duration was 4.90.

Numbers of patches range from 1 to 3 ,with 12 patients (48%) had single patch and 8 patients (32%) had two patches and remaining5 patients (20%)had 3 patches. The size of patch ranges from 1x1 to 5x3 cm.

Associated nail changes were found in 7 patients (28%). Atopy was found to be associated with 5 patients (20%). 12 patients (48%) had singe patch and multiple patches were found in 13 patients (52%).Past history of AA was found in 3 patients (12%). 4patients (16%) were found to be diabetic and 2 patients (8%) were found to have systemic hypertension.

Among the 25 patients treated with intra lesional steroids, acceptable regrowth of hair occurred in 22 patients (88%) of which 12(48%) patients showed excellent response and 7(28%) patients showed good response & 3(12%) patients showed moderate response. Poor response was seen in 2(8%) patients and very poor response in one patient(4%).



Regrowth of hair started after 4 weeks in seven, after 6 weeks in five patients with excellent response. In patients with poor response regrowth of hair

started only after 8 weeks but the density of hair regrowth continued to be poor during the study period. Patients with very poor response showed no growth at all except for vellus growth of hair which was poor during the entire study period.

One patient with good response and one patient with moderate response developed new patch during the treatment period.

One patient developed atrophy at the treatment site which was transient and resolved spontaneously. Two patients developed mild pain at the injection site.

The initial regrowth of hair was in the form of depigmented hair in 9 patients which showed very slow repigmentation.

Patients who had past history of alopecia areata responded poor to treatment compared to those without past history and this difference is statistically significant (p =0.02).

Treatment type	**Variable**		**Response**	
			Acceptable	**Unacceptable**
Intralesional steroid	Past h/o	Present	1 (3.3%)	2 (66.7%)
		Absent	21 (95.5%)	1 (4.5%)
Chisquare (Fischer exact)- 9.64 p value – 0.02 (statistically significant)				

There is significant difference in treatment response in those patients with associated nail changes (p=0.015). Patients without associated nail changes responded well to intralesional steroids.

Treatment type	Variable		Response	
			Acceptable	Unacceptable
Intralesional steroid	Nail changes	Present	4 (57.1%)	3 (42.9%)
		Absent	18 (100%)	0 (0%)
Chi square (Fischer exact)- 8.76 p value – 0.015 (statistically significant)				

History of atopy doesn't influence the treatment response in this study group (p=0.09).

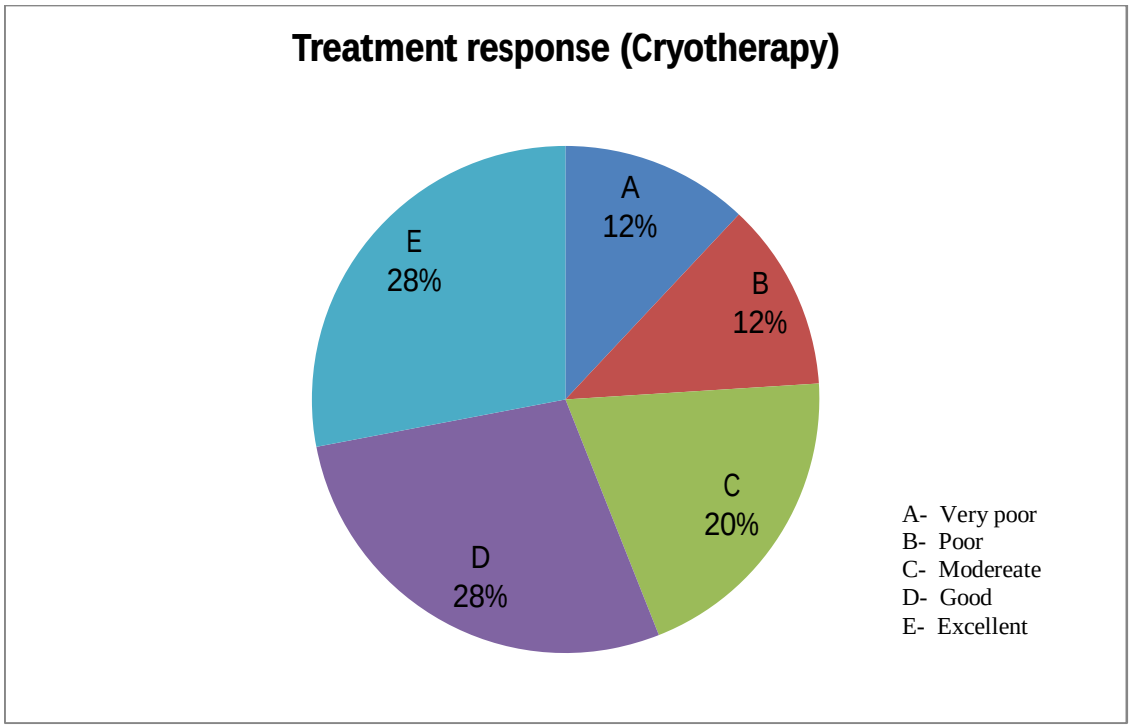
GROUP II: RESULTS OBTAINED WITH CRYOTHERAPY

The mean age of the patients included in this group was 25.92 years and mean duration was 6.34 months.

Numbers of patches range from 1 to 4, with 13 patients (52%) had single patch and 7 patients (28%) had two patches, 4 patients (16%) had 3 patches and remaining one patient (4%) had patches.

The size of patch ranges from 1x1 to 5x4 cm. Associated nail changes were found in 9 patients (36%). Atopy was found to be associated with 7 patients (28%). 13 patients (52%) had singe patch and multiple patches were found in 12 patients (48%).Past history of AA was found in 4 patients (16%). 1patients (4%) was found to be diabetic.

Acceptable regrowth of hair occurred in 19 (76%) patients of which 7(28%) patients had excellent response and 7(28%) patients showed good response & 5(20%) patients showed moderate response. Poor response was seen in 3(12%) patients and very poor response in 3 (12%) patients.



In patients with excellent response, regrowth of hair started by 4 weeks in four patients and by 6 weeks in three patients. In patients with good response, regrowth started by four weeks in 4 patients and by six weeks in three patients. In patients with poor response, regrowth started by 8th week in two patients and by 10th week in one patient.

One patient with excellent response showed initial increase in extension of patch at the margins but it was improved with subsequent treatment.

The untoward effects noticed by the patients at the time of treatment were erythema in two patients and edema in two patients which was bearable.

In this group, the association of atopy with treatment response is statistically significant (p=0.032). Patients without history of atopy responded well.

Treatment type	**Variable**		**Response**	
			Acceptable	**Unacceptable**
Cryotherapy	Atopy	Present	3 (42.9%)	4 (57.1%)
		Absent	16 (88.9%)	2 (11.1%)
Chisquare (Fischer exact)- 5.85 p value – 0.032 (statistically significant)				

The severity of nail changes influenced the treatment response which is statistically significant (p=0.012).

Treatment type	**Variable**		**Response**	
			Acceptable	**Unacceptable**
Cryotherapy	Nail changes	Present	4 (44.4%)	5 (55.6%)
		Absent	15 (93.8%)	1 (6.3%)
Chisquare (Fischer exact)- 7.67 p value – 0.012 (statistically significant)				

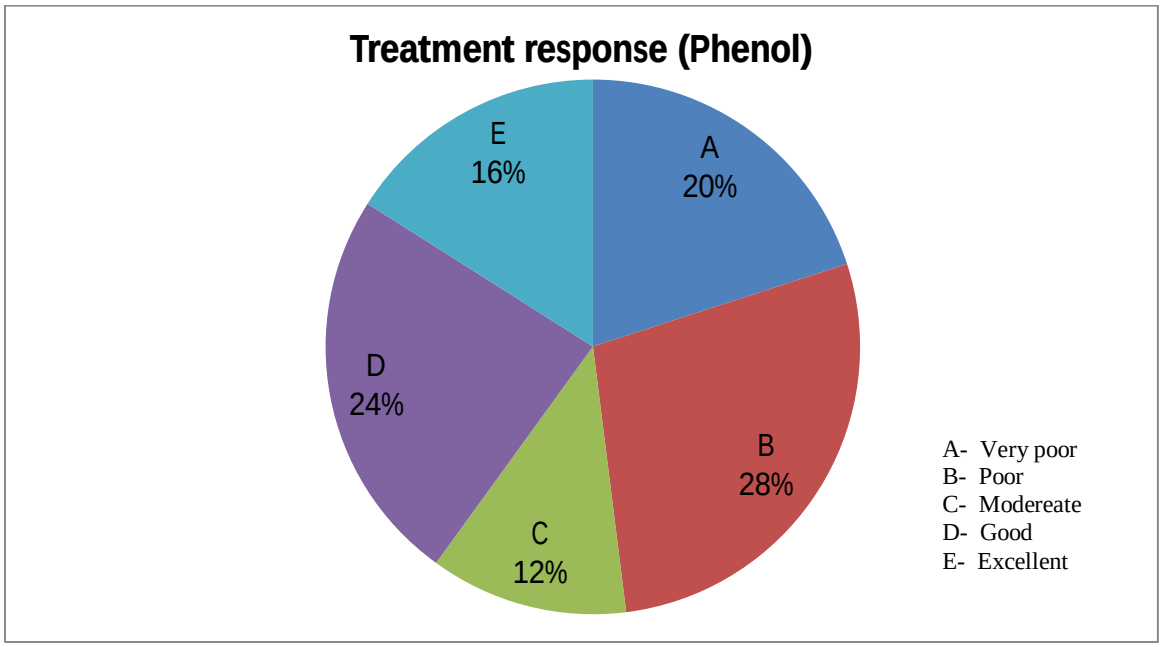
There is no significant association of treatment response with number of patches.

GROUP III: RESULTS OBTAINED WITH TOPICAL PHENOL

The mean age of the patients in this group was 29.88 years and the mean duration was 7.34 months.

Numbers of patches range from 1 to 3 , with 10 patients (40%) had single patch and 10 patients (40%) had two patches, 4 patients (16%) had 3 patches and remaining one patient (4%) had 4 patches. The size of patch ranges from 1x1 to 4x4 cm. Associated nail changes were found in 9 patients (36%). Atopy was found to be associated with 4 patients (16%). 10 patients (40%) had singe patch and multiple patches were found in 15 patients (60%).Past history of AA was found in 3 patients (12%). 2 patients (8%) were found to be diabetic.

Among the 25 patients treated with phenol, acceptable hair regrowth was found in 13(52%) patients, of which four(16%) had excellent response, six(24%) had good response and three patients(12%) had moderate response. Poor response was noticed in seven patients(28%) and five patients(20%) showed very poor response.



In patients with excellent response, hair regrowth was started by 4weeks in two patients and 6weeks in two patients. Complete hair regrowth was noted only by end of 12th week. In one of the patients there was increase in size of the patch during therapy but hair regrowth was complete.

In patients with good response, the patients started regrowing hair between 4 and 8 weeks.

In patients with moderate response, regrowth was started by 6 weeks and completed by 10weeks. The patch continued to increase in size at the margins.

The patients with very poor response did not show any evidence of hair regrowth.

Six out of twenty five patients developed adverse effects following treatment. Among them three patients showed hypo pigmentation. Hyper pigmentation, erythema, mild atrophy was seen each in one patient.

Patients with history of atopy responded poor to treatment and this difference is statistically significant (p=0.039).

