""STUDY OF INTRA-LESIONAL TRIAMCINOLONE ACETONIDE VERSUS AUTOLOGOUS PLATELET-RICH PLASMA IN THE TREATMENT OF ALOPECIA AREATA OF SCALP"

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LIST OF ABBREVIATIONS

PRP – Platelet-rich plasma

AA – Alopecia areata

MHC – Major histocompatibility complex

IFN – Interferon

NK – Natural killer

SALT – Severity of alopecia tool

TGF – Transforming growth factor

JAK – Janus kinase

PDE – Phosphodiesterase

MDHN – Mac Donald Hull and Norris grading

ABSTRACT

Introduction:

Alopecia areata is an autoimmune condition involving scalp and body, causing hair loss without inflammation or scarring. Hair follicle stem cells are not affected, and regrowth of hair is possible. Management is mainly aimed at controlling pathology.

Aim:

To evaluate and compare efficacy of intralesional triamcinolone acetonide with intralesional autologous platelet-rich plasma (PRP) in treatment of two different scalp alopecia areata patches in same individual.

Materials and methods:

This is a hospital-based prospective follow-up study. Patients of age group 6 years and above, with 2 or more patches of hair loss over scalp and those who have not taken any treatment in last 6 months were included.

Patients were given 2 separate treatment modalities in 2 different patches of hair loss in the same individual. One patch (Patch A) was treated with intralesional triamcinolone acetonide (10mg/ml) and other with autologous PRP (Patch B). 4 follow-ups were done at three weeks intervals. Comparison of results at each follow-up was based on trichoscopic findings and Mac Donald Hull and Norris (MDHN) grading system.

Results:

Out of 32 patients, based on the MDHN grading system, 24 (75%) showed Grade 3 and 8 (25%) showed Grade 4 with intralesional triamcinolone at 12 weeks. 16 (50%) patients showed Grade 2, 13 (40.6%) showed Grade 3 and 3 (9.4%) showed Grade 4 with autologous PRP at end of 12 weeks. On trichoscopy, both the treatment modalities showed an increase in short vellus hair and terminal hair and a reduction in yellow dots and exclamation hair at 12 weeks. No change in black dots was seen. No adverse effects were reported in both the interventions.

Conclusion:

Intralesional triamcinolone acetonide is more effective as compared to intralesional PRP in the treatment of alopecia areata and is a safe option. Following treatment, an increase in short vellus hair and terminal hair while a reduction in yellow dots and exclamation hair is seen. Black dots have no significant change.

Keywords: Alopecia areata, triamcinolone, platelet-rich plasma, trichoscopy

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INTRODUCTION

Alopecia areata is a form of non-scarring alopecia. It is an autoimmune condition involving the scalp and the body, causing hair loss without any clinical inflammatory signs.[1]

It is one of the most frequently encountered forms of hair loss in the dermatology practice and is implicated in 25% of all alopecia cases.[2] Both genders are equally affected, but some studies have shown a slight female preponderance, maybe due to greater concern, and females are more likely to seek medical attention as they age.[2-4]

A linear increase in the incidence of alopecia areata is seen with age, although any age group can be affected. The mean age of onset is 25-36 years, and the highest prevalence is seen in the age group of 30-59 years. Early age of onset (between ages 5–10 years) presents with a more severe subtype.[2-5]

The disease is characterised by a well defined, coin-shaped patch or a band like pattern of hair loss over the scalp and body. Various clinical patterns of hair loss can be seen and can show progression to alopecia totalis or alopecia universalis.[1,4,6]

Dermoscopic imaging of the scalp and hair is known as trichoscopy. It is a non-invasive, simple bedside technique which helps in diagnosing various hair and scalp disorders. Exclamation hair and black dots can be seen in the acute, progressive stage, while empty follicles and yellow dots are seen in long-standing cases. In regrowing patches, short vellus and coiled hair are seen.[7,8]

A rapid change of hair from anagen to telogen phase is seen in the affected patches, which leads to localised hair loss.[6] The disease process spares the destruction of hair follicle stem cells. Therefore, regrowth of hair is possible.[9]

Spontaneous remission can be seen in about 34-50% of the cases within 1 year, although the relapse rate is high.[4]

Management is mainly aimed at controlling the pathology and is not curative. Currently, many therapeutic options are available, and there is a continued search for newer modalities of controlling the symptoms. Most of the therapeutic methods are either immunosuppressive or immunomodulatory.[1]

Intra-lesional corticosteroids are the most popular modality of treatment.[10] The primary mechanism of action is immunosuppression by suppressing the T-cell mediated immune attack on the hair follicle. Various preparations used are triamcinolone acetonide, triamcinolone hexacetonide and hydrocortisone acetate. The preferred intra-lesional preparation is triamcinolone acetonide because of fewer side effects like atrophy.[11]

Platelet-rich plasma (PRP) is an autologous preparation of platelets in concentrated plasma. Platelets release various growth factors which act on stem cells in the hair follicles and stimulate the formation of new follicles and promote neovascularisation in alopecia areata patches.[12,10] It promotes cell proliferation and has anti-apoptotic properties, thus prolonging the anagen phase of the hair cycle and increasing hair growth.[12] Therefore, platelet-rich plasma has recently attracted attention because of its simplicity and efficacy in treatment without the risk of allergic reactions and fewer side effects like atrophy and hypopigmentation.[1]

This study was undertaken to find out the efficacy of intralesional autologous platelet-rich plasma compared to intralesional triamcinolone acetonide in the treatment of alopecia areata of the scalp, along with trichoscopic changes.

AIM OF THE STUDY:

To evaluate and compare the efficacy of intralesional triamcinolone acetonide (10mg/ml) with intralesional autologous platelet-rich plasma in the treatment of two different scalp alopecia areata patches of the same individual.

OBJECTIVE OF THE STUDY:

1. To compare the rate of regrowth of hair following both the interventions
2. To compare the incidence of side effects following both the interventions
3. To compare the trichoscopic changes following both the interventions

REVIEW OF LITERATURE

Anatomy of hair

Hair consists of the following structures:

1. *Hair shaft*: It is the visible part of the hair present outside the skin. It comprises of a thin, flexible structure of keratinised, non-living epithelial cells. It is further broken down into the following layers:
    i. *Cortex*: It makes up the majority of the hair fibre. It is responsible for establishing the physical and mechanical features of hair (including structure, texture and colour) and is mostly made up of microfibrils.
    ii. *Cuticle cells*: They are seen surrounding the cortex.[13]It comprises a single layer of cells enclosed in the inner (internal) root sheath and outer (external) root sheath. The inner sheath plays a vital role in the shaping of the hair shaft.[13,14]
    iii. *Central medulla*: This layer is present in thicker hair.[13]

2. *Hair follicle*: It is present below the skin surface and provides the base for the growth of hair. It is divided into three segments:
    i. *Infundibulum*: It extends from the ostium above to the opening of the sebaceous duct below.
    ii. *Isthmus*: It spans from the entry of the sebaceous duct above to the insertion of the arrector pili muscle below.

iii. *Hair bulb*: It extends to the base of the follicle.[14] This is responsible for the active production of hair. It encloses the dermal papilla in the dermis. The dermal papilla is a crucial structure which is rich in stroma, nerve fibres and capillaries. Certain essential growth factors responsible for hair growth, like insulin-like growth factor, stem cell factor, keratinocyte growth factor and bone morphogenetic protein, are produced by the dermal papilla, which are also crucial for the size and colour of the hair shaft. The hair bulb is divided by the Auber line into two regions. Below this line, immature cells are present, which are mitotically active and move upward as they enlarge and undergo elongation.

From outside to inside, the hair follicle is encased by the following layers:

i. *Outer root sheath*: The bulge is present at the insertion of the arrector pili muscle, which contains multipotent stem cells which differentiate into melanocytes and keratinocytes.

ii. *Inner root sheath:* It produces keratins and trichohyalin, which plays a role in affixing the hair shaft to the follicle. It consists of the Henle layer, the Huxley layer and the cuticle. The cuticle gives the hair its shape and an untangled appearance.[13]

Based on the gross morphology, hair can be divided into:

1. *Terminal hair:* These are dark-coloured, long, thick hair and visible to the human eye. They are present over the scalp, axillae, pubic area, beard, eyebrows and eyelashes.

2. *Vellus hair*: These are unmedullated and, thus, are shorter and more refined. They lack pigmentation. Hence they give a hairless appearance.[13,15]
3. *Lanugo hair*: These are fine unmedullated hair seen in neonates. They are usually shed in utero or within the first few weeks of life.

The palms and soles, lips, labia minora, and glans penis are the only portions of the body that are entirely hairless.[13]

Hair cycle

Individual hair follicles show cyclic growth. It passes through stages of rapid growth and elongation, alternating with quiescence and regression influenced by the interaction of growth factors and apoptotic signals and the cyclical activation of stem cells.[13,16]

It can be divided into the following phases:

1. *Anagen*: This is the phase of active hair fibre production, leading to the growth of a new hair shaft. About 85-95% of hairs are present in the anagen phase at any point in time. It can last for 2-6 years for scalp hair and only takes a few months for eyebrows and eyelashes.[14] It is further divided into 7 stages
    i. *Anagen I*: These are seen in xenografts and are not commonly seen *in situ* owing to their short duration. A small dermal papilla, a secondary hair germ shaped like a triangle or crescent and commencement of proliferation at the germ's base are all crucial traits at this stage.
    ii. *Anagen II*: Dermal papilla has a wider stalk. The secondary hair germ undergoes proliferation and becomes enlarged and the crescent more prominent. Localised expansion is seen at the base of the germ. The follicle still resides in the dermis.

iii. *Anagen III*: The hair follicles in this stage have a hair shaft and inner root sheath, and the hair bulb extends to the adipose layer. The dermal papilla enlarges further and assumes an oval shape. Matrix is formed and is 4-5 cell layers thick. It can be further divided into three sub-stages based on the appearance of the hair shaft: Anagen IIIa shows an absence of cortex, whereas Anagen IIIb and IIIc show the presence of cortex. Anagen IIIc hairs have long shafts, and melanogenesis begins in this stage, although hairs still lack visible pigmentation.

iv. *Anagen IV:* Hair shaft has reached complete maturity and contains a well-defined medulla, cortex and cuticle. Melanogenesis is now fully reactivated, and the pigment is grossly visible in the hair shafts. The hair shaft grows up to the level of the sebaceous gland. The hair bulb lies in the upper adipose layer, and a connective tissue sheath trail, which is seen proximal to the hair bulb, guides the downward growth of the hair follicle.

v. *Anagen V:* The hair bulb extends further down into the adipose layer through the sheath, which later disappears. The hair shaft's tip reaches the hair canal. The dermal papilla assumes an onion shape. The pigmentation in the hair matrix extends to the Auber's line.

vi. *Anagen VI*: The majority of the hair follicles are present in this stage. The hair bulb now lies deep in the adipose layer, and the hair shaft is visible above the skin. The melanin content in the hair follicles is maximum and extends below the Auber's line.[17]

2. *Catagen*: It begins at the end of anagen and is the transition or regression phase.[13,14] It lasts only for a few weeks, making it the shortest phase. The dermal papilla moves up and

comes in close contact with the bulge. A club hair is formed, containing a white, hard node at the end.[14] It can be further divided into the following three stages:

i. *Early Catagen*: Earliest signs of transition to catagen include reduction in the volume of hair matrix and dermal papilla, which later assumes an almond shape. Melanogenesis comes to a halt at this stage, leading to loss of pigmentation at the proximal end of the hair shaft. Also, some melanin incontinence into the dermal papilla may be seen. The length of the follicle and the morphology of the bulge region remain constant, and the lower portion of the hair follicle comes to lie below the dermal-adipose junction.

ii. *Mid-catagen*: There is a further reduction in the volume of the dermal papilla and matrix, which is now 1-2 cell layers thick and only partially covers the former. The club hair is prominent, assuming a brush-like morphology, and now lies above the dermal-adipose boundary. Between the club hair and the dermal papilla lies the newly formed epithelial strand, which is thin, lacks pigment and has a ruffled, zipper-like appearance.

iii. *Late catagen*: The dermal papilla is smaller and assumes a ball shape, and there is an absence of the hair matrix. The club hair becomes visible and is now prominent. There is further shortening of the epithelial strand, which contains apoptotic cells. A thickened connective tissue sheath, which extends below the dermal papilla into the adipose tissue, becomes prominent and contains melanin clumps. The sebaceous glands show apoptosis as well.[17]

3. *Telogen*: It is also known as the resting phase, where the hair follicle is dormant, and the growth of the hair shaft comes to a stop. At any time, about 10-15% of hairs are present

in this stage. Scalp hair remains in this stage for about a year and eyelashes for a few weeks.[13] The hair follicle lies above the dermal-adipose boundary. The club hair, which is unpigmented and serrated, becomes prominent. The dermal papilla is very compact, and the unpigmented epithelial strand (which is now called the secondary hair germ) separates it from the club hair. This stage lacks apoptotic cells.[17] The club hair is now dead and is eventually shed (through a process known as exogen), and the next anagen phase begins.[14]

Alopecia

Hair loss over the scalp or body, regardless of the cause, is termed alopecia.[18] It is of 2 types:

1. *Non-scarring alopecia*: Hair loss that is reversible is called non-cicatricial or non-scarring alopecia.[15] The hair follicle is preserved, and hair loss is due to changes in the hair cycle, size of follicle, breakage of hair or a combination of these.[4]
    i. *Androgenetic alopecia*: This is the most common type of hair loss. It is due to the miniaturisation of the hair follicles caused by dihydrotestosterone in genetically predisposed individuals. It affects about 50% of the men by 50 years and 40% of the women by 70 years. It is characterised by diffuse loss of hair at the crown. In males, there is recession of the frontal hairline, which is preserved in females.[4,15]
    ii. *Alopecia areata*
    iii. *Telogen effluvium*: Diffuse, excessive loss of hair over the scalp is known as telogen effluvium. It commonly affects females of the age group 30-60 years. It

can be acute (lasting for less than 6 months) or chronic (lasting longer than 6 months). Acute telogen effluvium may be secondary to various triggers such as childbirth, febrile illness, major surgery or rapid weight loss, which is usually present 2-4 months before the onset of hair loss. Chronic type is commonly associated with female pattern hair loss. This is due to the premature transition of hair to the telogen phase, which is then shed.[4,15]

iv. *Anagen effluvium*: This is the loss of hair during the anagen phase secondary to radiation therapy or chemotherapy. Onset is usually 1-4 weeks following exposure. It is characterised by diffuse loss of hair with narrow and fractured hair shafts, which is a characteristic sign.[15]

v. *Trichotillomania*: It's a hair-pulling impulsive behaviour disorder in which sufferers can't stop themselves from pulling out their own hair. It is characterised by patches of hair loss that are irregular (also known as tonsural pattern), along with short, broken hair which are irregularly distributed. In children, boys are affected more than girls, and it is self-limiting. On the other hand, women are more commonly affected in adult trichotillomania, and it is commonly associated with mood or anxiety disorders.[4,15]

vi. *Traction alopecia*: Hair loss resulting from chronic mechanical tension on the hair, commonly secondary to hair styling. In the early stages, hair loss is transient. On prolonged force, loss of follicles may lead to irreversible loss of hair. Scarring and inflammation may be seen.[4,15]

vii. *Tinea capitis*: It is a dermatophyte infection seen commonly in children, characterised by loss of hair, which is patchy, with inflammation and scaling of the scalp.

viii. *Short anagen syndrome*: Commonly seen in children, this condition is characterised by a reduction in hair length, where hair density and strength is normal.[4]

ix. *Loose anagen syndrome*: There is diffuse hair loss with short, thin, dull and unruly hair. Females and children are more commonly affected. Due to premature keratinisation of the IRS, the adhesion with cuticle is impaired, leading to hair breakage.[4,15]

x. *Temporal alopecia triangularis*: This alopecia is characterised by a bald spot which is triangular or lancet-shaped with a normal number of hair follicles. The hair present on the patch are mostly vellus hair and very few terminal hair.

2. *Scarring alopecia*: Hair loss that causes atrophy of the skin and destruction of follicles, thus making it irreversible, is known as cicatricial or scarring or permanent alopecia.[15] It is secondary to inflammation or malignancy (like cutaneous lymphoma).[4]

    i. *Lichen planopilaris*: It is a type of lichen planus. It is a chronic inflammatory disease characterised by permanent destruction of hair follicles. There is patchy loss of hair with central scarring and follicular erythema at the edges and scaling.[4,15] It is commonly observed in women involving the crown and parietal areas of the scalp. This is a T-lymphocyte mediated attack on the hair follicles.[15]

ii. *Frontal fibrosing alopecia*: It is a variant of lichen planopilaris commonly affecting post-menopausal women. The pattern of hair loss involves the frontal and frontotemporal hairline and eyebrows.[4]

iii. *Chronic cutaneous lupus erythematosus*: It is characterised by well defined, scaly, erythematous, indurated papules and plaques, which later progress to atrophic plaques with follicular plugging, telangiectasia and depigmentation. Women of 20-45 years of age are commonly affected. Discoid lupus erythematosus is the most common form and accounts for 50-85% of cases.[4,15]

iv. *Central centrifugal cicatricial alopecia*: Loss of hair starts at the posterior crown or vertex and spreads in a centrifugal pattern to involve the whole scalp. It is seen in middle-aged African-American women and may be influenced by factors such as chemicals and pressure applied to the hair.[4,15]

v. *Folliculitis decalvans*: It is an inflammatory reaction of the hair follicle to *Staphylococcus aureus* colonisation with neutrophilic and lymphocytic infiltrates. There is patchy hair loss with pustules at the margins.[4]

Trichoscopy

Dermoscopic imaging of the scalp and hair is known as trichoscopy. The term "Trichoscopy" was coined by Lidia Rudnicka and Malgorzata Olszewska in 2006.[7] It is a non-invasive, simple bedside technique which helps in diagnosing various hair and scalp disorders.[7,8] A handheld dermoscope or a digital videodermoscopy system can be used. Because of its application in the differential diagnosis of hair and scalp disorders, this method has shown

increasing popularity. Structures visualised with a dermoscope are analysed.[19] Basic structures visualised can be divided into the following groups:

1. *Hair shaft*: Normal hair shafts show uniformity in shape and colour. The medulla may be continuous, interrupted, fragmented or absent. Short, hypopigmented vellus hairs make up approximately 10% of normal human scalp hairs. Hair shaft abnormalities such as exclamation mark hair, tapered hair, tulip hair, pigtail hair, comma hair or corkscrew hair, as well as genetic hair shaft disorders, like monilethrix, trichorrhexis nodosa or pili torti, can be observed. Each follicular unit contains 2-3 hairs in a healthy individual. This number can be reduced in non-scarring alopecias and increased in tufted folliculitis, folliculitis decalvans and lichen planopilaris.[20] Exclamation mark hair are seen in alopecia areata and trichotillomania. They show narrowing of the hair root, which suggests a defect in the anchoring of the hair.[4]
2. *Hair follicle openings*: "Dots" is the term used to depict hair follicle openings. Trichoscopy helps in observing whether the follicle openings are normal, empty, fibrotic or contain hyperkeratotic plugs or hair residues.
    a. *Black dots*: Formerly known as cadaverized hairs, black dots are commonly seen in alopecia areata, dissecting cellulitis, tinea capitis, chemotherapy-induced alopecias, trichotillomania and following laser depilation. They represent pigmented hair which are broken or destroyed at the level of the scalp.
    b. *Yellow dots*: These structures are found in alopecia areata, discoid lupus erythematosus and female pattern androgenetic alopecia and can be rarely seen in telogen effluvium and trichotillomania. They represent the follicular infundibula containing keratotic material or sebum. In female pattern androgenetic alopecia,

they are distributed predominantly over the frontal area of the scalp. In dissecting cellulitis, yellow dots appear as large 3D soap bubbles imposed over dark dystrophic hairs.

c. *White dots*: White dots can be described as 2 types. The classic, big, irregular white dots are seen in lichen planopilaris and correspond to areas of perifollicular fibrosis. Small, regular, pinpoint white dots are the other type, seen in sun-exposed areas and in dark skin individuals and are observed regardless of hair loss. They represent empty follicles or the opening of eccrine sweat ducts and are observed on a pigmented background.

d. *Red dots*: These are seen in discoid lupus erythematosus and suggest a good prognosis in these patients.

e. *Other*: Brown or brown-grey dots are regularly distributed dots seen over the eyebrows in patients with frontal fibrosing alopecia. This indicates a good prognosis for regrowth of eyebrows.[19,20]

3. *Perifollicular epidermis*: Abnormalities in scalp skin colour or structure can be visualised using trichoscopy. Hyperpigmentation is seen commonly in androgenetic alopecia, and perifollicular fibrosis is seen in certain cicatricial alopecias.

    a. *Scaling*: Diffuse white scales are observed in psoriasis, discoid lupus erythematosus and dry skin. Seborrheic dermatitis and ichthyosis show diffuse yellowish scales. White perifollicular tubular scaling is seen in lichen planopilaris, while yellow tubular perifollicular scaling with a collar can be found in folliculitis decalvans.[19]

    b. *Colour*:

i. *Brownish hyperpigmentation*: Pigment distribution in a honeycomb pattern is seen in healthy individuals and is more prominent in dark-skinned individuals. Perifollicular brown areas (peripilar sign) is common in androgenetic alopecia and telogen effluvium which is due to sun exposure leading to tanning.[7,19] Scattered pattern is seen in discoid lupus erythematosus and actinic keratosis.

ii. *White areas*: Cicatricial alopecias show the presence of white areas.

iii. *Violaceous blue areas*: These areas can be seen in lichen planopilaris and discoid lupus erythematosus.

iv. *Pink areas*: They represent early fibrosis in cicatricial alopecias.

v. *Yellow areas*: These are found in dissecting cellulitis, follicular pustules and secondary to bacterial infections.

vi. *Red areas*: These are seen in inflammatory conditions, extravasation of red blood cells, erosions and vascular abnormalities.

c. *Discharge*: Yellow to yellow-red discharge is seen in folliculitis decalvans, bacterial infections, dissecting cellulitis and tinea capitis. Monoclonal gammopathy shows white follicular spicules.

d. *Others*: Starburst pattern of hyperplasia is a feature of folliculitis decalvans.[19]

4. *Blood vessels*: The type, arrangement and number of cutaneous microvasculature observed in trichoscopy is important in the diagnosis of scalp disorders.[19] They are more prominent in light-skinned individuals, whereas in dark-coloured skin, the pigment pattern overlaps the vessels.[7]

a. *Interfollicular red loops*: Multiple, regularly arranged, hairpin shaped vessels observed in normal scalp or in inflammatory disorders like scalp psoriasis, seborrheic dermatitis or discoid lupus erythematosus.[7,19] Absence of these structures can point towards epidermal atrophy.[7]
b. *Interfollicular twisted loops*: Twisted coils of vessels are seen better when the probe is placed tangential to the skin surface. It is seen in conditions such as psoriasis and folliculitis decalvans.
c. *Arborizing red lines*: These represent the sub-papillary plexus and are seen at higher magnifications, visualised as red lines underlying the loops. These can be visible on normal scalp.[7]

Alopecia areata

Alopecia is derived from the Greek word "*alopex*", meaning fox, due to the loss of fur seen in fox mange. "*Areata*" is a Latin word meaning "area". The use of the word alopecia dates back to Hippocrates. John Jonston, a Polish physician, was the first to use the term "Alopecia area" in his book "MedicinaPractica". Sauvages de Lacroix, a French physician, introduced the term "Alopecia areata" in his book "MedicinaPractica", where it was used as a broad term for patchy hair loss secondary to various conditions.[21]

Alopecia areata (AA) is an autoimmune condition involving the scalp and body, causing hair loss without scarring.[1]

Epidemiology

Alopecia areata is responsible for about 25% of all alopecia cases.[2] It affects both the genders equally, but studies have shown a female preponderance due to higher concern.[2-4]

Incidence increases with age in a linear fashion, although any age group can be affected. Highest prevalence is seen in the age group of 30-59 years. A more severe course, like progression to alopecia universalis, is seen in children (onset between ages 5-10 years).[2-5]

Aetiology

Genetics

A high incidence of disease in siblings and family members is seen among patients with AA. Men show a higher incidence of a positive family history compared to women. Genome-wide studies have shown various single-nucleotide polymorphisms related to AA. Human leukocyte antigen-DR (HLA-DR) on chromosome 6 is more likely to contain genes responsible for AA. These genes influence the CD4+ and CD8+ T-cells implicated in the pathogenesis of AA.[3,4]Genes encoding Natural killer cell receptor D (NKG2D) also play an important role and is implicated in the pathogenesis of AA.[5]

Environmental factors

Environmental factors can either exacerbate or induce the disease process. An important cause for AA is psychological and physiological stress. There is a correlation between elevated adrenocorticotrophic hormone, corticosterone and estradiol and elevated pro-inflammatory cytokines in the skin. Infections, vaccinations, hormone fluctuations and changes in diet lead to stress.[3]

Immune privilege zone

The hair follicle is a zone of immune privilege. This is due to low expression of MHC-I and β2 macroglobulin, production of immunosuppressants like α-melanocyte-stimulating hormone and transforming growth factor-β (TGF-β) and reduced antigen-presenting cell activity. This inhibits the infiltration of CD56+/NKG2D+ NK cells. Autoimmune responses against autoantigens related with pigment production in melanocytes in the hair follicle are inhibited.[3,4]

In AA, an unknown antigen leads to the breakdown of this zone. Inflammatory cells, NK cells, CD8+ and CD4+ cell infiltration is seen in the hair follicle induced by interferon-γ (IFN-γ) and interleukin-2, resulting in hair loss.[3]

Pathophysiology

An important trigger is the breakdown of immune privilege. It is primarily a hair cycle disorder, where the anagen hair follicles are converted to catagen phase prematurely due to the attack on hair matrix with inflammatory cells.[5] There is an increase in dystrophic hair due to increased hair shaft fragility.[4] The hair follicle stem cells are spared, and hence, regeneration is possible. Also, the follicles entering anagen phase fail to advance beyond anagen III/IV phase.[5]

Clinical features

AA is characterised by well-defined patches of hair loss, affecting any hair-bearing part of the body but commonly over the scalp and beard, with the skin appearing normal or slightly erythematous.[4,5] Various types of Alopecia areata have been described:

1. *Patchy AA*: It consists of single or multiple, discrete or conjoined patches of hair loss.
2. *Alopecia totalis*: It is the total or near-total loss of scalp hair.