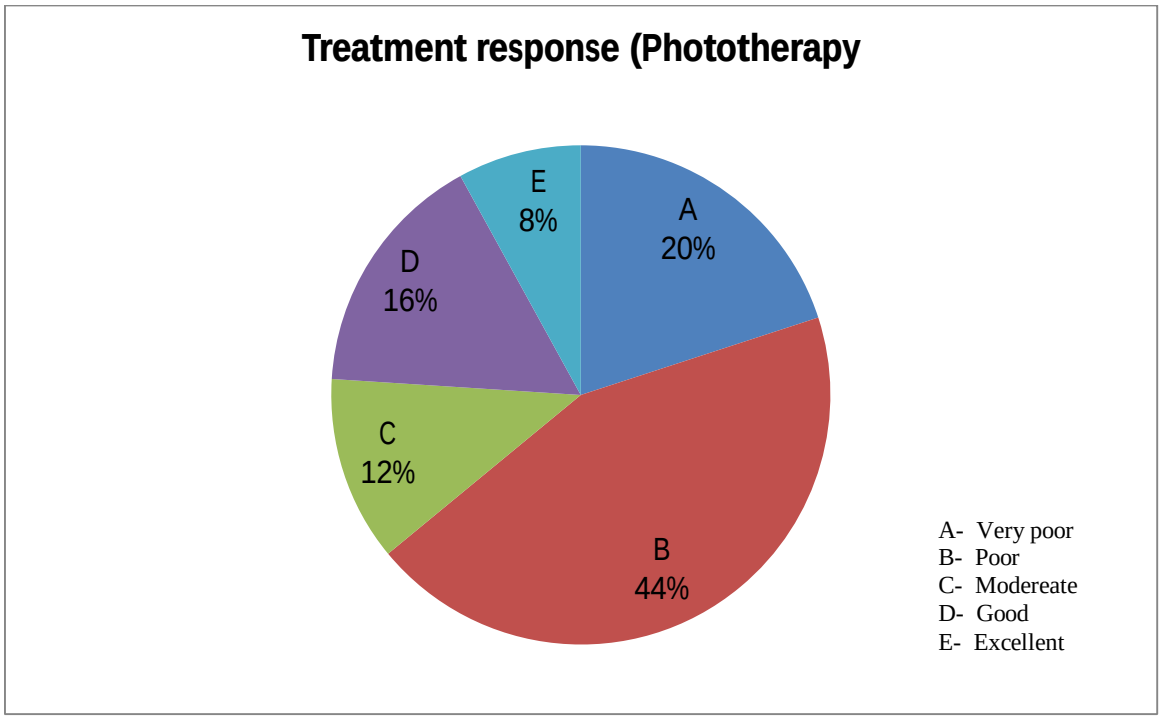
Treatment type	**Variable**		**Response**	
			Acceptable	**Unacceptable**
Topical phenol	Atopy	Present	0 (0%)	4 (100%)
		Absent	13 (61.9%)	8 (38.1%)
Chisquare (Fischer exact)- 5.15 p value – 0.039 (statistically significant)				

GROUP IV: RESULTS OBTAINED WITH PHOTOTHERAPY

Acceptable hair regrowth was noted in 9(36%) out of 25 patients. Numbers of patches range from 1 to 8 , with 8 patients (32%) had single patch and 13 patients (52%) had two patches, 1 patient(4%) had three patch, 2 patients(8%) had 4 patches and remaining 1 patients (4%)had 8 patches.

The size of patch ranges from 1x1 to 5x4 cm. Associated nail changes were found in 9 patients (36%). Atopy was found to be associated with 3 patients (12%). 8 patients (32%) had single patch and multiple patches were found in 17 patients (68%). 1patient (4%) were found to be diabetic and 1 patient (4%) were found to have systemic hypertension.

Among them excellent response was seen in two patients(8%), good response in four(16%) and moderate response in three patients(12%). 11 patients showed poor response(44%) and 5 patients showed very poor response(20%).

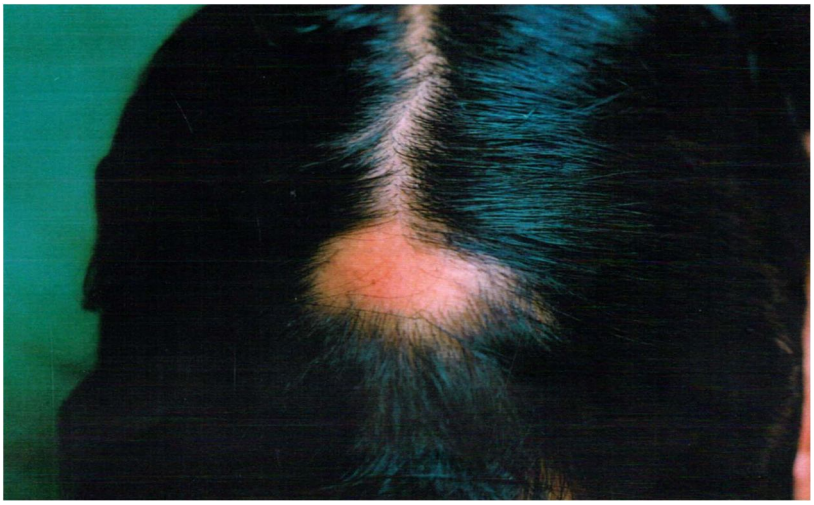


In patients showing excellent response, the hair regrowth started at 4weeks with gradual increase in thickness and density of hair. In patients with good response, regrowth started by 4weeks in two patients and 6 weeks in two patients. In patients with poor response, regrowth started by 6 to 8weeks but there was no improvement in density.

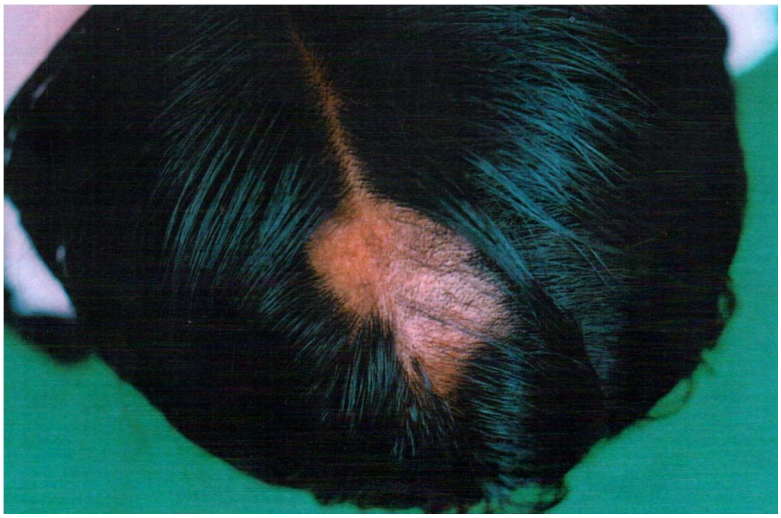
In patients with very poor response there was no regrowth of hair. None of the patients in this study group developed new patch during the study period. Adverse effects in the form of erythema and burning sensation developed in three patients during the first week of therapy. These effects were mild and tolerable and the patients wished to continue the therapy.

Clinical Images

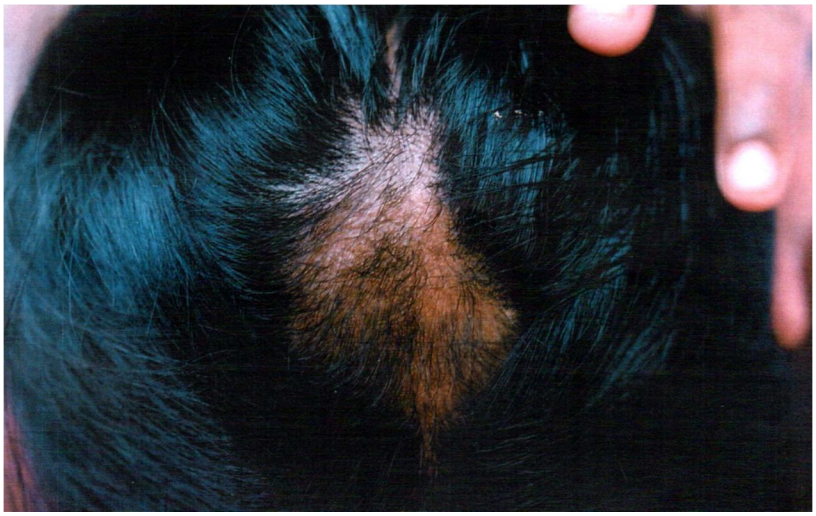
TREATMENT WITH INTRALESIONAL STEROIDS



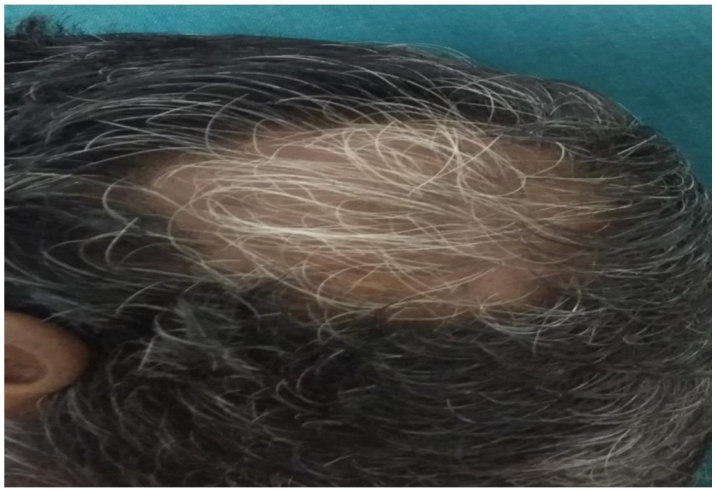
IN BETWEEN THERAPY



AT THE END OF THERAPY



TREATMENT WITH INTRALESIONAL STEROIDS. AT 8 WEEKS



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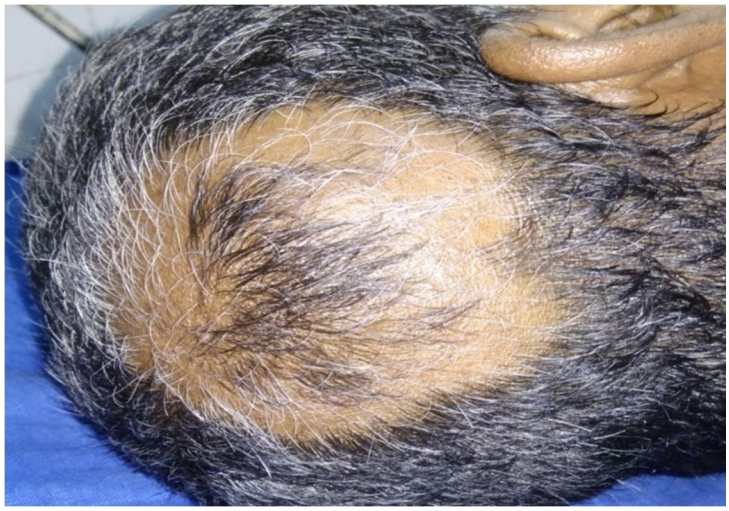
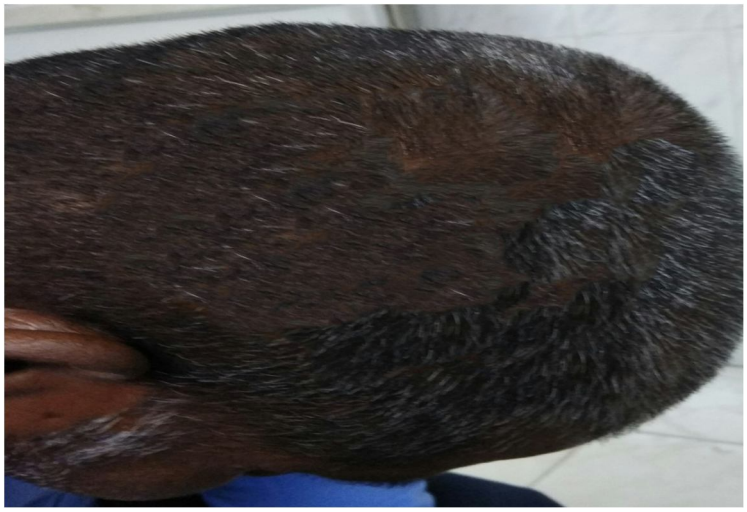
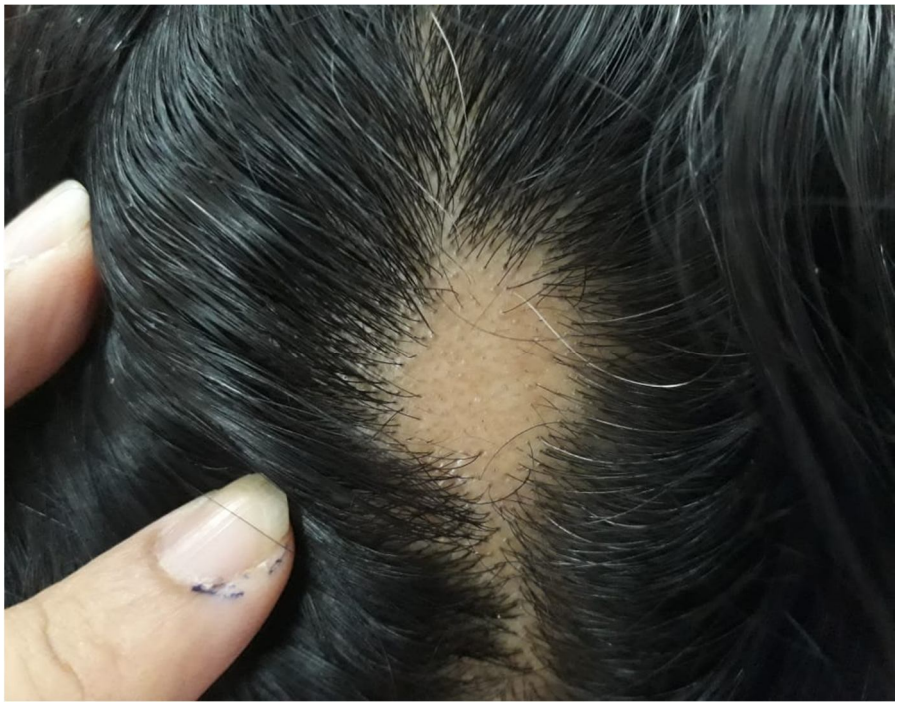


AT THE END OF THERAPY

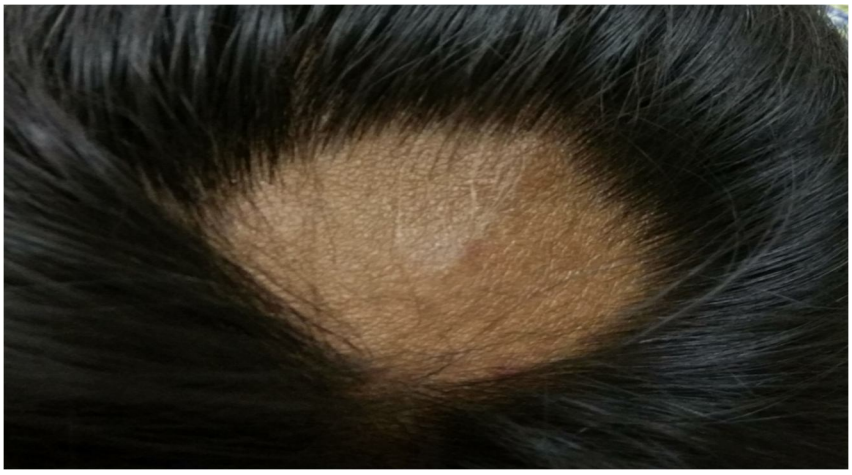


TREATMENT WITH CRYOTHERAPY
BEFORE THERAPY

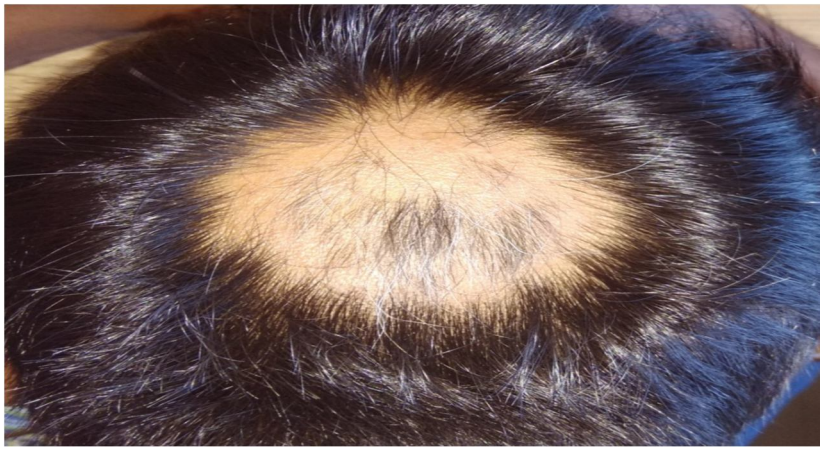


AFTER THERAPY

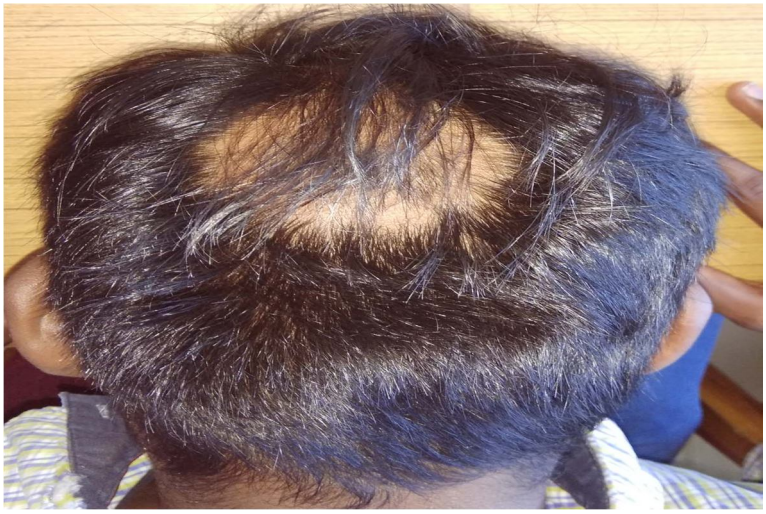
TREATMENT WITH PHENOL
BEEFORE THERAPY



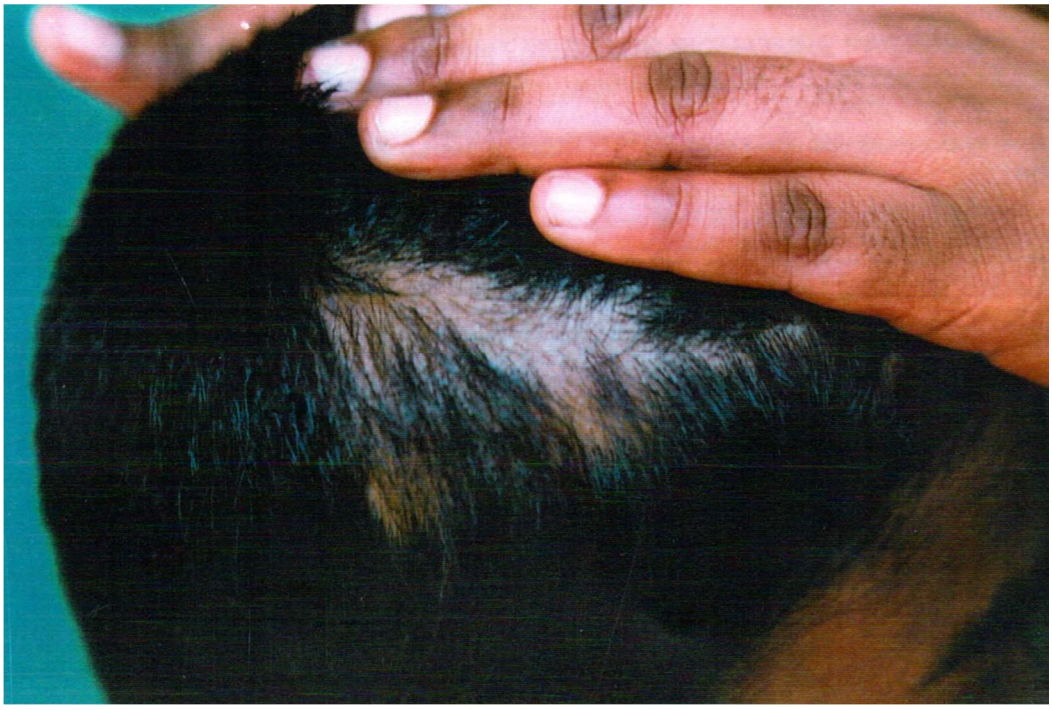
IN BETWEEN THE THERAPY



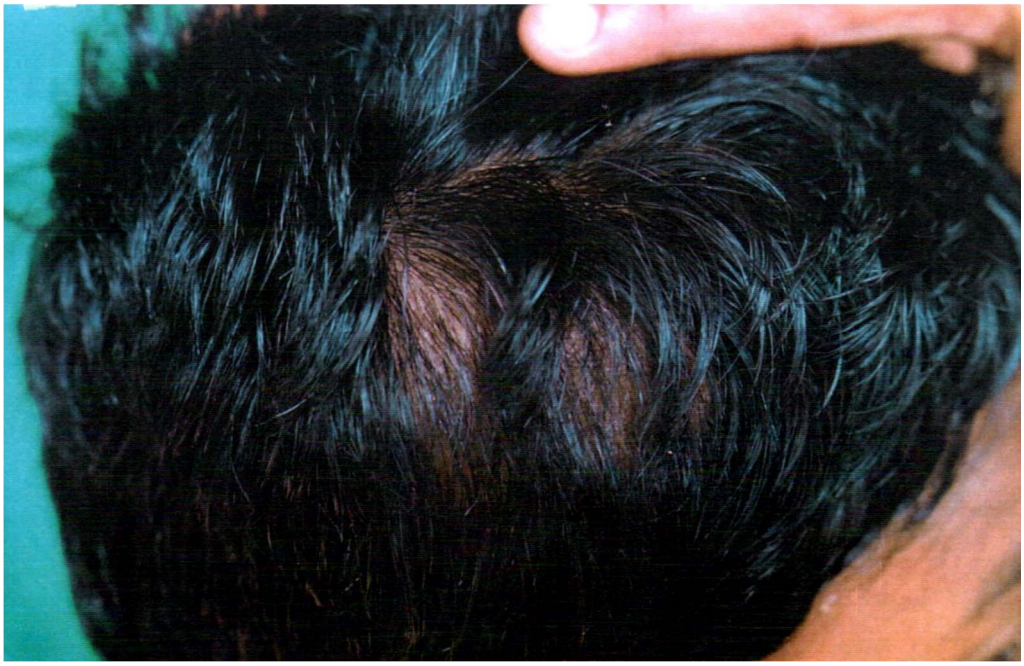
AT THE END OF TREATMENT

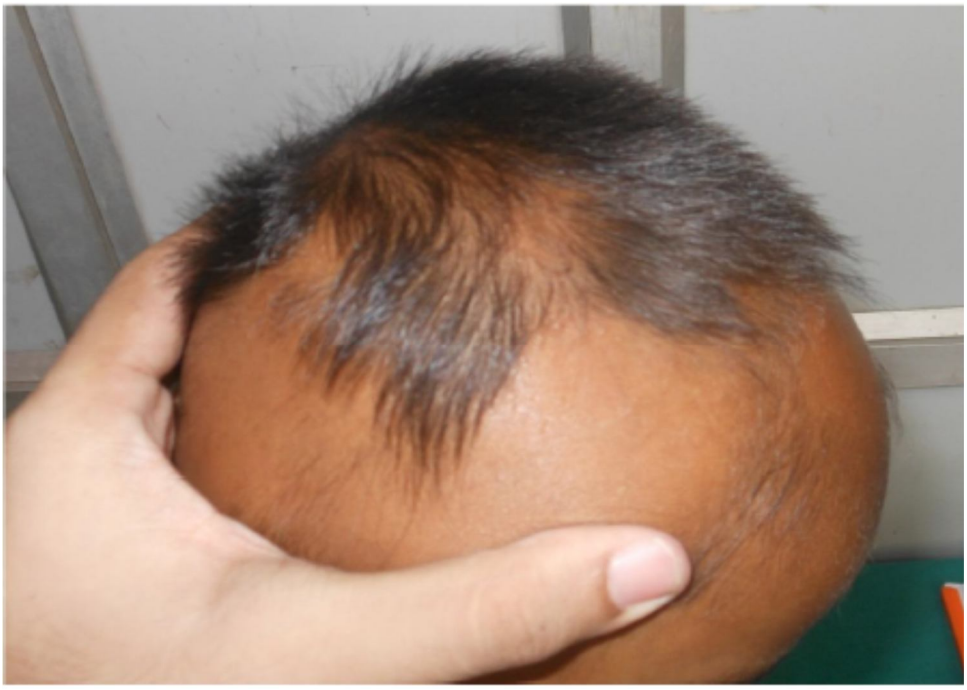


TREATMENT WITH PHOTOTHERAPY
BEFORE THERAPY

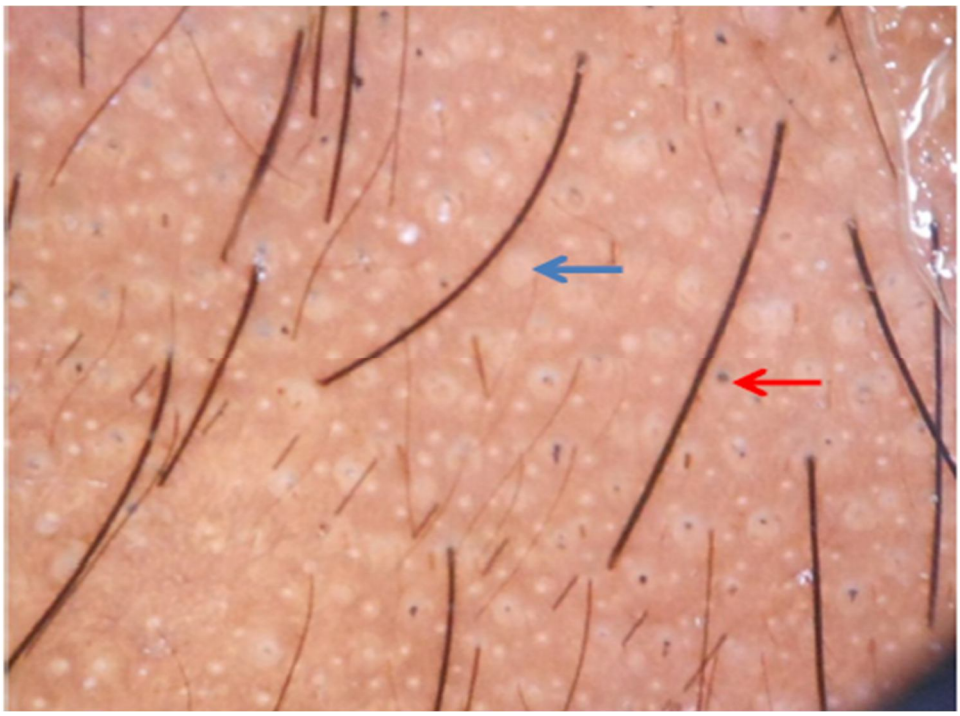