3. *Alopecia universalis*: It is the total or near-total loss of all hair over hair-bearing areas of the body.
4. *Alopecia incognita*: It is the absence of nail involvement seen in patients with diffuse hair loss with positive hair pull test, yellow dots and short regrowing hairs.
5. *Ophiasis*: Band-shaped loss of hair along the circumference of the head, i.e., along the temporal and occipital areas of the scalp.
6. *Sisaipho*: It is alopecia involving extensive areas of the scalp with sparing of the periphery.
7. *Marie Antoinette syndrome/Canities subita*: It is an acute diffuse loss of hair, seen as "overnight greying" of hair due to sparing of non-pigmented hair.[4]

Nail involvement is seen in 10-15% of cases and indicates a more severe form of the disease. Fine stippled pitting is commonly seen.[5] Other features include trachyonychia, Beau's lines, onychorrhexis, thinning or thickening of the nail plate, onychomadesis, leuconychia (punctuate or transverse type), red lunulae and koilonychia.[3]

New patches can appear after varying intervals of time or coalesce to form large areas of hair loss. Spontaneous regrowth of hair can be seen in 34-50% of the patients within a few months to years, or the patches may show an increase in size. Regrowing hairs are initially fine and lack pigment and gradually acquire normal thickness and colour.[4] Progression to alopecia totalis or universalis is seen in about 14-25% of the patients. Full recovery in these patients is seen in <10% of the cases.[5]

An important prognostic factor is the extent of hair loss and age of the patient at onset. Onset of the disease in childhood and ophiasis pattern carries a poor prognosis. An associated family history, nail involvement, history of atopy or other associated autoimmune disorders

(thyroid disease, lupus erythematosus, vitiligo, psoriasis, rheumatoid arthritis, inflammatory bowel disease) also show a poor prognosis. Later age of onset of the disease usually carries a favourable prognosis.[5]

Histopathology

A peribulbar lymphocytic infiltration composed of CD4+ and CD8+ T-cells surrounds anagen follicles in the acute and subacute stages of the disease, forming a "swarm of bees" pattern. A shift from catagen stage to telogen stage is seen with miniaturisation of the follicle. Other features seen around the follicle include oedema, microvesiculation, apoptosis, and the presence of macrophages and foreign body giant cells.

Inflammation may persist into the chronic stage or may resolve. There is an increase in the number of catagen and telogen hair. Pigment incontinence may be seen.

Minimal inflammation and an increase in anagen hair marks the recovery stage of the disease.[3]

Trichoscopy in alopecia areata

Diagnosis of alopecia is mainly clinical, and trichoscopy is helpful for differential diagnosis. Trichoscopic findings of alopecia areata depend on the disease activity, severity and duration. The characteristic features are yellow dots, exclamation mark hairs, tapered hairs, black dots, broken hairs and regrowing hairs.[19]

Lacarrubba *et al.*[22] conducted a study on 200 patients with alopecia areata and divided the features based on the duration into an acute and chronic disease. Acute alopecia areata showed features like exclamation mark hair, black dots and vellus hair. In long-standing cases, smooth,

thin scalp skin and prominent follicular openings with keratotic plugs were evident. Regrowing hair was seen as sparse vellus hairs.

A study conducted by Inui *et al.*[23] showed similar findings in 300 patients with alopecia areata. Black dots, yellow dots and short vellus hairs were considered markers for disease activity and severity. Exclamation mark hair indicated worsening of disease activity. Short vellus hair was found in long-standing and inactive disease. Patients with improving disease also showed the presence of black dots, tapering hairs and broken hairs in this study.

Thus, exclamation hair and black dots are seen in the acute, progressive stage, while empty follicles and yellow dots are seen in long-standing cases. In regrowing patches, short vellus hair are seen.[7,8]

Prognosis

Hair follicle stem cells are spared in the inflammatory process. Therefore, hair regrowth is possible, even in chronic cases and those with alopecia totalis and universalis. Some patients (34-50%) show spontaneous regrowth of hair, while it is persistent in others, and new patches may appear. Progression to alopecia totalis or universalis is seen in 14-25% of the cases. Indicators for poor prognosis are:

1. Onset at a young age
2. Family history of alopecia areata
3. Extensive involvement, as seen in alopecia totalis or universalis
4. Ophiasis pattern of hair loss
5. Long-standing disease >1 year
6. Nail involvement

7. Atopy
8. Association with other autoimmune diseases.[2,4]

Management

1. **Local injectable therapy:**

Intralesional steroid

Intralesional steroids are the most commonly used treatment in alopecia areata. This is more suited for patchy alopecia areata. Triamcinolone acetonide is the most commonly used formulation, given as 5-10 mg/ml at intervals of 2-6 weeks. The steroid is injected into the upper subcutaneous tissue via a fine needle injection using multiple injections.[4,5]

Steroids exert their effect via their anti-inflammatory and anti-proliferative effects. It acts by reducing the local inflammatory cells along with downregulation of several interleukins and chemokines and upregulation of genes encoding keratins.[4]

A significant adverse effect to be kept in mind is local cutaneous atrophy which can resolve spontaneously in a few months. It does not prevent the development of new patches at other sites and is not suitable for use in rapidly progressive disease, in alopecia totalis or universalis. Relapse can be seen following the cessation of treatment.[4,5]

Intralesional PRP

Platelet-rich plasma is an autologous preparation of platelets in concentrated plasma. Various growth factors and cytokines are released from the platelets, such as platelet-derived growth factor, transforming growth factor, vascular endothelial growth factor, insulin-like growth factor, epidermal growth factor and interleukin-1. It has an anti-apoptotic effect on the

dermal papilla cells and prolongs the anagen phase of the hair cycle, thus stimulating hair growth and prolonging the survival of the hair follicle.[3,12]

For the preparation of PRP, 10 ml of the patient's venous blood is collected in a vaccutainer containing anticoagulant sodium citrate and is centrifuged by double spin centrifugation. Initially, the blood is centrifuged by 'light spin' centrifugation (1600 rpm for 10 minutes). The upper layer contains the buffy coat and plasma, which is taken into another vaccutainer, followed by 'heavy spin' centrifugation (3400 rpm for 7 minutes). Then, three-fourths of the supernatant plasma is discarded, and the resultant buffy coat is used as PRP.[1,24]

Intralesional triamcinolone acetonide vs intralesional autologous PRP

Ranpariya *et al.*[1] conducted a study to determine the efficacy of intralesional triamcinolone acetonide (10mg/ml) with intralesional PRP in the treatment of alopecia areata. After 3 months of treatment, the triamcinolone treated group showed an adequate response with a higher mean score according to the Mac Donald Hull and Norris grading system and limited side effects. Recurrence was observed in 3 patients while on treatment with PRP.

A study by Balakrishnan *et al.*[24] was done on 40 patients of alopecia areata equally divided into 2 groups, one receiving intralesional triamcinolone and the other intralesional PRP over a period of 12 weeks and results were compared based on SALT scores. PRP was found to be more effective, but the reduction in SALT scores among the two groups after the first and final review was not found to be statistically significant.

Another study comparing the response to the two treatment modalities done by Agrawal *et al.*[10] depicted triamcinolone to have a better response than PRP. Trichoscopic evaluation was done based on the presence of dystrophic hair. 78.6% of the patients treated with triamcinolone

showed no dystrophic hair at the end of the study as compared to 69.2% of the patients receiving PRP.

A similar study conducted by Chang *et al.*[25] showed a favourable response to intralesional corticosteroids in 6 patients with more than 50% scalp involvement.

Trink *et al.*[26] conducted a half-head study to evaluate the efficacy and safety of PRP in 45 patients with alopecia areata against a placebo. One half of the scalp was treated with intralesional PRP, triamcinolone acetonide or a placebo and the other half received no treatment. A significant increase in hair growth was seen on treatment with PRP as compared to triamcinolone treated patients. Also, there was reduction in dystrophic hairs and burning and itching sensation in PRP treated areas. The difference seen in this study was not found to be statistically significant.

In a similar study in 2014 by Shumez *et al.*[9], PRP showed a faster response in alopecia areata when compared with triamcinolone at 6 weeks. This difference was also not statistically significant.

Ganjoo *et al.*[27] conducted a study on 63 patients of alopecia areata treated with intralesional triamcinolone acetonide (5mg/ml) once every 4 weeks over a period of 24 weeks. There was an increase in the number of vellus hairs while reduction was seen in exclamation mark hairs, broken hairs and black dots, which were considered markers for disease activity. Yellow dots remained unchanged and were not thought to be linked to disease activity.

Comparison of topical minoxidil 5% monotherapy and intralesional PRP with placebo was done by El Taieb *et al.*[28], where intralesional PRP showed a faster response. An increase in short vellus hairs and reduction in yellow dots were seen in patches treated with 5% minoxidil.

In patches treated with intralesional PRP, short vellus hairs and yellow dots were found to be reduced, and fully pigmented hairs were more following 3 months of treatment.

2. **Topical therapy:**

Topical steroids

This modality of treatment is best suited for limited patchy alopecia areata and in mild cases, although it has been found to be ineffective for alopecia totalis and universalis. A potent topical steroid, such as clobetasol, can be used in the form of lotion, foam or shampoo. Treatment can be stopped if there is no satisfactory response after 6 months. Occasionally, patients may develop folliculitis.[4]

Minoxidil

Topical minoxidil stimulates proliferation at the base of the hair bulb and differentiation above the dermal papilla leading to regrowth of hair. It is commonly used along with other treatment modalities. It can be used in extensive disease but is ineffective in alopecia totalis or universalis. Adverse effects are rare and include pruritus or dermatitis.[2,4]

Contact immunotherapy

Contact immunotherapy is used for patchy alopecia areata and is not suitable for patients with extensive hair loss. This modality involves the topical application of a potent allergen over the scalp. The first application helps to sensitise the patient. Following sensitisation, weekly applications of the allergen in concentrations to induce a mild contact dermatitis is performed.

Dinitrochlorobenzene, squaric acid dibutylester and diphenylcyclopropenone are the commonly used allergens. The mechanism of action of these allergens is unclear. These antigens

are thought to induce "antigenic competition" and cause migration of CD4+ T-cells away from the perifollicular area of the alopecia areata patch. Non-specific T-suppressor cell stimulation in the skin, increased TGF-β expression locally and activation of myeloid suppressor cells are the other proposed mechanisms.

Treatment can be discontinued if a satisfactory response is not obtained after 6 months. Prolonged treatment duration is required to observe a favourable result. Long term side effects are not expected. The most common adverse effect observed is severe dermatitis, which can be prevented by careful titration of the concentration of the allergen. Transient lymphadenopathy is another common adverse event. Rarely, urticaria, hyperpigmentation or hypopigmentation can be seen. Sensitisation of health professionals involved in delivering the therapy can occur, which can be prevented by avoiding contact with the skin while administration.[4]

Anthralin

Anthralin is another topical modality used which acts via its anti-inflammatory and anti-proliferative effects. It is used as a short contact therapy with a gradual increase in contact time until erythema or pruritus develops. It can cause severe irritation, folliculitis, regional lymphadenopathy and staining of skin, clothes and hair.[2,4]

Prostaglandin analogues

Prostaglandin analogues cause hypertrichosis of eyelashes and malar area hair as an adverse effect when used in open-angle glaucoma. Latanoprost and bimatoprost are used in alopecia areata of eyelash.[2]

3. **Phototherapy:**

Psoralen plus ultraviolet light A (PUVA) reduces the perifollicular inflammatory infiltrate seen in alopecia areata. It has been found to be useful in the treatment of alopecia totalis or universalis when used in conjunction with oral steroids. Some reports have also shown a poor response. Adverse effects reported include mild erythema, burning sensation and increased risk of melanoma.[2,4]

Excimer laser provides a safe and effective alternative to medical treatments. Narrow-band ultraviolet B (NBUVB) therapy is ineffective.[2,4]

4. **Systemic therapy:**

Systemic corticosteroids

High-dose pulsed corticosteroids are successful in the treatment of alopecia areata using either oral or intravenous regimens. Some patients may require daily doses with gradual tapering. Due to the associated adverse effects, they are best preserved for refractory, extensive and rapidly progressing cases. Relapse rates are high following the discontinuation of treatment. [3,4]

Systemic immunomodulators

Methotrexate, cyclosporine and azathioprine are immunomodulatory drugs that have shown variable clinical response. They reduce the perifollicular lymphocytic infiltrates. One of the adverse effects of cyclosporine is hypertrichosis by prolongation of the anagen phase.[5,6]

JAK inhibitors and PDE4 inhibitors

JAK inhibitors exert their action by inhibiting the upregulation of IFN-γ and reducing inflammation. Tofacitinib, ruxolitinib and baricitinib are the JAK inhibitors tried in alopecia areata. PDE4 inhibitor used is apremilast which is effective in extensive alopecia areata.[3]

Biologics

Infliximab, etanercept, adalimumab and efalizumab have been tried in the treatment of alopecia areata but were found ineffective or showed worsening while on treatment, suggesting that TNF-α is not an essential mechanism involved in pathogenesis.[2,6]

5. **Others:**

Camouflage

In extensive alopecia, the use of hairpieces like wigs or hair additions is a feasible option. Tattooing or artificial fibres can be used for eyelashes and eyebrows. They carry the risk of tractional alopecia and hair breakage from the clips and glue.