AFTER THERAPY

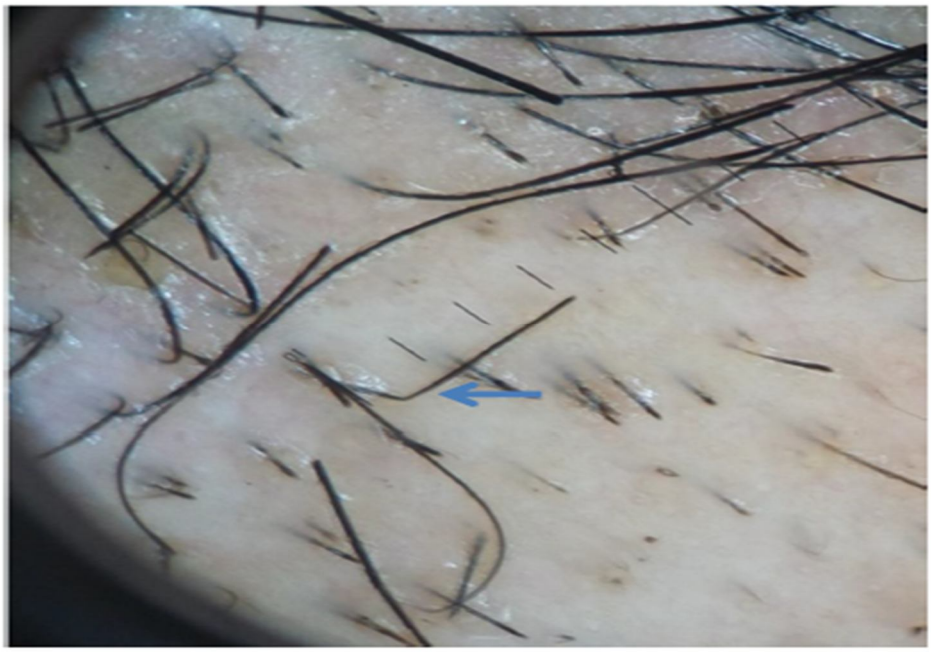




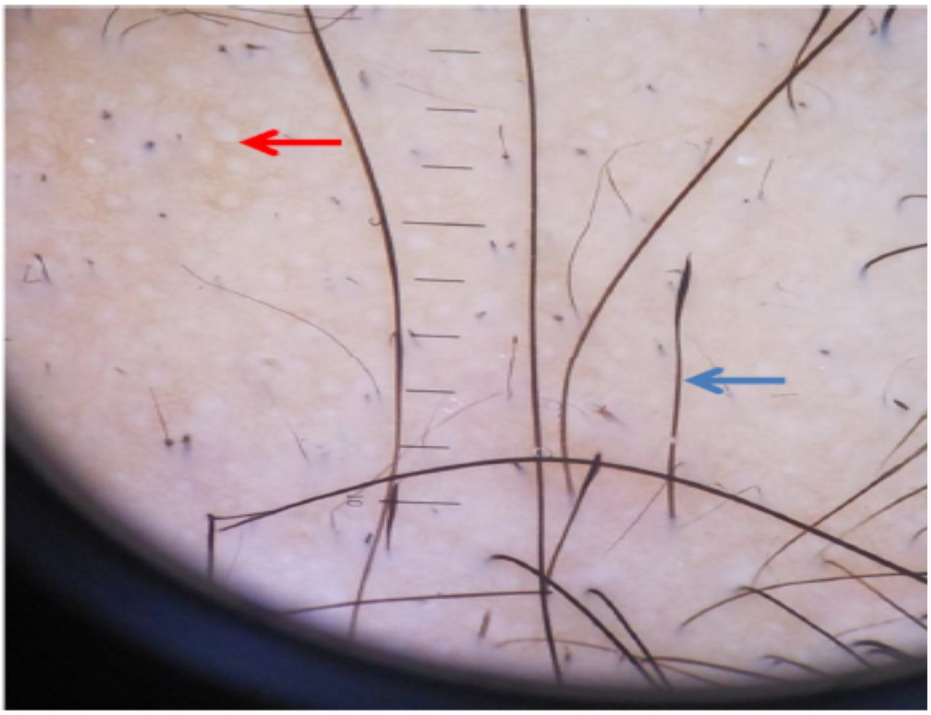
OPHIASIS TYPE



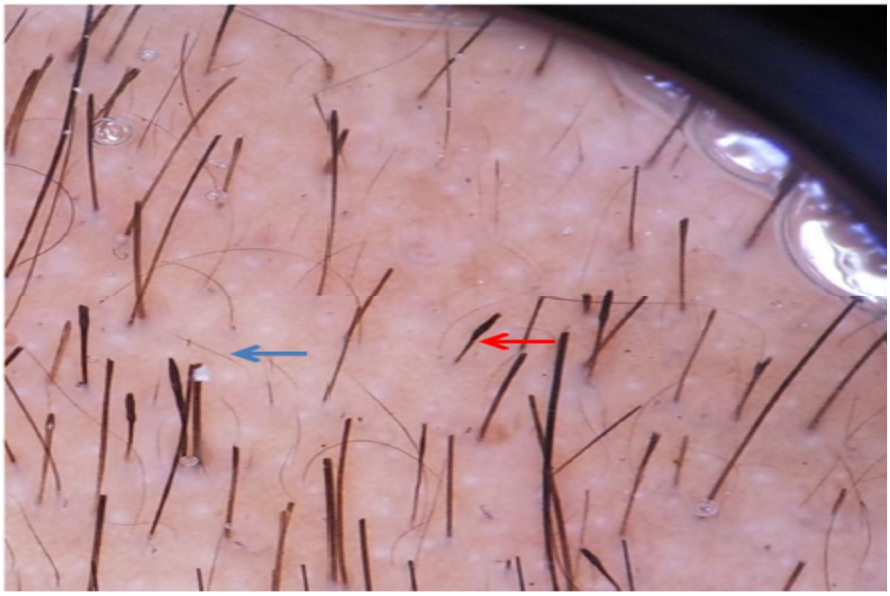
Black dots (red arrow) & yellow dots (blue arrow)



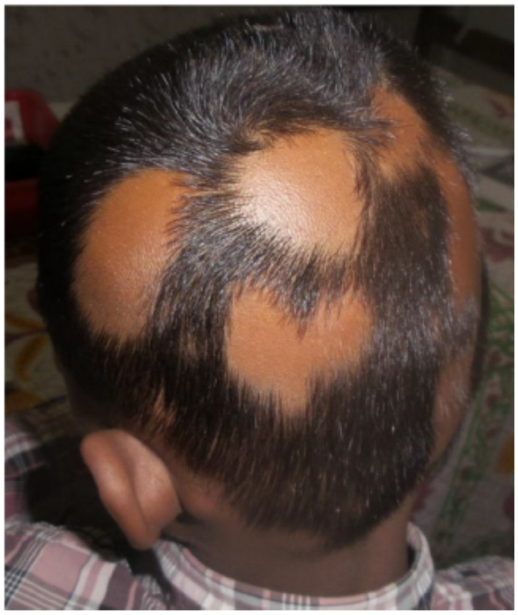
Dermoscopic coudability (blue arrow)



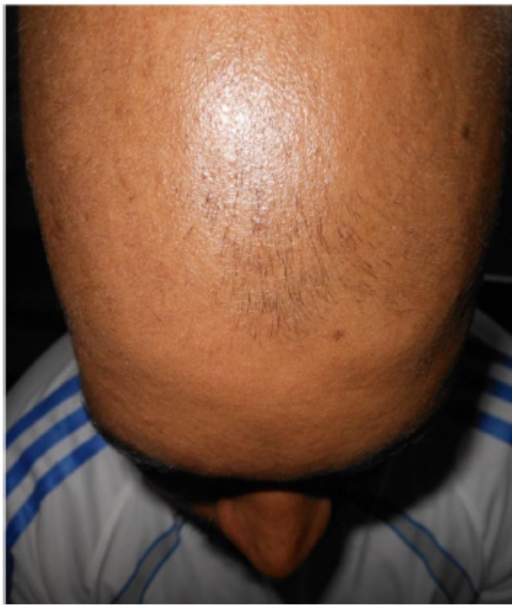
Exclamation mark hairs (blue arrow), yellow dots (red arrow) & black dots



Exclamation mark hair (red arrow) & vellus hair (blue arrow)



ALOPECIA AREATA – PATCHY TYPE

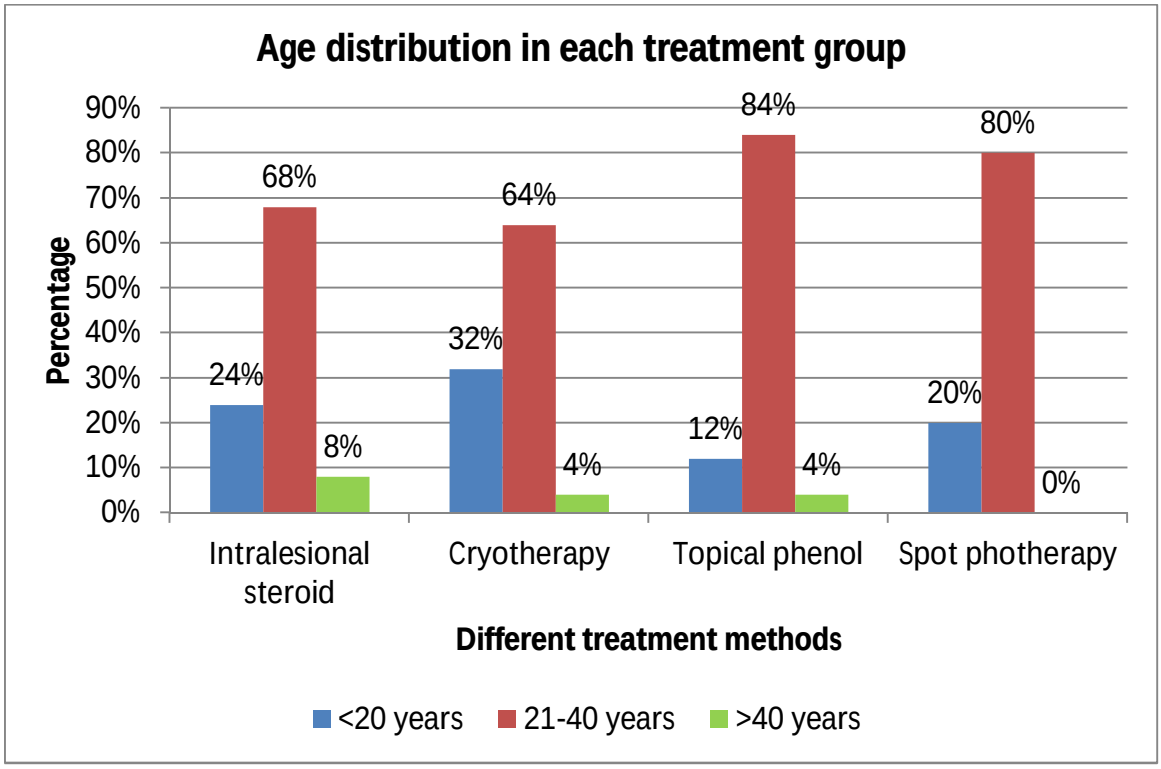


ALOPECIA SUBTOTALIS

Discussion

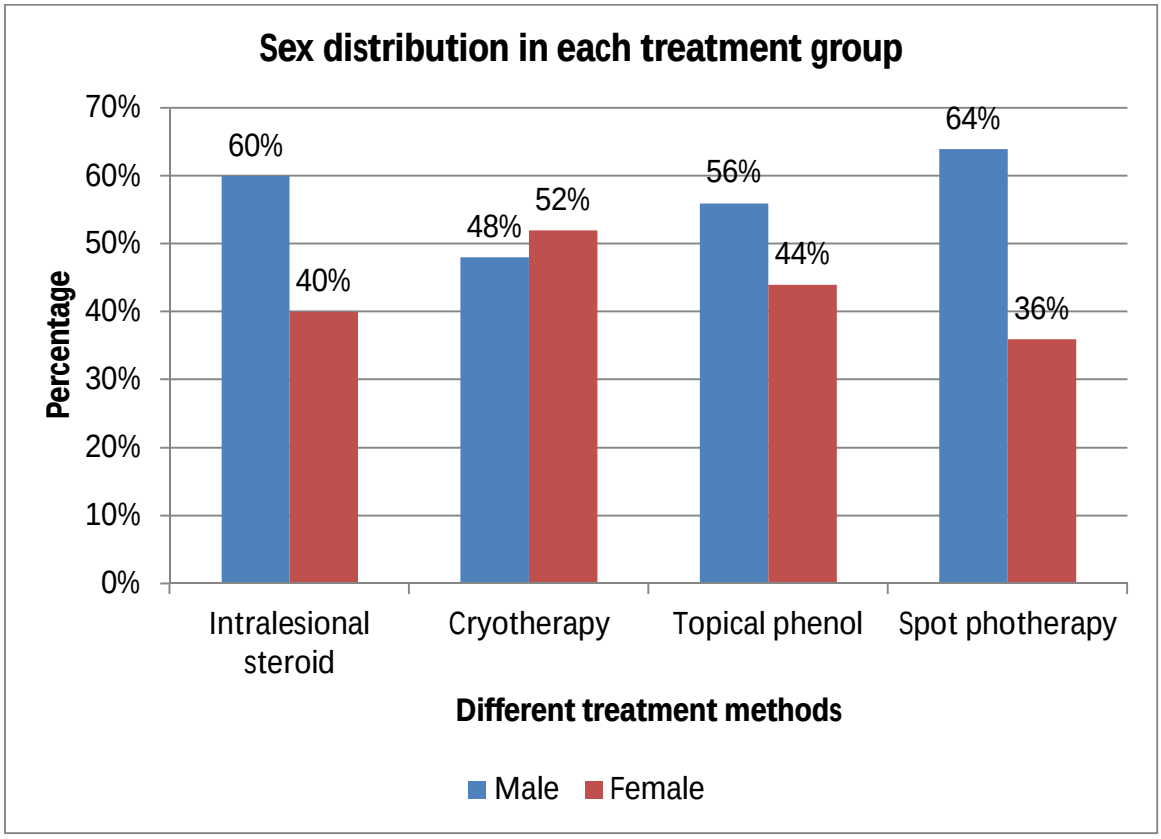
DISCUSSION

In this study of 100 patients with age ranging from 7 to 49 years, the maximum number of 74 patients was in the age group of 21-40 years , 22 patients were in age group of less than 20 years and the minimum number of four subjects was in 40-49 years age group.



Thus, a total of 90% occurred below the age of 40 years, which shows decreasing incidence with age. This observation was similar to another study which states that 85.5% of the Asian patients with AA have disease onset before 40 years of age[85].

Males outnumbered the females in this study; there were 57% males and 43% females. Some studies show a significant male preponderance in the adult age group, although others identify contrasting results[86].



Nail changes were evident in 34% of the total 100 patients in the form of leukonychia, pitting, longitudinal ridging, onycholysis and beau's lines.

Gandhi *et al*. observed pitting to be the most common nail finding in patients with alopecia areata, which is similar to our study[87].

GROUP I: PATIENTS TREATED WITH INTRALESIONAL STEROIDS

In this group of 25 patients, there were 15 male (60%) and 10 female (40%). The duration of the disease ranges from 2weeks to twelve months (mean – 4.90).

Acceptable regrowth of hair occurred in 88% (22patients), of which 12 patients showed excellent response and 7 patients showed good response & 3 patients showed moderate response. Poor response was seen in 2 patients and very poor response in one patient.

Amirnia et al[78] found the efficacy of ILS was 83.3%, which is comparable to our study and he has also found a statistically significant response in patients treated with ILS compared to cryotherapy (P= 0.05).

Chang, Kyung Hee[75] et al showed in his study that six out of ten patients showed considerable improvement in contrast to our study where 88% improvement occurred. Abell and Munro[55] in 1973 reported hair regrowth in 62% of the patients at 4-6 weeks with Triamcinolone acetonide (using the Porto-jet injector) compared with 7% in control subjects injected with isotonic saline. Another study by Mona et al, showed that intralesional steroid has better hair regrowth 74.3% as compared to topical betamethasone which showed efficacy of 46.9%[84]

100% of cases with single patch improved and 76.9% improvement occurred in patients with multiple patches, unlike in other studies where extensive AA is of little improvement[76] which shows number of patches having no influence in the response outcome.

One patient each with poor and very poor response and 2 patients with good response had personal history of atopy. Two patients with good and excellent response and one patient with poor response also had family history of atopy, so atopy doesn't alter the course of the disease

Two patients with excellent response had dental sepsis and one patient with good response had chronic gingivitis, which was treated.

One patient with good and moderate response developed new patches elsewhere. This showed that though treatment was initiated it did not alter the course of the disease progression.

Intralesional Triamcinolone acetonide is a safe and effective treatment for patients with extensive alopecia areata.

GROUP II: PATIENTS TREATED WITH CRYOTHERAPY

In patients treated with cryotherapy, there were 12 male (48%) and 13 female (52%). The duration of the disease ranges from 2weeks to eighteen months (mean – 6.34).

76% (19 out of 25) patients showed acceptable regrowth of hair. Kim et al. reported that superficial cryotherapy showed therapeutic efficacy in 24 of 36 AA patients (66.7%)[79].

Out of 19, 7 patients had excellent response and 7 patients showed good response & 5 patients showed moderate response.

Zawar et al[77] studied the efficacy of cryotherapy in recalcitrant alopeciaareata and reported sustained hair regrowth in 80% patients.

Lei et al in his study[65] showed that hair regrowth occurred in greater than 60% of affected areas in 70 of 72 patients, which is in contrast to our study wherein 76% of the patients showed improvement.

Regrowth of hair was first noticed between two to four weeks in eight patients which is comparable to earlier studies where regrowth was reported by two weeks[65]

One patient with excellent response had dental caries and one patient with good response had chronic pharyngitis. Hence in this group focal sepsis doesn't seem to influence the response outcome.

80.0% of the patients with single and multiple patches showed improvement which shows that there is no significance in the number of patches in the response outcome.

From the above it can be concluded that liquid nitrogen seems to be one of the cost effective therapy that could be tried in the treatment of alopecia areata.

GROUP III: RESULTS OBTAINED WITH TOPICAL PHENOL

There were14 male (56%) and 11 female (44%). The duration of the disease ranges from 2weeks to two years (mean – 7.34).

The mean age of the patients in this group was 29.88 years and the mean duration was 7.34 months.

Among the 25 patients treated with phenol, acceptable hair regrowth was found in 13(52%) patients, of which four had excellent response, six had good response and three patients had moderate response.

A study done by Mehta et al[80] found 47.06% response at the end of three months in patients treated with phenol, which is comparable to our study which showed 52% response rate. Another study by Ravi et al[83] showed 66.7% response

for 88% phenol. Ravi et al also found intralesional corticosteroids were found to be more effective than topical phenol, which is similar to our study. Savant et al in his study showed 72.5% good response and 27.5% poor response.

Poor response was noticed in seven patients and five patients showed very poor response.

88% phenol can be considered as a treatment option for stable alopecia areata due to its low cost, ease of application and easy availability.

GROUP IV: RESULTS OBTAINED WITH PHOTOTHERAPY

In this group of 25 patients, there were 16 males (64%) and 9 females (36%). The duration of the disease ranges from 2weeks to fifteen months (mean – 4.02).

Acceptable hair regrowth was noted in 9(36%) out of 25 patients. Among them excellent response was seen in two patients, good response in four and moderate response in three patients. 11 patients showed poor response and 5 patients showed very poor response.

Bayramgürler et al[82]. which showed an excellent response in 20% of patients.

Kaur et al[83] in his study showed 17.5% patients had more than 50%of hair regrowth which is lower than our study.

Larger sample size and long term follow up are needed to assess the long term outcome of this treatment modality.

Among the different modalities of treatment used in our study, intralesional steroids were found to be superior than other treatment modalities. Statistically significant difference in treatment response was found between patients treated with intralesional steroid and topical phenol(p=0.005), intralesional steroids and phototherapy (p=0.001) , cryotherapy and phototherapy (p= 0.004).

Variable		**Response**	
		Acceptable	**Unacceptable**
Treatment	Intralesional steroid	22(88%)	3(12%)
	Topical phenol	13(52%)	12(48%)
Chisquare–7.71 pvalue–0.005 (statistically significant)			

Variable		**Response**	
		Acceptable	**Unacceptable**
Treatment	Intralesional steroid	22(88%)	3(12%)
	Spot phototherapy	9(36%)	16(64%)
Chisquare–14.34 pvalue–0.0001(statistically significant)			

Variable		Response	
		Acceptable	**Unacceptable**
Treatment	Cryotherapy	19(76%)	6(24%)
	Spot phototherapy	9(36%)	16(64%)
Chisquare–8.11 pvalue–0.004(statistically significant)			

Conclusion

CONCLUSION

1. Cosmetically acceptable regrowth of hair was seen in 88% of patients treated with intralesional steroids, 76% of patients with cryotherapy, 52 % in patients treated with phenol and 36%of patients treated with phototherapy with NBUVB.
2. Excellent response occurred in 48%(12patients) treated with ILS, 28% (7patients) with cryotherapy,16%(4patients) with phenol , 8%(2 patients) with NBUVB
3. Statistically significant improvement occurred in patients treated with intralesional steroids and topical phenol (p=0.005), intralesional steroids and phototherapy (p=0.001) , cryotherapy and phototherapy (p= 0.004).
4. Atopy in the form of personal and family history was the only significant association found in the present study group (30% patients)
5. Nail changes were seen in 34% of patients, with pitting as the most common finding.
6. Prevalence of alopecia areata is slightly higher in males compared to females. Higher incidence was found between 21 – 40 years of age
7. Statistically significant association was found between the association of atopy and treatment response (p= 0.002)

8. Statistically significant association was found between the association of nail changes and treatment response (p= 0.001)
9. The number of patches (single/ multiple) among different study groups doesn't influence the response outcome (P=0.26).
10. All the four treatment modalities produced only transient adverse effects which did not interfere with the patient compliance.
11. All the four modalities listed above may be considered in the therapy of alopecia areata as the adverse effect noticed were transient. Role of NBUVB in treatment of alopecia areata needs further evaluation in large group of patients