Miscellaneous treatments that have been used are:

1. Capsaicin
2. Topical bexarotene 1%
3. Fractional Er: Glass laser
4. Topical tretinoin 0.05%.[2]

METHODOLOGY

SOURCE OF DATA

Patients with clinically diagnosed alopecia areata, attending outpatient department of Dermatology, Venereology and Leprosy of B.L.D.E (Deemed to be University) Shri. B.M. Patil Medical College Hospital and Research Centre, Vijayapura, were enrolled for the study.

Period of study:

The study was conducted during the period of November 2019 to May 2021.

Study design:

A hospital-based prospective follow-up study.

Sample size:

With Anticipated Mean Difference of mean grading score between the patch A and patch B as 0.9 at endline and Anticipated SD as 1.2 the minimum sample size per group is 63 With 95% power and 1% level of significance.

Total cases required 63

By using the formula:

$$n = \frac{(z_\alpha + z_\beta)^2 2\ SD^2}{MD^2}$$

Where Z= Z statistic at a level of significance

MD= Anticipated mean difference

SD= Anticipated Standard deviation

Note: Due to COVID-19 pandemic and reduced patients in out-patient department, the total number of patients collected was 32.

METHOD OF COLLECTION OF DATA:

Patients of ages 6 years and abovehaving clinically diagnosed alopecia areata of the scalpwereenrolled for the study.

Inclusion criteria:

1. Patients of age group 6 years and above
2. Patients with ≥2 patches of hair loss over the scalp
3. Patients who have not taken any form of treatment in the last 6 months

Exclusion criteria:

1. Pregnant and lactating mothers

Methods:

Detailed history with respect to the onset and duration of alopecia and if any treatment was taken and pre-existing medical conditions were recorded.

Initial clinical examination of the patient was done to determine the site, size, and grade of alopecia. These findings were recorded in the proforma. Informed consent for the study was undertaken from all the patients.

Methodology:

Patients were two separate treatment modalities in two different patches of the same individual allotted randomly. One patch is selected and treated with intralesional triamcinolone acetonide (10mg/ml) (Patch A) while another patch with autologous PRP (Patch B). Four follow-ups were done with three weeks interval time. The remaining patches were treated with intralesional triamcinolone acetonide only and were not included in the study.

In Patch A, intralesional triamcinolone acetonide (10mg/ml) was injected intradermally using an insulin syringe at the patches, 1 cm apart and 0.1 ml into each site.

In Patch B, 10 ml of the patient's venous blood was collected in a sterile vaccutainer containing anticoagulant sodium citrate under strict aseptic conditions. This blood was centrifuged to separate the PRP by double spin centrifugation. It was initially centrifuged by 'light spin' centrifugation (1600 rpm for 10 minutes), leading to supernatant plasma, further centrifuged by 'heavy spin' centrifugation (3400 rpm for 7 minutes). Approximately three-fourths of the supernatant plasma was discarded, and the resultant buffy coat was used as PRP. PRP was given as intradermal injection at a rate of 0.05–0.1 ml/cm^2 1 cm apart.

Results were assessed and compared based on the trichoscopic findings and by Mac Donald Hull and Norris grading system[29] at baseline and each follow-up for 4 sittings done 3 weeks apart. The Mac Donald Hull and Norris grading system followed is as under:

Grade 1: Regrowth of vellus hair

Grade 2: Regrowth of sparse pigmented terminal hair

Grade 3: Regrowth of terminal hair in clusters

Grade 4: Complete regrowth of terminal hair over alopecia patch

Trichoscopic features were assessed on findings of black dots, yellow dots, exclamation mark hair, short vellus hair and terminal hair in the alopecia areata patches in at least one field of the lesion.

STATISTICAL ANALYSIS:

All characteristics were summarised descriptively. For continuous variables, the summary statistics of mean±standard deviation (SD) were used. For categorical data, the number and percentage were used in the data summaries and diagrammatic presentation. Chi-square (χ^2) test was used for association between two categorical variables.

The formula for the chi-square statistic used in the chi square test is:

$$\chi_c^2 = \sum \frac{(O_i - E_i)^2}{E_i}$$

The subscript "c" are the degrees of freedom. "O" is observed value and E is expected value. C= (number of rows-1)*(number of columns-1)

If the p-value was < 0.05, then the results were considered to be statistically significant otherwise it was considered as not statistically significant. Data were analysed using SPSS software v.23(IBM Statistics, Chicago, USA)and Microsoft office 2007.

ETHICAL CLEARANCE:

Institutional ethical committee clearance was undertaken for the study.

RESULTS

A hospital based prospective follow-up study was conducted from a period of November 2019 to May 2021. A total of 32 patients with alopecia areata were included in the study. Comparison was done on two separate patches of hair loss over the scalp on the same individual divided as:

- Patch A- Treated with intralesional triamcinolone acetonide
- Patch B- Treated with intralesional autologous PRP

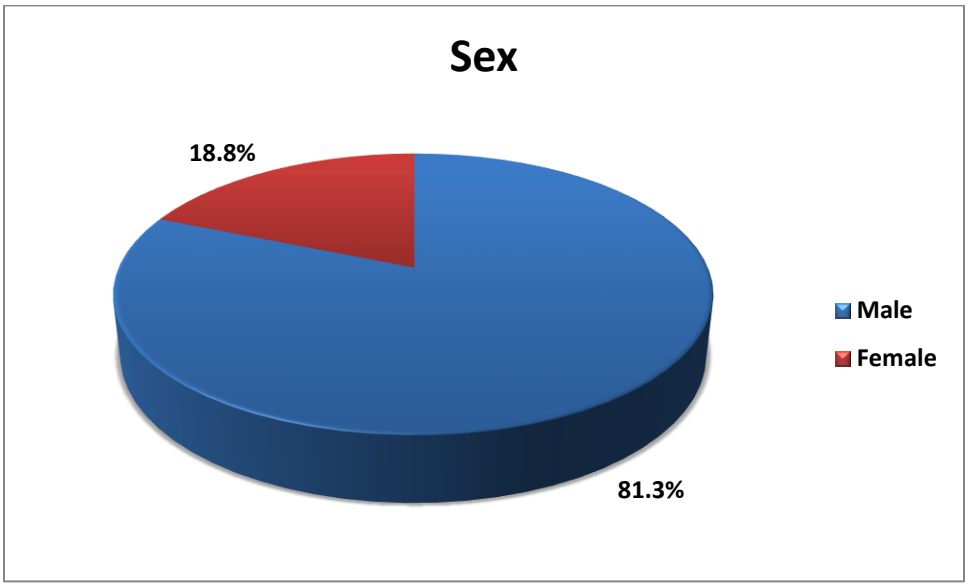
Gender distribution

Among 32 patients, 26 (81.3%) were males and 6 (18.8%) were females. Males outnumbered females in this study with a ratio of 4.3:1.

Table 1: Distribution of Cases according to Sex

Sex	N	Percent
Male	26	81.3
Female	6	18.8
Total	32	100

Figure 1: Distribution of Cases according to Sex



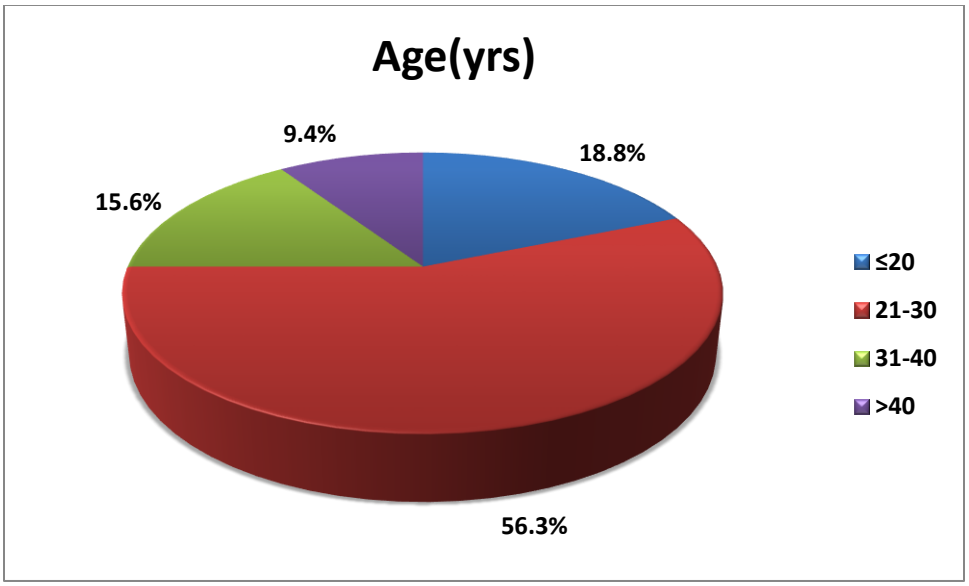
Age group

The age group of the patients included in the study ranged from 15 years to 45 years. Most of the patients belonged to the third decade (18 patients- 56.3%).

Table 2: Distribution of Cases according to Age

Age(yrs)	N	Percent
≤20	6	18.8
21-30	18	56.3
31-40	5	15.6
>40	3	9.4
Total	32	100

Figure 2: Distribution of Cases according to Age



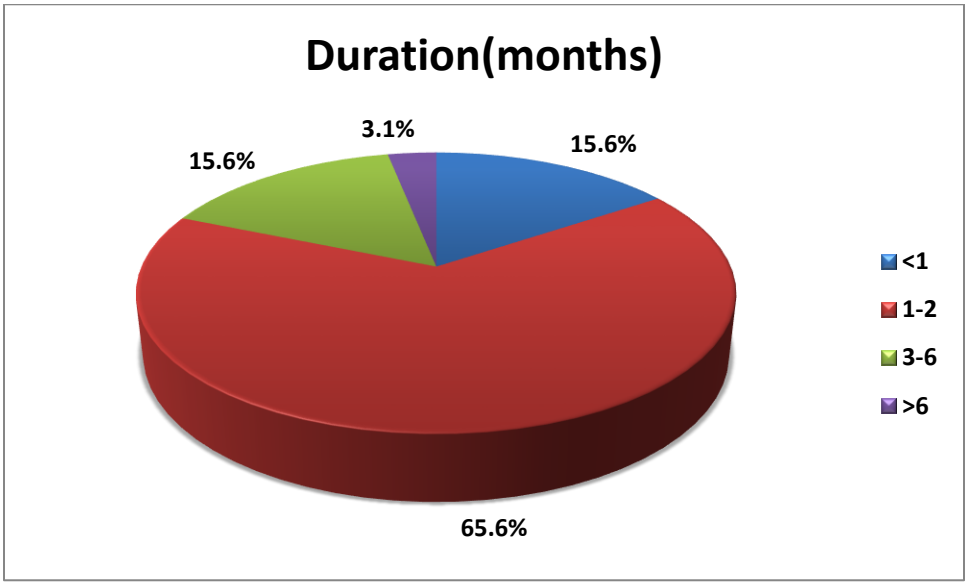
Duration of illness

In this study, duration of illness ranged from 2 weeks-6 months, with one of patients having duration of 2 years. Most of the patients (21 patients- 65.6%) had disease duration of 1-2 months.

Table 3: Distribution of Duration between Study Groups

Duration(months)	N	Percent
<1	5	15.6
1-2	21	65.6
3-6	5	15.6
>6	1	3.1
Total	32	100

Figure 3: Distribution of Duration between Study Groups



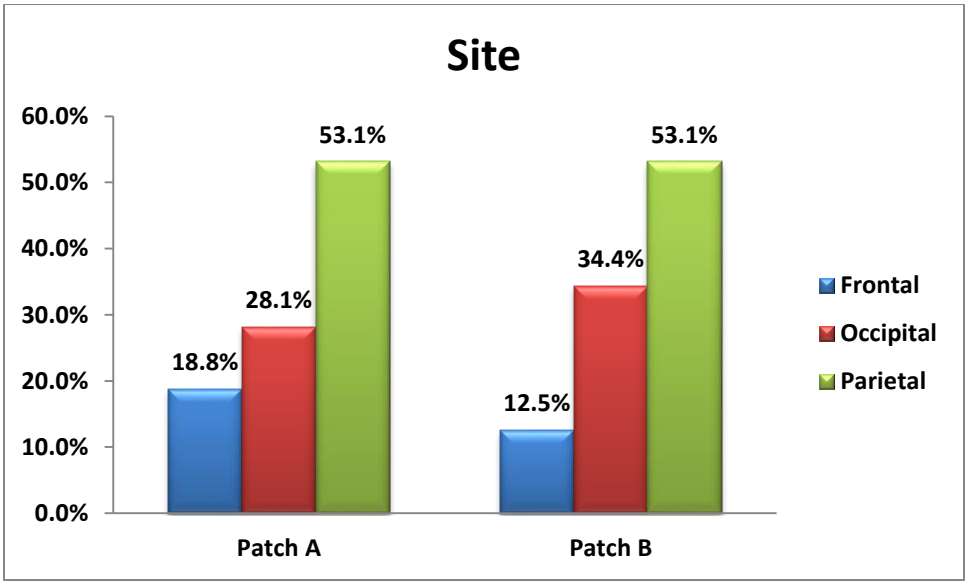
Site involved

Parietal area of the scalp was the most common site involved in this study, seen in 17 (53.1%) of the patients in both the patches, followed by occipital area in 9 (28.1%) patients of patch A and 11 (34.4%) patients of patch B. The difference between both the groups was not statistically significant.