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Annexures

ABBREVIATIONS

AD	-	ATOPIC DERMATITIS
AR	-	ALLERGIC RHINITIS
BA	-	BRONCHIAL ASTHMA
BL	-	BEUS LINE
CU	-	CHRONIC URTICARIA
DC	-	DENTAL CARIES
F	-	FRONTAL
G	-	GINGIVITIS
L	-	LEUCONYCHIA
LG	-	LINEAR GROOVES
LSC	-	LICHEN SIMPLEX CHRONICUS
LP	-	LICHEN PLANUS
MNG	-	MULTINODULAR GOITRE
M	-	MELANONYCHIA
O	-	OCCIPITAL
OL	-	ONYCHOLYSIS
P	-	PARIETAL
PA	-	PITYRIASIS ALBA
Ph	-	PHARYNGITIS
SEB DERM	-	SEBORRHOEIC DERMATITIS
T	-	TEMPORAL
T.CRURIS	-	TINEA CRURIS
TV	-	TINEA VERSICOLOR
VV	-	VITILIGO VULGARIS

MASTER CHART FOR PATIENTS ON INTRALESIONAL STEROID THERAPY

S/NO	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25
AGE	23	34	16	28	37	40	34	25	14	17	19	32	29	41	31	36	24	21	31	19	20	23	27	39	49
SEX	M	M	F	F	F	M	M	F	M	M	M	M	F	M	F	F	M	F	F	M	M	M	M	F	M
DURATION (MONTHS)	9	10	9	8	2	3	7	3	2	3	2	12	1	3	1	0.5	1	1	2	7	12	9	8	5	2
SYMPTOMS		-	-	ITCHING	-	-	-	-	-	-	-	MILD ITCHING	-	-	-	-	-	-	-	-	-	-	-	-	-
TREATMENT HISTORY																									
TOPICAL	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-
SYSTEMIC	v	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-
FAMILY HISTORY																									
DM	+	-	-	-	+	-	-	-	-	+	-	-	+	-	-	+	-	-	-	-	-	-	-	-	+
HT	-	-	+	-	-	-	-	-	-	-	-	+	-	-	-	-	-	+	-	-	-	-	-	-	-
ATOPY	+	-	-	-	-	-	-	-	+	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-
PERSONAL HISTORY																									
DM	-	-	-	-	-	+	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	+	+
HT	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+
ATOPY	AR	-	-	-	-	-	-	CU	-	-	-	-	CU	-	-	-	-	AR	-	-	-	-	-	BA	-
AUTOIMMUNE	-	MNG	-	-	-	-	HYPOTHYROID	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
OTHERS	-	-	-	-	-	T.CRURIS	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
PAST HISTORY OF AA	+	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-
GEN. EXM	N	G	N	N	DC	N	N	N	N	N	N	N	N	N	N	N	DC	N	N	N	N	N	N	N	N
SYS. EXM	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
DERM EXM																									
NO OF PATCHES	2	1	2	3	1	1	2	2	1	1	1	3	2	3	1	1	1	2	1	3	3	2	2	1	1
SITE	O	P	O,T	O.P	P	O	O,P	O	P	O	O	O(2),P	P	O,T(2)	P	O	O	P(2)	O	O, P(2)	O(2),T	O	P	P	O
SIZE(CM)	3X2,, 2X1	3X3	3X2,3X3	3X2, 2X1,1X1	4X3	5X3	4X2,3X3	3X3,4X3	4X4	4X3	3X2	2X1,2X1,3X2	3X2,3X3	3X2 -1X1	3X2	3X3	4X3	3X2,2X2	3X2	4X3,1X1	3X3- 2X1	2X2,2X1	3X2,2X2	3X2	2X2
NAILS	PITS	-	-	-	LG	-	-	PITS	-	-	-	PITS	L	-	-	-	-	PITS	-	-	L	-	-	-	-
RESPONSE AT12 WEEKS	A	D	E	E	E	D	E	B	D	E	E	C	D	C	E	E	E	D	E	D	B	E	D	C	E

MASTER CHART FOR PATIENTS ON CRYOTHERAPY

S/NO	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25
AGE	30	34	37	26	23	26	28	36	33	42	28	20	31	27	16	13	7	14	19	15	17	30	40	34	22
SEX	F	F	M	F	M	M	F	M	M	F	F	F	M	F	M	F	F	M	M	M	F	F	M	F	M
DURATION(MONTHS)	0.5	2	6	3	2WEEKS	3	9	12	18	8	9	12	5	3	8	4	12	15	1	2	1	0.5	12	10	2
SYMPTOMS	-	-	-	-	-	-	-	MILD ITCHING	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
TREATMENT HISTORY																									
TOPICAL	-	-	+	-	-	-	-	-	-	-	+	-	-	-	-	-	-	+	-	-	-	-	-	-	-
SYSTEMIC	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-
FAMILY HISTORY																									
DM	-	-	+	-	-	-	+	-	+	+	-	-	+	-	-	-	-	-	-	-	-	+	+	-	-
HT	-	+	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-
ATOPY	-	-	-	+	-	-	-	-	-	-	-	-	-	-	+	-	+	-	-	-	-	-	-	-	-
PERSONAL HISTORY																									
DM	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-
HT	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
ATOPY	AR	-	BA	-	-	-	-	BA	-	CU	-	-	-	-	-	-	PA	-	-	-	-	-	CU	AR	-
AUTOIMMUNE	-	-	-	-	-	-	-	-	MNG	-	-	-	-	-	HYPOTHYROID	-	-	-	-	-	-	-	-		
OTHERS	-	-	-	-	-	-	-	-	-	INTERTRIGO	-	-	-	-	-	-	-	-	-	-	-	-	LSC	-	-
PAST HISTORY OF AA	-	-	+	-	-	-	-	-	-	-	+	-	-	-	-	-	-	+	-	-	-	-	-	+	-
GEN. EXM	N	DC	N	N	N	N	N	N	N	N	N	PH	N	N	N	N	N	N	N	N	N	N	N	N	N
SYS. EXM	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
DERM EXM																									
NO OF PATCHES	1	1	2	1	1	1	2	2	3	1	3	1	1	1	2	1	1	3	2	1	2	1	3	4	2
SITE	O	P	O,P	O	P	O	O,T	P,T	P(2),T	O	O,P,T	P	F	P	P,F	P	T	O,T(3)	O	P	T,P	P	O, P(2)	O(2), P(2)	O
SIZE(CM)	3X2	2X2	3X2,3X3	4X3	3X2	2X2	4X3,3X2	5X4.2X1	4X4- 1X1	3X2	3X2- 1X1	2X2	3X2	3X2	4X3,3X2	2X2	3X2	4X3-1X1	3X2,1X1	2X2	3X3,3X2	5X4	4X4-2X2	5X4-2X1	3X2-,2X2
NAILS	L	-	PITS	-	-	-	L	-	LG	-	PITS	-	-	-	-	-	PITS, T	L	-	-	-	-	-	PITS	BL
RESPONSE AT 12 WEEKS	C	E	B	C	E	D	D	A	B	C	D	D	E	E	C	E	A	B	D	E	E	D	C	A	D

MASTER CHART FOR PATIENTS ON TOPICAL PHENOL

S/NO	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25
AGE	19	23	26	33	36	31	29	41	38	37	19	27	28	40	36	34	23	28	29	19	31	37	29	27	27
SEX	M	M	F	M	F	M	F	M	M	M	F	F	M	M	M	F	F	M	F	M	F	M	M	F	F
DURATION(MONTHS)	3	1	0.5	3	12	18	9	4	24	5	6	8	12	15	10	2	6	7	1	12	9	2	3	5	6
SYMPTOMS	-	-	-	-	-	-	ITCH	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
TREATMENT HISTORY																									
TOPICAL	-	-	-	-	-	+	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
SYSTEMIC	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
FAMILY HISTORY																									
DM	-	-	-	+	-	-	+	-	-	-	-	-	-	-	+	-	-	-	-	-	-	+	-	-	+
HT	-	-	-	-	-	-	-	-	+	+	-	-	-	-	+	-	-	-	-	-	-	+	-	-	-
ATOPY	-	+	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-
PERSONAL HISTORY																									
DM	-	-	-	-	-	-	-	+	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-
HT	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
ATOPY	AR	-	-	-	CU	-	-	-	-	-	-	-	-	-	CU	-	-	-	-	AD	-	-	-	-	-
AUTOIMMUNE	-	-	-	-	-	-	-	-	VV	-	-	-	-	-	-	-	LP	-	-	-	-	-	-	-	-
OTHERS	N	N	N	N	N	N	N	N	N	N	VERRUCA	N	N	N	N	N	SEBDERM	N	N	N	N	N	N	N	N
PAST HISTORY OF AA	-	-	-	+	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-
GEN. EXM	N	N	N	N	N	DC	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
SYS. EXM	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
DERM EXM																									
NO OF PATCHES	1	1	1	2	1	3	2	1	3	4	3	2	2	3	2	1	2	2	1	2	2	2	1	1	1
SITE	O	P	P	O,P	P	O(2), P	P,T	O	O(2),T	O(2), P(2)	O, P(2)	O,P	O	O,P(2)	O	P	T	O,T	O	P	O	O,P	P	O	O
SIZE(CM)	4X3	3X3	4X2	3X2-2X1	3X2	4X3-2X1	3X2,2X2	3X2	4X3-2X1	4X3-2X2	4X3-1X1	3X2,2X2	4X3,3X2	4X3-2X1	3X3,2X1	3X2	3X2,2X1	3X2,2X1	3X2	4X3,3X2	4X4,3X2	3X3,2X2	3X2	3X2	3X3
NAILS	-	-	OL, PITS	-	-	PITS	-	-	LG	-	-	-	-	PITS	-	-	-	-	PITS,T	PITS	PITS	LG	-	-	L
RESPONSE AT 12 WEEKS	B	C	E	D	A	B	C	D	A	A	B	D	B	A	A	D	D	C	E	B	B	D	E	E	B

MASTER CHART FOR PATIENTS ON SPOT PHOTOTHERAPY

S/NO	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25
AGE	27	29	21	22	28	36	28	21	23	19	16	32	30	27	23	29	28	31	34	20	27	37	25	19	12
SEX	M	F	M	F	M	M	M	F	M	M	F	M	M	F	F	M	M	M	F	M	M	F	M	M	F
DURATION(MONTHS)	1	1	2	0.5	6	2	4	2.5	3	1	1	1.5	2	12	7	9	15	5	3	1	5	3	5	3	5
SYMPTOMS	-	-	-	-	-	-	-	-	-	-	-	-	-	ITCHING	-	-	-	-	-	-	-	-	-	-	-
TREATMENT HISTORY																									
TOPICAL	+	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
SYSTEMIC	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
FAMILY HISTORY																									
DM	-	-	-	-	+	-	-	-	-	-	-	-	+	-	-	+	-	-	+	-	-	-	-	-	-
HT	-	-	-	-	-	+	-	-	-	-	-	+	+	-	-	-	-	-	-	-	-	+	-	-	+
ATOPY	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-
PERSONAL HISTORY																									
DM	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-
HT	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
ATOPY	CU	-	-	-	-	-	-	-	-	-	-	CU	-	-	-	-	-	-	-	AR	-	-	-	-	-
AUTOIMMUNE	v	MNG	-	-	-	-	-	-	-	-	-	-	VV	-	-	-	-	-	-	-	-	-	-	-	-
OTHERS	-	-	T.CORPORIS	-	-	-	-	-	-	-	-	-	-	-	-	PSORIASIS	-	-	-	-	-	-	-	-	-
PAST HISTORY OF AA																									
GEN. EXM	N	N	N	N	N	N	N	N	DC	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
SYS. EXM	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
DERM EXM																									
NO OF PATCHES	4	2	2	1	2	2	8	2	1	1	2	1	3	2	2	2	4	1	1	1	2	1	2	2	2
SITE	O(2),P,F	O,P	O	P	O,P	O,T	O(3), P(2),T(2),F	O	P	T	O	P	O(2),P	O	O,P	P,T	O,P(2),T	O	P	O	P,T	O	T,P	O,P	O,P
SIZE(CM)	4X4 -2X2	3X2,2X2	4X3,3X2	3X3	X3,3X2	3X2,2X2	3X3-1X1	3X3,3X2	4X3	3X2	3X2,2X1	3X2	3X2-2X1	3X3,3X2	2X2,2X1	2X2,2X1	4X3-2X1	3X2	3X2	3X3	5X4-2X1	3X2	2X2,3X2	3X2,2X1	2X2,4X2
NAILS	LG		L	OL			PITS, L						PITS		LG		PITS				M				PITS
RESPONSE AT 12 WEEKS	B	C	B	E	B	C	A	B	D	B	B	A	B	B	C	A	B	D	A	B	B	D	A	D	E

TABLE 1- INTRALESIONAL STEROID

PROFILE OF THE PATIENTS									RESPONSE						
S/NO	AGE	SEX	NO OF PATCHES	DURATION (MONTHS)	ATOPY - FAMILY	ATOPY- PERSONAL	COUDABLITY SIGN	EXCLAMATION HAIR	2WKS	4 WKS	6WKS	8 WKS	10WKS	12WKS	ADVERSE EFFECTS
1	23	M	2	9	+	AR	+	+	A	A	A	A	A	A	-
2	34	M	1	10	-	-	+	+	A	B	B	C	D	D	-
3	16	F	2	9	-	-	-	-	A	B	C	C	D	E	-
4	28	F	3	8	-	-	+	+	A	B	B	C	C	E	-
5	37	F	1	2	-	-	+	+	A	A	B	C	D	E	-
6	40	M	1	3	-	-	-	+	A	A	B	C	C	D	-
7	34	M	2	7	-	-	+	+	A	A	B	C	D	E	-
8	25	F	2	3	-	CU	-	+	A	A	A	A	B	B	-
9	14	M	1	2	+	-	+	-	A	A	B	B	C	D	-
10	17	M	1	3	-	-	+	+	A	B	C	C	D	E	TRANSIENT ATROPHY
11	19	M	1	2	-	-	-	+	A	B	C	C	D	E	-
12	32	M	3	12	-	-	+	+	A	A	B	B	C	C	PAIN
13	29	F	2	1	-	CU	-	+	A	A	B	C	C	D	-
14	41	M	3	3	-	-	+	+	A	A	B	B	C	C	-
15	31	F	1	1	-	-	-	-	A	B	C	C	D	E	-
16	36	F	1	0.5	+	-	+	+	A	B	C	D	D	E	-
17	24	M	1	1	-	-	+	+	A	A	B	C	C	E	-
18	21	F	2	1	-	AR	+	+	A	A	B	C	C	D	-
19	31	F	1	2	-	-	-	+	A	A	B	C	D	E	-
20	19	M	3	7	-	-	+	+	A	B	B	C	C	D	MINIMAL PAIN
21	20	M	3	12	-	-	-	-	A	A	A	A	B	B	-
22	23	M	2	9	-	-	-	-	A	A	B	C	D	E	-
23	27	M	2	8	-	-	+	+	A	A	B	C	C	D	-
24	39	F	1	5	-	BA	-	-	A	A	B	B	C	C	-
25	49	M	1	2	-	-	+	+	A	B	B	C	D	E	-

TABLE 2 - CRYOTHERAPY

PROFILE OF THE PATIENTS									RESPONSE						
S/NO	AGE	SEX	NO OF PATCHES	DURATION(MONTHS)	ATOPY-FAMILY	ATOPY - PERSONAL	COUDABLITY SIGN	EXCLAMATION HAIR	2WKS	4WKS	6WKS	8WKS	10WKS	12WKS	ADVERSE REACTIONS
1	30	F	1	0.5	-	AR	-	+	A	A	B	B	C	C	-
2	34	F	1	2	-	-	+	-	A	B	C	C	D	E	-
3	37	M	2	6	-	BA	+	+	A	A	A	B	B	B	EDEMA
4	26	F	1	3	+	-	+	+	A	A	B	B	B	C	-
5	23	M	1	2WEEKS	-	-	-	+	A	A	B	B	C	E	-
6	26	M	1	3	-	-	+	-	A	A	B	B	C	D	-
7	28	F	2	9	-	-	+	+	A	B	B	C	C	D	-
8	36	M	2	12	-	BA	+	+	A	A	A	A	A	A	-
9	33	M	3	18	-	-	+	+	A	A	A	B	B	B	ERYTHEMA
10	42	F	1	8	-	CU	-	+	A	A	B	B	C	C	-
11	28	F	3	9	-	-	+	+	A	B	B	C	D	D	-
12	20	F	1	12	-	-	-	-	A	B	B	C	C	D	-
13	31	M	1	5	-	-	+	+	A	B	C	C	D	E	-
14	27	F	1	3	-	-	+	+	A	A	B	B	C	E	-
15	16	M	2	8	+	-	+	+	A	A	B	B	C	C	-
16	13	F	1	4	-	-	-	+	A	B	C	C	D	E	-
17	7	F	1	12	+	PA	+	+	A	A	A	A	A	A	-
18	14	M	3	15	-	-	-	-	A	A	A	A	B	B	EDEMA
19	19	M	2	1	-	-	+	+	A	A	B	C	C	D	-
20	15	M	1	2	-	-	+	+	A	A	B	C	D	E	-
21	17	F	2	1	-	-	-	+	A	B	B	C	C	E	-
22	30	F	1	0.5	-	-	+	+	A	A	B	C	C	D	-
23	40	M	3	12	-	CU	+	+	A	A	B	B	C	C	-
24	34	F	4	10	-	AR	-	+	A	A	A	A	A	A	ERYTHEMA
25	22	M	2	2	-	-	+	+	A	B	B	C	C	D	-

TABLE 3 PHENOL															
PROFILE OF THE PATIENTS										RESPONSE					
S/NO	AGE	SEX	NO OF PATCHES	DURATION (MONTHS)	ATOPY - FAMILY	ATOPY-- PERSONAL	COUDABLITY SIGN	EXCLAMATION HAIR	2WKS	4 WKS	6WKS	8WKS	10WKS	12WKS	DVERE EFFECTS
1	19	M	1	3	-	AR	+	+	A	A	A	B	B	B	-
2	23	M	1	1	+	-	-	+	A	A	B	B	C	C	-
3	26	F	1	0.5	-	-	+	+	A	B	B	C	D	E	-
4	33	M	2	3	-	-	+	+	A	B	B	C	C	D	-
5	36	F	1	12	+	CU	-	-	A	A	A	A	A	A	-
6	31	M	3	18	-	-	+	+	A	A	A	B	B	B	HYPOPIGMENTATION
7	29	F	2	9	-	-	+	+	A	A	B	B	C	C	-
8	41	M	1	4	-	-	-	+	A	A	B	B	C	D	-
9	38	M	3	24	-	-	-	-	A	A	A	A	A	A	HYPOPIGMENTATION
10	37	M	4	5	-	-	+	+	A	A	A	A	A	A	ERYTHEMA
11	19	F	3	6	-	-	+	+	A	A	A	A	B	B	-
12	27	F	2	8	-	-	+	+	A	B	B	C	C	D	-
13	28	M	2	12	-	-	-	+	A	A	A	A	B	B	-
14	40	M	3	15	-	-	+	-	A	A	A	A	A	A	MILD ATROPHY
15	36	M	2	10	-	CU	+	+	A	A	A	A	A	A	-
16	34	F	1	2	-	-	+	+	A	A	B	C	C	D	-
17	23	F	2	6	-	-	+	+	A	B	B	C	C	D	-
18	28	M	2	7	-	-	+	+	A	A	B	B	C	C	-
19	29	F	1	1	+	-	-	+	A	A	B	C	D	E	-
20	19	M	2	12	-	AD	+	-	A	A	A	A	B	B	HYPOPIGMENTATION
21	31	F	2	9	-	-	+	+	A	A	A	B	B	B	-
22	37	M	2	2	-	-	+	+	A	A	B	C	C	D	-
23	29	M	1	3	-	-	+	-	A	A	B	C	C	E	-
24	27	F	1	5	-	-	-	+	A	B	C	C	D	E	HYPERPIGMENTATION
25	27	F	1	6	-	-	+	+	A	A	A	A	B	B	-

TABLE 4 PHOTOTHERAPY

PROFILE OF THE PATIENTS									RESPONSE						
S/NO	AGE	SEX	NO OF PATCHES	DURATION(MONTHS)	ATOPY - FAMILY	ATOPY - PERSONAL	COUDABLITY SIGN	EXCLAMATION HAIR	2WKS	4WKS	6WKS	8WKS	10WKS	12WKS	ADVERSE EFFECTS
1	27	M	4	1	-	CU	-	+	A	A	A	B	B	B	ERYTHEMA
2	29	F	2	1	+	-	+	-	A	A	B	B	C	C	
3	21	M	2	2	-	-	+	+	A	A	A	A	B	B	
4	22	F	1	0.5	-	-	+	+	A	A	B	C	D	E	
5	28	M	2	6	-	-	+	+	A	A	A	B	B	B	
6	36	M	2	2	-	-	-	-	A	B	B	C	C	C	
7	28	M	8	4	-	-	+	+	A	A	A	A	A	A	
8	21	F	2	2.5	-	-	+	+	A	A	A	B	B	B	ERYTHEMA
9	23	M	1	3	-	-	+	+	A	B	B	C	C	D	
10	19	M	1	1	-	-	-	+	A	A	A	B	B	B	
11	16	F	2	1	-	-	+	+	A	A	B	B	B	B	
12	32	M	1	1.5	-	CU	+	+	A	A	A	A	A	A	
13	30	M	3	2	-	-	+	+	A	A	B	B	B	B	BURNING SENSATION
14	27	F	2	12	-	-	+	+	A	B	B	B	B	B	
15	23	F	2	7	-	-	+	+	A	B	B	C	C	C	
16	29	M	2	9	-	-	+	+	A	A	A	A	A	A	
17	28	M	4	15	-	-	-	+	A	A	B	B	B	B	
18	31	M	1	5	-	-	+	-	A	A	B	B	C	D	
19	34	F	1	3	+	-	-	+	A	A	A	A	A	A	
20	20	M	1	1	-	AR	+	+	A	A	A	B	B	B	
21	27	M	2	5	-	-	+	+	A	A	B	B	B	B	
22	37	F	1	3	-	-	+	+	A	B	B	C	C	D	
23	25	M	2	5	-	-	+	+	A	A	A	A	A	A	
24	19	M	2	3	-	-	-	+	A	B	B	C	C	D	
25	12	F	2	5	-	-	+	-	A	A	B	C	D	E	

PROFORMA

"COMPARISION OF VARIOUS TREATMENT MODALITIES IN PATIENTS WITH ALOPECIA AREATA"

Name : Age / Sex :
OP / IP No. : Occupation
Address :

Chief Complaints:
Loss of hair – duration
Pruritis
Others (specify)

Past History:

- H/o Similar complaints in the past
- H/o Hypertension, Diabetes, Asthma, Epilepsy

Personal History:

- Type of Diet
- Childhood eczema
- Thyroid disease
- Septic foci (ENT, dental , others)

Family history:

- Diabetes mellitus
- Hypertension
- Atopy
- Other endocrine disorders
- Other autoimmune disorders

General Examination:

- Pallor, Icterus, Clubbing, Cyanosis, Pedal edema, Lymphadenopathy

Systemic Examination:

- CVS, RS, P/A, CNS
- Pulse
- BP

Dermatological Examination:

- Examination of patches :
  - Size
  - Shape
  - Number
  - Location
  - Presence of white hair
- Coudablity sign
- Exclamation mark hair
- Nail changes

  Pitting

  Others (specify)
- Associated changes

  Vitiligo

  Other dermatoses (Specify)
- Oral mucosa
- Palms & Soles
- Genitalia
- Eyes

Investigation:

- CBC
- LFT
- RFT
- VCTC
- VDRL
- Thyroid Function Test
- KOH smear

Therapy given:

- Duration of treatment
- Clinical response
- Adverse effects

INFORMATION SHEET

TITLE: **"COMPARISION OF VARIOUS TREATMENT MODALITIES IN PATIENTS WITH ALOPECIA AREATA"**

Investigators : **Dr. S. FATHIMA**

Name of the Participant Age : Sex :

Study Setting : Department of Dermatology
Madras Medical College & RGGGH,
Chennai – 3.