Table 4: Distribution of Site between Study Groups

Site	Patch A		Patch B		p value
	N	%	N	%	
Frontal	6	18.8	4	12.5	0.741
Occipital	9	28.1	11	34.4	
Parietal	17	53.1	17	53.1	
Total	32	100	32	100	

Figure 4: Distribution of Site between Study Groups



Response in both patches according to the Mac Donald Hull and Norris grade

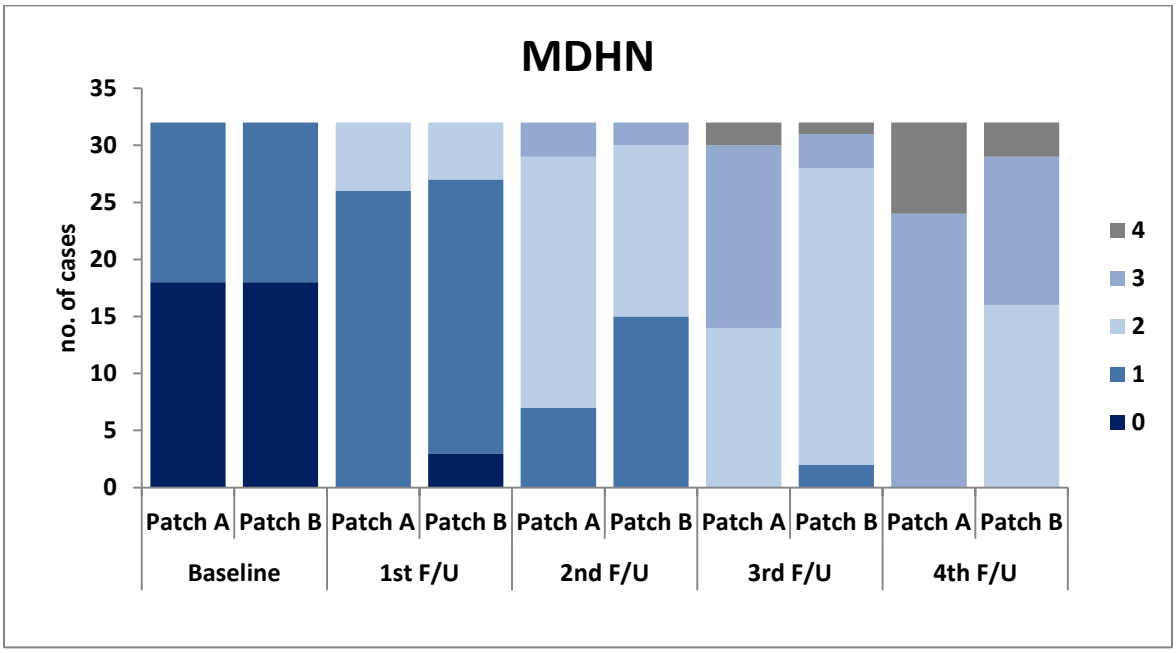
18 (56.3%) patients were in grade 0 and 14 (43.8%) patients in grade 1 at baseline in patch A. At 12 weeks, 24 (75%) patients were in grade 3 and 8 (25%) patients were in grade 4. In patch B, 18 (56.3%) patients were in grade 0 and 14 (43.8%) were in grade 1 at baseline, and at 12 weeks, 16 (50%) patients were in grade 2, 13 (40.6%) were in grade 3 and 3 (9.4%) patients in grade 4. A faster improvement in the Mac Donald Hull and Norris grade was seen in patch A when compared to patch B. The difference was found to be statistically insignificant in both the patches at 1st and 2nd follow-up while it was significant in the 3rd and 4th follow-up.

Table 5: Distribution of MDHN between Study Groups and follow-up

MDHN	Baseline				1st F/U				2nd F/U				3rd F/U				4th F/U			
	Patch A		Patch B		Patch A		Patch B		Patch A		Patch B		Patch A		Patch B		Patch A		Patch B	
	N		N		N		N		N		N		N		N		N		N	
0	18	56.3	18	56.3	0	0.0	3	9.4	0	0.0	0	0.0	0	0.0	0	0.0	0	0.0	0	0.0
1	14	43.8	14	43.8	26	81.3	24	75.0	7	21.9	15	46.9	0	0.0	2	6.3	0	0.0	0	0.0
2	0	0.0	0	0.0	6	18.8	5	15.6	22	68.8	15	46.9	14	43.8	26	81.3	0	0.0	16	50.0
3	0	0.0	0	0.0	0	0.0	0	0.0	3	9.4	2	6.3	16	50.0	3	9.4	24	75.0	13	40.6
4	0	0.0	0	0.0	0	0.0	0	0.0	0	0.0	0	0.0	2	6.3	1	3.1	8	25.0	3	9.4
Total	32	100	32	100	32	100	32	100	32	100	32	100	32	100	32	100	32	100	32	100
p value	-				0.205				0.109				<0.001*				<0.001*			

Note: p value* significant at 5 level of significance (p<0.05)

Figure 5: Distribution of MDHN between Study Groups and follow-up



Trichoscopy changes in both patches

In patch A, 23 (71.9%) patients had black dots, 20 (62.5%) had yellow dots, 17 (53.1%) had exclamation hair, 18 (56.3%) had short vellus hair and none had terminal hair at baseline. At the end of 12 weeks, 20 (62.5%) patients showed black dots, 9 (28.1%) patients each had yellow dots and exclamation hair and all patients showed short vellus and terminal hair.

In patch B, black dots was seen in 22 (68.8%) patients, yellow dots, exclamation hair and short vellus hair was seen in 19 (59.4%) patients and terminal hair was not seen in any patient. At 12 weeks, black dots was seen in 23 (71.9%) patients, yellow dots was seen in 7 (21.9%) patients, exclamation hair in 9 (28.1%) patients and all had short vellus and terminal hair.

The p values were not statistically significant when compared with the 2 patches at baseline and at each follow-up. Between baseline and at 12 weeks, p values were significant for exclamation hair for patch B and for short vellus hair in both the patches.

Table 6: Trichoscopy findings between Study Groups and follow-up (A) values in both the patches, (B) p values between patch A and patch B, and (C) p values between baseline and 4th follow-up

Table 6A:

Trichoscopy	Baseline				1st F/U				2nd F/U				3rd F/U				4th F/U			
	Patch A		Patch B		Patch A		Patch B		Patch A		Patch B		Patch A		Patch B		Patch A		Patch B	
	N		N		N		N		N		N		N		N		N		N	
Black dots	23	71.9	22	68.8	25	78.1	25	78.1	22	68.8	22	68.8	20	62.5	21	65.6	20	62.5	23	71.9
Yellow dots	20	62.5	19	59.4	18	56.3	23	71.9	16	50.0	12	37.5	10	31.3	10	31.3	9	28.1	7	21.9
Exclamation hair	17	53.1	19	59.4	19	59.4	16	50.0	15	46.9	15	46.9	13	40.6	15	46.9	9	28.1	9	28.1
Short vellus hair	18	56.3	19	59.4	32	100	30	93.8	32	100	32	100	32	100	32	100	32	100	32	100
Terminal hair	0	0.0	0	0.0	6	18.8	5	15.6	25	78.1	20	62.5	32	100	31	96.9	32	100	32	100
Total	32	100	32	100	32	100	32	100	32	100	32	100	32	100	32	100	32	100	32	100

Table 6B:

	p values between Patch A & Patch B				
Trichoscopy	**Baseline**	**1st F/U**	**2nd F/U**	**3rd F/U**	**4th F/U**
Black dots	0.784	-	-	0.794	0.424
Yellow dots	0.798	0.193	0.313	-	0.564
Exclamation hair	0.614	0.451	1.000	0.614	-
Short vellus hair	0.800	0.151	-	-	-
Terminal hair	-	0.740	0.171	0.313	-

Table 6C:

Trichoscopy	**p values between baseline and 4th F/U**	
	Patch A	**Patch B**
Black dots	0.414	0.813
Yellow dots	0.090	0.096
Exclamation hair	0.073	0.016*
Short vellus hair	<0.001*	<0.001*
Terminal hair	0.414	0.813

Note: p value* significant at 5 level of significance ($p<0.05$)

Figure 6: Trichoscopy findings between Study Groups and follow-up for (A) black dots, (B) yellow dots, (C) exclamation hair, (D) short vellus hair, and (E) terminal hair

Figure 6A:

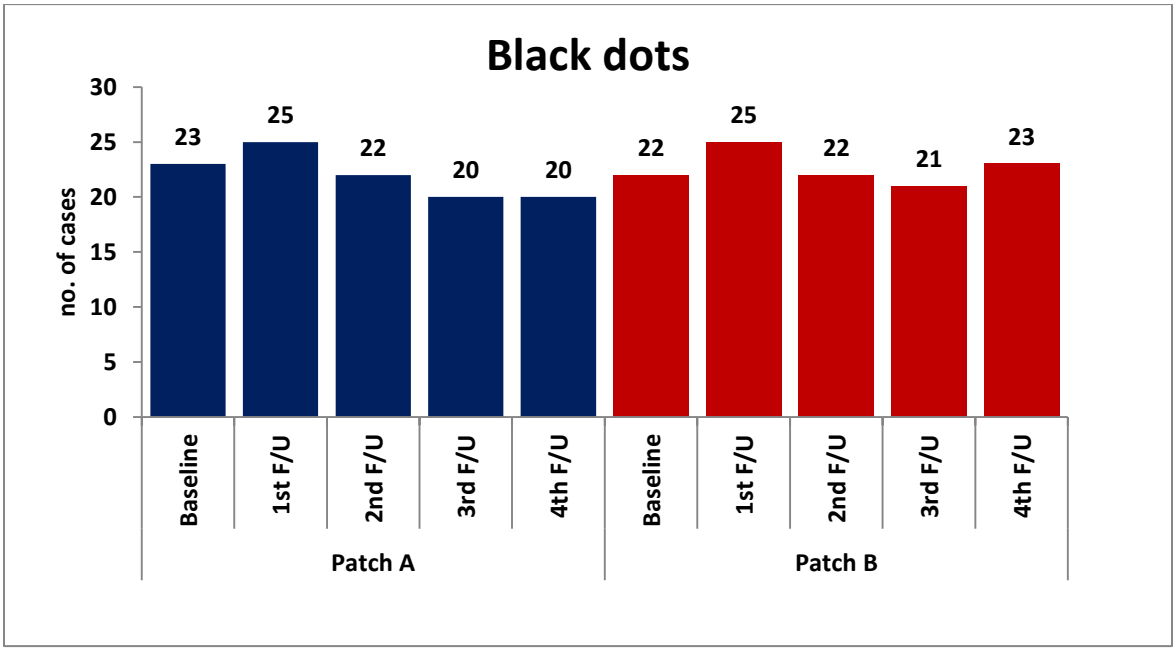


Figure 6B:

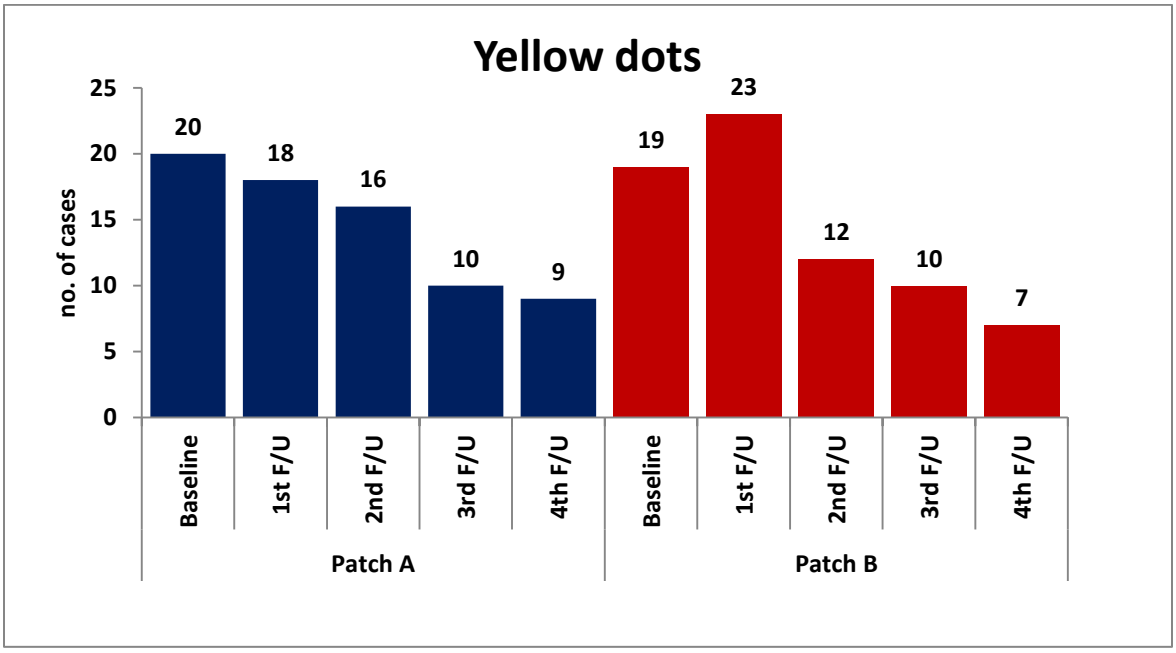


Figure 6C:

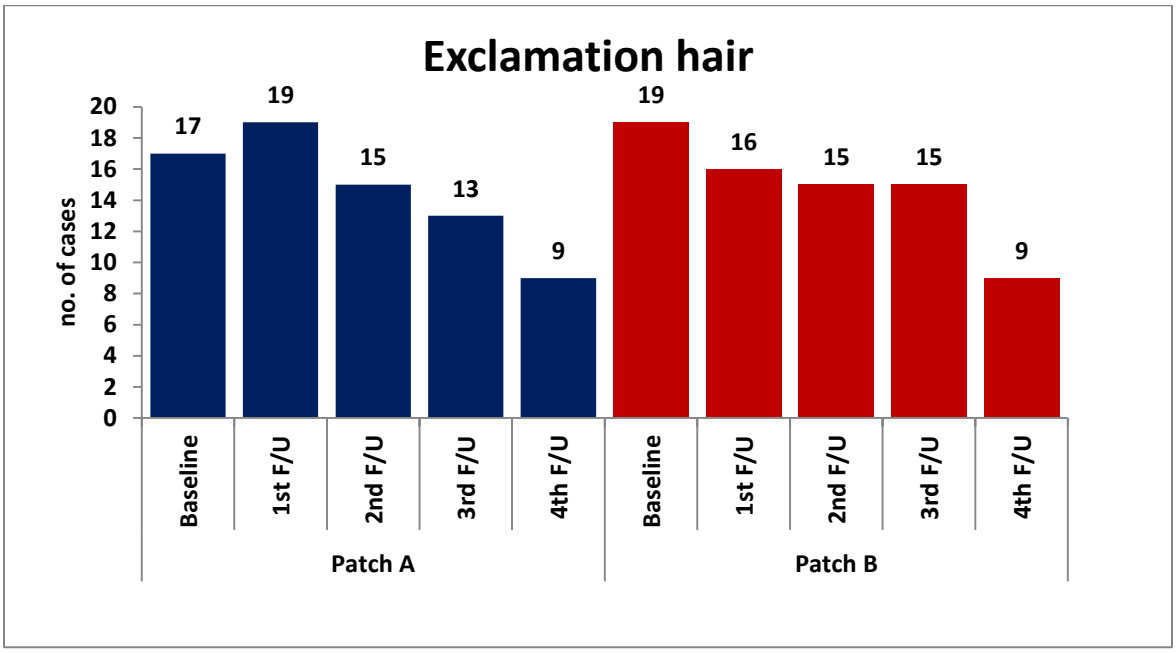


Figure 6D:

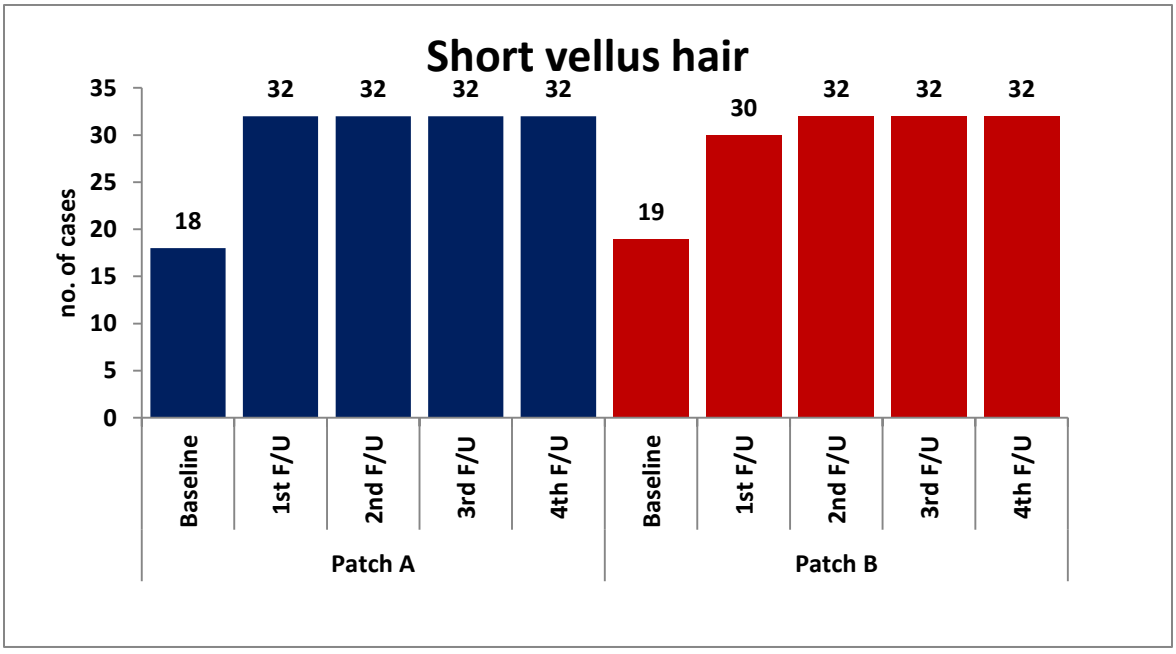
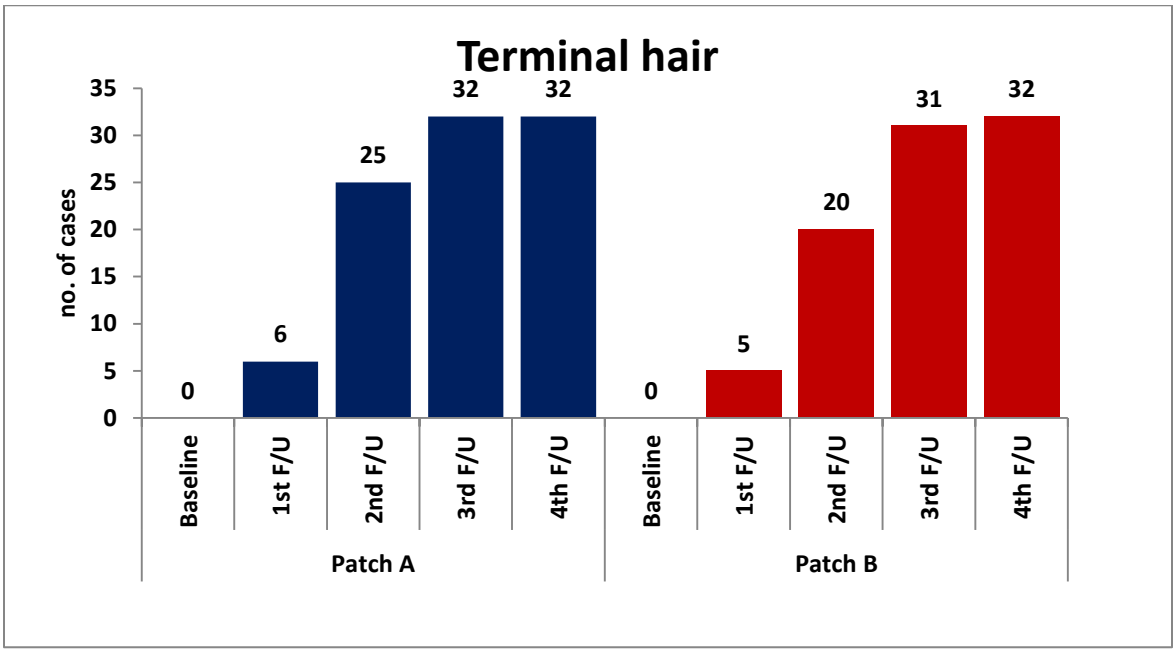


Figure 6E:



Adverse effects and recurrences

No adverse effects or recurrences were seen in both patches.

Figure 7: Clinical picture of Patch A: (A) at baseline (MDHN grade- 0) (B) at 12 weeks (MDHN grade- 4)

Figure 7A:

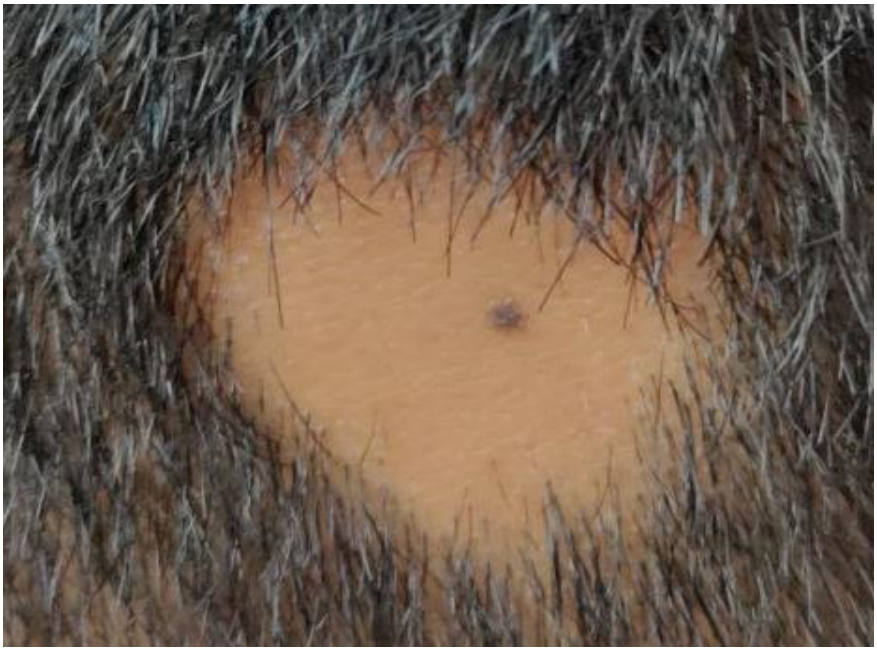


Figure 7B:

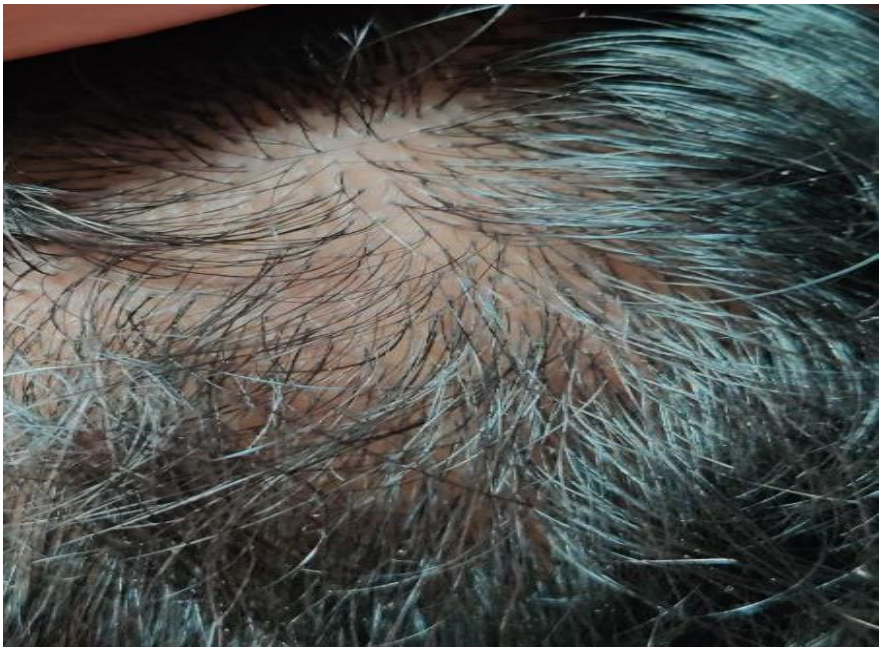


Figure 8: Trichoscopy picture of patch A (A) at baseline (B) at 12 weeks showing black dots (black arrow), yellow dots (orange arrow), exclamation mark hair (purple arrow), short vellus hair (red arrow) and terminal hair (blue arrow)

Figure 8A:

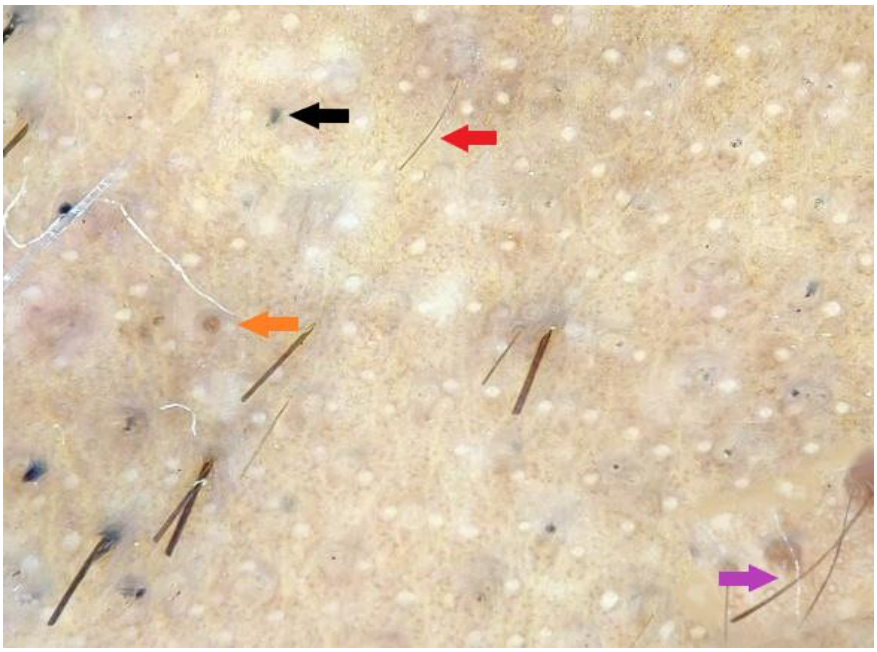


Figure 8B:

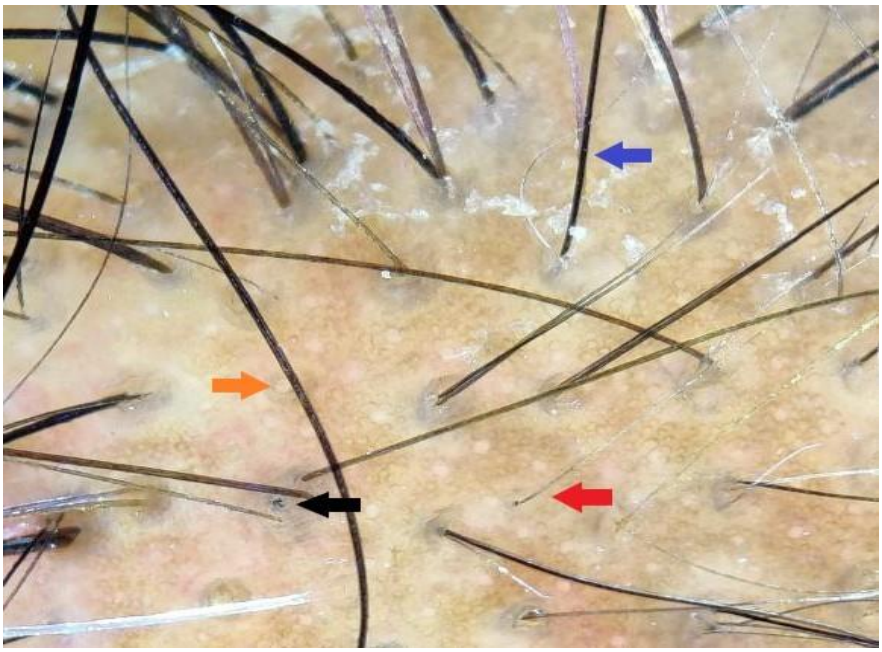


Figure 9: Clinical picture of patch B (A) at baseline (MDHN grade-0) (B) at 12 weeks (MDHN grade- 3)

Figure 9A:

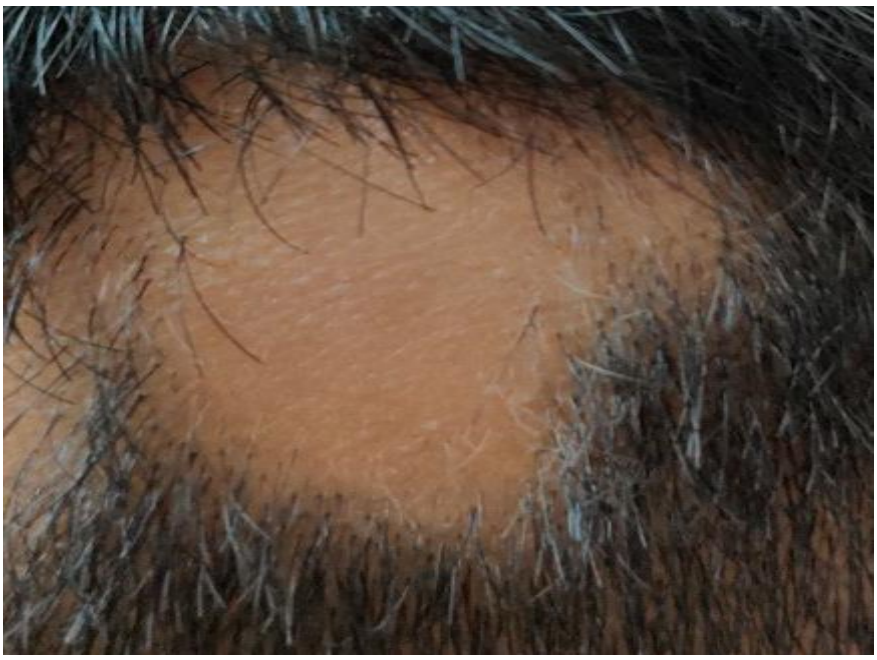


Figure 9B:

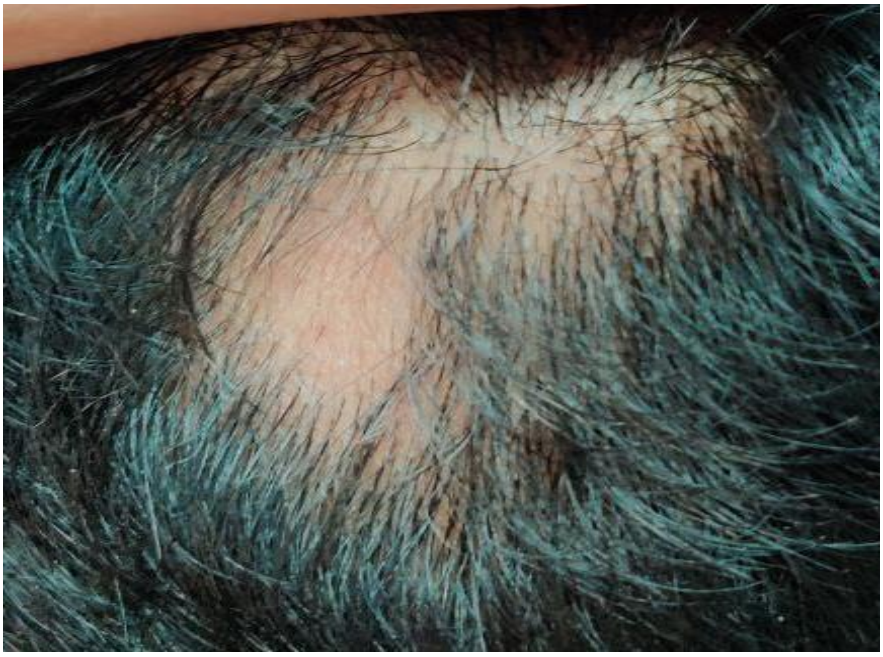


Figure 10: Trichoscopy picture of patch B (A) at baseline (B) at 12 weeks showing black dots (black arrow), yellow dots (orange arrow), exclamation mark hair (purple arrow), short vellus hair (red arrow) and terminal hair (blue arrow)

Figure 10A:

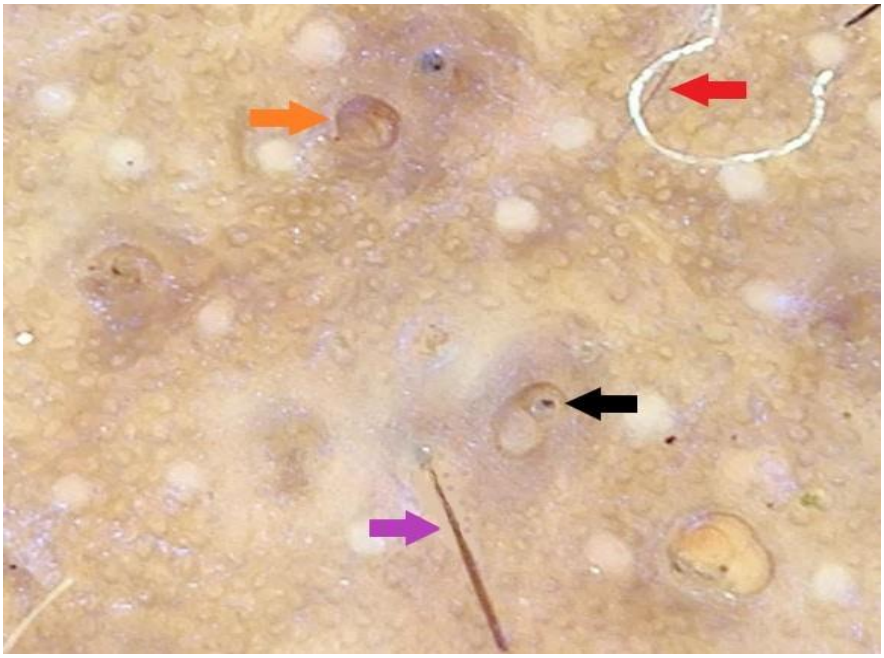
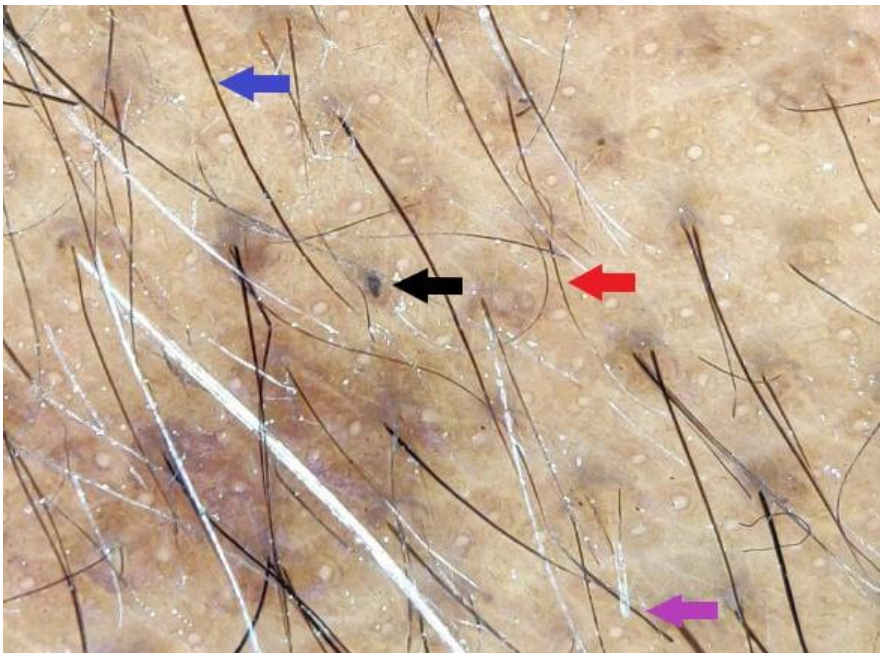


Figure 10B:



DISCUSSION

Alopecia areata is a frequently encountered form of hair loss implicated in 25% of all alopecia cases.[2] It is a non-scarring alopecia causing hair loss over scalp and body.[1] It is characterised by a well defined patch of hair loss which can show progression to alopecia totalis or universalis. Management is mainly aimed at controlling the pathology.[1,6]

Trichoscopy is a non-invasive, simple, bed side technique which helps in diagnosing various hair and scalp disorders.[7] In alopecia areata, the characteristic features are black dots and exclamation hair, yellow dots and regrowing vellus hair.[19]

Intra-lesional corticosteroids are the most commonly used modality of treatment owing to its immunosuppressive action by suppressing the T-cell mediated attack on the hair follicle. The preferred intralesional preparation is triamcinolone acetonide because of fewer side effects like atrophy.[11]

Platelet rich plasma is an autologous preparation of platelets in concentrated plasma. Various growth factors released exert an anti-apoptotic effect on the stem cells, prolong anagen phase of the hair cycle and stimulate the formation of new follicles and hair growth, along with its anti-inflammatory effect.[3,12] Platelet rich plasma has recently attracted attention for the treatment of alopecia areata owing to its simplicity and efficacy without the risk of allergic reactions or atrophy.[1]

This study was undertaken to find out the efficacy of intralesional autologous platelet rich plasma as compared to intralesional triamcinolone acetonide in the treatment of alopecia areata, along with trichoscopic changes in response to treatment.