- You are invited to take part in this study. The information in this document is meant to help you decide whether or not to take part. Please feel free to ask if you have any queries or concerns.
- We are conducting a study on**"COMPARISION OF VARIOUS TREATMENT MODALITIES IN PATIENTS WITH ALOPECIA AREATA"**
- This study will not affect your treatment.
- Patients are randomly selected and divided into four groups.(A,B,C,D) of 25 patients in each.
- Group A – Treated with intralesional steroid (triamcinolone acetonide)
- Group B – Treated with cryotherapy (liquid nitrogen)
- Group C – Treated with topical phenol (88%)
- Group D – Treated with phototherapy(NBUVB)
- The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.
- Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled.
- The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of investigator Signature of participant

Dr. S. Fathima

Date: Date:

ஆய்வு தகவல் தாள்

ஆராய்ச்சியின் தலைப்பு : புழுவெட்டு நோயாளிகளிடையே பல்வேறு சிகிச்சை முறைகளை ஒப்பிட்டு பார்த்தல் குறித்த ஆய்வு.

ஆய்வாளர் : மரு. ச. பாத்திமா

பங்கேற்பாளர் : வயது : பாலினம் :

ஆராய்ச்சி மையம் : தோல்நோய் துறை,
இராஜீவ் காந்தி அரசு பொது மருத்துவமனை, சென்னை.

இந்த ஆய்வில் பங்கேற்பதற்காக தாங்கள் அழைக்கப்படுகிறீர்கள். இந்த ஆவணத்தில் உள்ள தகவல்கள் தாங்கள் இந்த ஆய்வில் பங்கேற்க முடிவு செய்துக் கொள்ள உதவும். இதில் ஏதேனும் சந்தேகம் இருந்தால் வெளிப்படையாக கேள்விகளைக் கேட்டு தெரிந்துக் கொள்ளலாம்.

நாங்கள் இராஜீவ் காந்தி அரசு பொது மருத்துவமனையில் புழுவெட்டு நோயாளிகளிடையே பல்வேறு சிகிச்சை முறைகளை ஒப்பிட்டு பார்த்தல் குறித்த ஆய்வை நடத்துகிறோம்.

அதற்கு உங்கள் பங்களிப்பு எங்களுக்கு பெரிதும் உதவக்கூடும்.

இந்த ஆய்வின் நோக்கம்:

இவ்வாராய்ச்சியில் தங்களிடையே அடிப்படை மற்றும் உங்களுடைய நோய் குறித்த விரிவான கேள்விகள் கேட்கப்படும். பின்னர் நீங்கள் மருத்துவப் பரிசோதனைக்கு உட்படுத்தப்படுவீர்கள். பின்பு தோல் சம்பந்தமான வெளிப்பாடுகள் குறித்து மருத்துவப் புகைப்படம் எடுக்கப்படும்.

அனைவரிடமும் இரத்தம் மாதிரி பெறப்பட்டு அது வழக்கமான இரத்தப் பரிசோதனைகளும் (CBC, LFT, RFT, VCTC, VDRL, TFT) மற்றும் தேவைப்படுகின்ற நோயாளிகளுக்கு KOH Smear பரிசோதனையும் செய்யப்படும். பிறகு நான்கு குழுக்களாக (ABCD) பிரிக்கப்பட்டு, **A**-பிரிவில் உள்ளவர்களுக்கு இன்ட்ராலீஷ்னல் ஸ்டீராய்டு ஊசி மருந்து, **B**-பிரிவில் உள்ளவர்களுக்கு திரவ நைட்ரஜன் க்ரையோதெரபி, **C**-பிரிவில் உள்ளவர்களுக்கு மேற்பூச்சு மீனால், **D**-பிரிவில் உள்ளவர்களுக்கு ஒளி கதிர் சிகிச்சை அளிக்கப்படும்.

தங்களது மருத்துவ சிகிச்சை குறித்த தகவல்கள் இரகசியமாக பாதுகாக்கப்படும். ஆய்வின் போதோ அல்லது முடிவுகளை வெளியிடும் போதோ தங்களது பெயரையோ, அடையாளங்களையோ வெளியிடமாட்டோம் என்பதை தெரிவித்துக் கொள்கிறோம்.

இந்த ஆய்வில் பங்கேற்பது உங்களுடைய விருப்பத்தின் பேரில் தான் இருக்கிறது. மேலும் நீங்கள் எந்நேரமும் இந்த ஆய்விலிருந்து பின்வாங்கலாம் என்பதையும் தெரிவித்துக் கொள்கிறோம். இந்த ஆய்வில் பங்கேற்காவிட்டாலும் நீங்கள் வழக்கமான சிகிச்சையை தொடர்ந்து பெறலாம்.

இந்த ஆய்வின் முடிவு தங்களுக்கு ஆய்வின் இறுதியிலோ அல்லது ஆய்வின் போதிலோ தெரியப்படுத்தப்படும்.

ஆய்வாளர் கையொப்பம்

பங்கேற்பாளர் / பாதுகாவலர்
கையொப்பம்

தேதி :

INFORMED CONSENT FORM

Title of the study :**"COMPARISION OF VARIOUS TREATMENT MODALITIES IN PATIENTS WITH ALOPECIA AREATA"**

Name of the Participant :

Name of the Principal (Investigator) : **Dr. S. Fathima**

Name of the Institution : Department of Dermatology & Leprosy, Rajiv Gandhi Govt. General Hospital, Chennai.

Documentation of the informed consent

I _____ have read it has been read for me, the information in this form. I was free to ask any questions and they have been answered. I am over 18 years of age and exercising my free power of choice, hereby give my consent to be included as a participant in the study.

1. I have read and understood this consent form and the information provided to me.
2. I have had the consent document explained in detail to me.
3. I have been explained about the nature of the study.
4. My rights and responsibilities have been explained to me by the investigator.
5. I agree to cooperate with the investigator and I will inform her immediately if I suffer from unusual symptoms.
6. I have not participated in any research study at any time.
7. I am aware of the fact that I can opt out of the study at any time without having to give any reason and this will not affect my future treatment in this hospital.
8. I hereby give permission to the investigators to release the information obtained from me as a result of participation in this study to the regulatory authorities, government agencies, and Institutional Ethics Committee. I understand that they are publicly presented.
9. My identity will be kept confidential if my data are publicly presented.
10. I am aware that if I have any question during this study, I should contact the investigator.

Participant's Initials :_____

For adult participants:

Name and signature / thumb impression of the participant (or legal representative if participant incompetent)

Name_____ Signature_____ Date_____

Name and Signature of impartial witness (required for illiterate patients):
Name _____ Signature_____ Date_____
Address and contact number of the impartial witness:
Name and Signature of the investigator or his representative obtaining consent:
Name _____ Signature_____ Date_____

சுய ஒப்புதல் படிவம்

ஆராய்ச்சியின் தலைப்பு : புழுவெட்டு நோயாளிகளிடையே பல்வேறு சிகிச்சை முறைகளை ஒப்பிட்டு பார்த்தல் குறித்த ஆய்வு.

பெயர் : வயது : தேதி : உள் / நோயாளி எண் :

..... என்பவராகிய நான் இந்த ஆய்வின் விவரங்களும் அதன் நோக்கங்களும் முழுமையாக அறிந்து கொண்டேன். எனது சந்தேகங்கள் அனைத்திற்கும் தகுந்த விளக்கம் அளிக்கப்பட்டது. இந்த ஆய்வில் முழு சுதந்திரத்துடன் மற்றும் சுயநினைவுடன்
பங்கு கொள்ள சம்மதிக்கிறேன்.

எனக்கு விளக்கப்பட்ட விஷயங்களை நான் புரிந்து கொண்டு நான் எனது சம்மதத்தைத் தெரிவிக்கிறேன். இச்சுய ஒப்புதல் படிவத்தை பற்றி எனக்கு விளக்கப்பட்டது.

இந்த ஆய்வினை பற்றிய அனைத்து தகவல்களும் எனக்கு தெரிவிக்கப்பட்டது. இந்த ஆய்வில் எனது உரிமை மற்றும் பங்கினை பற்றி அறிந்து கொண்டேன்.

இந்த ஆய்வில் பிறரின் நிர்பந்தமின்றி என் சொந்த விருப்பத்தின் பேரில்தான் பங்கு பெறுகிறேன் மற்றும் நான் இந்த ஆராய்ச்சியிலிருந்து எந்நேரமும் பின் வாங்கலாம் என்பதையும் அதனால் எந்த பாதிப்பும் ஏற்படாது என்பதையும் நான் புரிந்து கொண்டேன்.

இந்த ஆய்வில் கலந்து கொள்வதன் மூலம் என்னிடம் பெறப்படும் தகவலை ஆய்வாளர் இன்ஸ்டிட்யூசனல் எத்திக்ஸ் கமிட்டியினரிடமோ, அரசு நிறுவனத்திடமோ தேவைப்பட்டால் பகிர்ந்து கொள்ளலாம் என சம்மதிக்கிறேன்.

இந்த ஆய்வின் முடிவுகளை வெளியிடும்போது எனது பெயரோ, அடையாளமோ வெளியிடப்பட்டாது என அறிந்து கொண்டேன். இந்த ஆய்வின் விவரங்களைக் கொண்ட தகவல் தாளைப் பெற்று கொண்டேன். இந்த ஆய்விற்காக இரத்தப் பரிசோதனைகளும் (CBC, LFT, RFT, VCTC, VDRL, TFT) மற்றும் தேவைப்படுகின்ற நோயாளிகளுக்கு KOH Smear பரிசோதனையும் செய்து கொள்ள சம்மதிக்கிறேன்.

இந்த ஆய்வில் பங்கேற்கும் பொழுது ஏதேனும் சந்தேகம் ஏற்பட்டால், உடனே ஆய்வாளரை தொடர்பு கொள்ள வேண்டும் என அறிந்து கொண்டேன்.

இச்சுய ஒப்புதல் படிவத்தில் கையெழுத்திடுவதன் மூலம் இதிலுள்ள அனைத்து விஷயங்களும் எனக்கு தெளிவாக விளக்கப்பட்டது என்றும் தெரிவிக்கிறேன் என்று புரிந்து கொண்டேன். இச்சுய ஒப்புதல் படிவத்தின் ஒரு நகல் எனக்கு கொடுக்கப்படும் என்றும் தெரிந்து கொண்டேன்.

பங்கேற்பாளர் / பாதுகாவலர் கையொப்பம் தேதி :

ஆய்வாளர் கையொப்பம் தேதி :

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI 600 003

EC Reg.No.ECR/270/Inst./TN/2013
Telephone No.044 25305301
Fax: 011 25363970

CERTIFICATE OF APPROVAL

To
Dr.S.Fathima
I Year Post Graduate in MD DVL
Department of Dermatology
Madras Medical College
Chennai 600 003

Dear Dr.S.Fathima,

The Institutional Ethics Committee has considered your request and approved your study titled **"COMPARISON OF VARIOUS TREATMENT MODALITIES IN PATIENTS WITH ALOPECIA AREATA " - NO.25082017**

The following members of Ethics Committee were present in the meeting hold on **01.08.2017** conducted at Madras Medical College, Chennai 3

1. Prof.Dr.C.Rajendran, MD., :Chairperson
2. Prof.R.Narayana Babu,MD.,DCH.,Dean, MMC,Ch-3 : Deputy Chairperson
3. Prof.Sudha Seshayyan,MD., Vice Principal,MMC,Ch-3: Member Secretary
4. Prof.N.Gopalakrishnan,MD,Director,Inst.of Nephrology,MMC,Ch : Member
5. Prof.S.Mayilvahanan,MD,Director,Inst. of Int.Med,MMC, Ch-3 : Member
6. Prof.Rema Chandramohan,Prof.of Paediatrics,ICH,Chennai : Member
7. Prof. Susila, Director, Inst. of Pharmacology,MMC,Ch-3: Member
8. Prof.K.Ramadevi,MD., Director, Inst. of Bio-Chemistry,MMC,Ch-3 : Member
9. Thiru S.Govindasamy, BA.,BL,High Court,Chennai : Lawyer
10. Tmt.Arnold Saulina, MA.,MSW., :Social Scientist
11. Tmt.J.Rajalakshmi, JAO,MMC, Ch-3 : Lay Person

We approve the proposal to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

Member Secretary – Ethics Committee

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE
CHENNAI-600 003

URKUND

Urkund Analysis Result

Analysed Document:	theesis-COMPARISON_OF_VARIOUS_TREATMENT_MODALITIES_IN_PATIENTS_W (D42504527)
Submitted:	10/13/2018 9:44:00 AM
Submitted By:	fthms15@gmail.com
Significance:	1 %

Sources included in the report:

http://www.jpsr.pharmainfo.in/Documents/Volumes/vol6issue04/jpsr06041401.pdf

Instances where selected sources appear:

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PLAGIARISM CERTIFICATE

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