Out of 32 patients included in this study, 26 (81.3%) of the patients were males while 6 (18.8%) were females. The male to female ratio was 4.3:1. Out of 30 patients in study conducted by Ranpariya *et al.*[1], male preponderance was seen with a male to female ratio of 3:1. Another study by Shumez *et al.*[9] showed similar results, with a male to female ratio of 4:1.These studies were conducted in Indian population. Certain older studies by Bhat *et al.*[30] and Sharma *et al.*[31] have shown more number of female patients affected (male to female ratio of 1:2 in both studies).As a result, there is a trend towards male predominance of alopecia areata in India.

The age group of the patients in this study ranged from 15 years to 45 years, with a mean of 27.1 years, with a predominance of patients in the age group of 21-30 years. Other studies done by Ranpariya *et al.*[1], Balakrishnan *et al.*[24], Shumez *et al.*[9] and Agrawal *et al.*[10] showed majority of the patients belonged to the third decade.

Duration of disease in this study was 2 weeks-6 months with one patient having disease duration of 2 years. 65.6% of the patients had disease duration of 1-2 months, indicating an acute onset. Studies by Shumez *et al.*[9] and Agrawal *et al.*[10] have also shown majority of the patients having a disease duration of 1-6 months.

Based on the site involved, parietal area of the scalp was most involved (53.1% of the patients in both the patches), followed by occipital area (28.1% of the patients in patch A and 34.4% of the patients in patch B) in this study. Shumez *et al.*[9] showed that occiput was the most common area involved over the scalp while vertex was the most common site in study by Balakrishnan *et al.*[24]

Based on the Mac Donald Hull and Norris grading system, 18 (56.3%) patients were in grade 0 and 14 (43.8%) patients in grade 1 at baseline in patch A. At 12 weeks, 24 (75%) patients were in grade 3 and 8 (25%) patients were in grade 4. In patch B, 18 (56.3%) patients

were in grade 0 and 14 (43.8%) were in grade 1 at baseline, and at 12 weeks, 16 (50%) patients were in grade 2, 13 (40.6%) were in grade 3 and 3 (9.4%) patients in grade 4. A faster increase in the score was seen in patch A than patch B. Therefore, the growth of hair was better following treatment with intralesional triamcinolone acetonide than with PRP.

Similar findings were seen in a study conducted by Ranpariya *et al.*[1] which showed a higher mean score according to the Mac Donald Hull and Norris grade with limited side effects in patches treated with intralesional triamcinolone. Also, patches treated with PRP showed recurrence in 3 patients. Other studies by Agrawal *et al.*[10] and Chang *et al.*[25] also showed similar results.

Balakrishnan *et al.*[24], Trink *et al.*[26] and Shumez *et al.*[9] also conducted similar studies which concluded that PRP was a more effective and safe treatment option when compared to intralesional triamcinolone, but the findings in these studies was found to be not statistically significant.

On trichoscopy, in patch A, 23 (71.9%) patients had black dots, 20 (62.5%) had yellow dots, 17 (53.1%) had exclamation hair, 18 (56.3%) had short vellus hair and none had terminal hair at baseline. At the end of 12 weeks, 20 (62.5%) patients showed black dots, 9 (28.1%) had yellow dots and exclamation hair and all patients had short vellus and terminal hair. Therefore, there was a reduction in the number of patients with black dots, yellow dots and exclamation hair and increase in those having short vellus hair and terminal hair in patches treated with triamcinolone. The variation in short vellus hair from baseline to 12 weeks was statistically significant, while the other values did not show a statistical significance.

Study by Agrawal *et al.*[10] also showed 78.6% of the patients treated with triamcinolone showed no dystrophic hair at the end of the study as compared to 69.2% of the patients receiving

PRP. An increase in the number of vellus hairs while reduction in exclamation mark hairs, broken hairs and black dots was seen following treatment with intralesional triamcinolone over a period of 6 weeks in a study by Ganjoo *et al.*[27] The number of patients with yellow dots remained unchanged.

In patch B, black dots was seen in 22 (68.8%) patients, yellow dots, exclamation hair and short vellus hair was seen in 19 (59.4%) patients each and terminal hair was not seen in any patient. At 12 weeks, black dots was seen in 23 (71.9%) patients, yellow dots was seen in 7 (21.9%) patients, exclamation hair in 9 (28.1%) patients and all had short vellus and terminal hair. Thus, an increase in the number of patients with black dots, short vellus hair and terminal hair was seen while a reduction in patients with yellow dots and exclamation hair was seen following treatment with PRP. The increase in patients with short vellus hair and reduction in exclamation hair only showed statistically significant values.

Following 3 months of treatment with intralesional PRP in a study by El Taieb *et al.*[28] on alopecia areata patches, short vellus hairs and yellow dots were found to be reduced, and fully pigmented hairs were more. A study by Inui *et al.*[23] showed a persistence of black dots in patients with improving disease, which was also seen in this study.

No adverse effects or recurrances were reported following either treatment modality.

Thus, this study shows that intralesional triamcinolone acetonide is a better and safe treatment option when compared with intralesional autologous PRP for the treatment of alopecia areata of the scalp. On trichoscopy, exclamation hair and yellow dots are markers for disease activity and are reduced following treatment. Increase in the number of short vellus hair and terminal hair indicate response to treatment. Black dots are also seen in the acute stage of the

disease, but did not show a significant change following treatment in this study, which may indicate a persistence of disease activity. Therefore, although trichoscopy indicates a response to treatment and control of disease activity in both the treatment modalities, the regrowth of hair following triamcinolone was better.

CONCLUSION

This study was conducted to compare the efficacy of intralesional triamcinolone acetonide versus autologous PRP in the treatment of alopecia areata of the scalp along with the trichoscopic findings. A total of 32 patients were included. Male preponderance was seen in this study with a male to female ratio of 4.3:1. The age of the patients included in this study ranged from 15 years to 45 years, with a predominance of patients in the third decade. Duration of disease was 2 weeks-2 years, with most of the patients having disease duration of 1-2 months. Parietal area was the most common site of involvement.

At 12 weeks, following treatment with triamcinolone, 24 (75%) of the patients were in grade 3 and 8 (25%) of the patients were in grade 4, i.e., complete regrowth of hair. In patches treated with PRP, 16 (50%) were in grade 2, 13 (40.6%) were in grade 3 and 3 (9.4%) of the patients were in grade 4. The values showed statistical significance at the 3rd and 4th follow-up. Thus, more number of patches treated with triamcinolone had complete regrowth of hair when compared to those treated with PRP and no adverse effects or recurrence was reported following either treatment modality.

In response to both the treatment modalities, there is a significant increase in short vellus hair and terminal hair while a reduction in yellow dots and exclamation hair is seen on trichoscopy, which indicates response to treatment and reduction of disease activity in both the groups, but the only values that showed statistical significant were reduction in exclamation hair following treatment with PRP and increase in short vellus hair following treatment in both the patches. Black dots did not show a significant change from baseline in both the treatment groups.

Therefore, intralesional triamcinolone is more effective than PRP and is a safe treatment modality for alopecia areata.

SUMMARY

A hospital based prospective follow-up study was conducted from a period of November 2019 to May 2021 to compare the efficacy of intralesional triamcinolone acetonide versus autologous platelet-rich plasma in the treatment of alopecia areata of the scalp in two different patches of hair loss in the same individual. A total of 32 patients were included in this study. The patches of hair loss were allotted into 2 groups-

- Patch A- Treated with intralesional triamcinolone acetonide
- Patch B- Treated with intralesional PRP

The injections were administered at 3 weeks intervals for 4 sittings and response was recorded by the MacDonald Hull and Norris grading system and trichoscopy at baseline and each follow up. The following observations were made:

- Male patients were predominant in this study.
- Age of the patients ranged from 15-45 years, with most of the patients belonging to the third decade.
- Duration of the disease was 2 weeks-2 years. Most of the patients had duration of 1-2 months.
- Parietal area of the scalp was the most common site involved, followed by occiput.
- Assessment based on Mac Donald Hull and Norris grading system showed patch A having a significant hair growth compared to patch B at the end of 12 weeks, with more number of patients in patch A having complete regrowth of hair.

- Trichoscopy showed a reduction in yellow dots and exclamation hair and an increase in short vellus hair and terminal hair in both the patches at 12 weeks. Black dots did not show a significant change following treatment.
- No complications or recurrances were seen in both the patches.

This study shows that intralesional triamcinolone acetonide is a better and safe treatment option when compared with intralesional autologous PRP for the treatment of alopecia areata of the scalp. Although regrowth of hair with triamcinolone was better, on trichoscopy, there is a reduction in yellow dots and exclamation hair and an increase in short vellus hair and terminal hair in alopecia areata following treatment with both modalities, which indicates a good response to treatment in both the groups. Black dots did not show a significant change following treatment, which may indicate persistence of disease activity.

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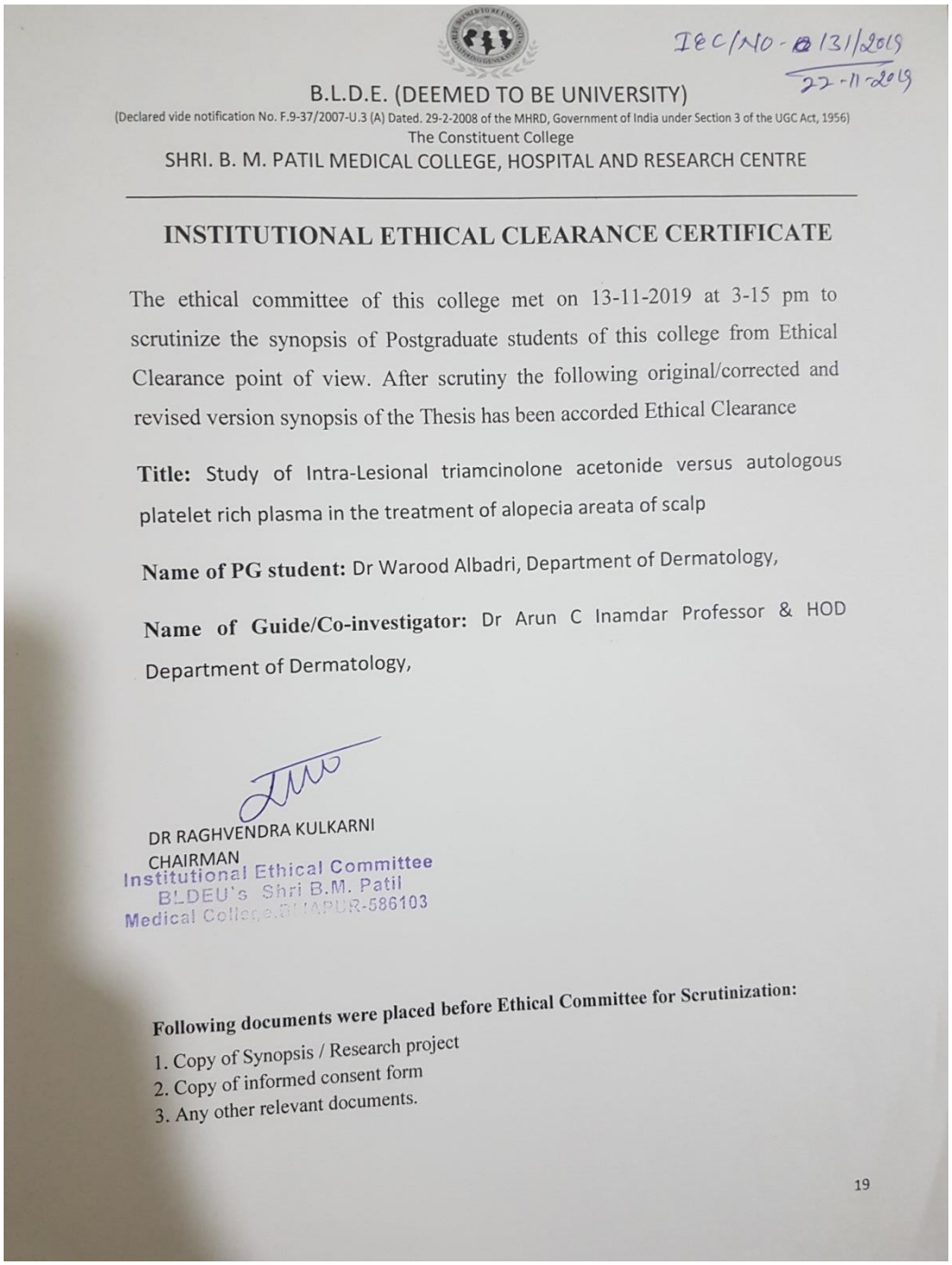
ETHICAL CLEARANCE CERTIFICATE



IEC/NO-131/2019
22-11-2019

B.L.D.E. (DEEMED TO BE UNIVERSITY)
(Declared vide notification No. F.9-37/2007-U.3 (A) Dated. 29-2-2008 of the MHRD, Government of India under Section 3 of the UGC Act, 1956)
The Constituent College
SHRI. B. M. PATIL MEDICAL COLLEGE, HOSPITAL AND RESEARCH CENTRE

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The ethical committee of this college met on 13-11-2019 at 3-15 pm to scrutinize the synopsis of Postgraduate students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected and revised version synopsis of the Thesis has been accorded Ethical Clearance

Title: Study of Intra-Lesional triamcinolone acetonide versus autologous platelet rich plasma in the treatment of alopecia areata of scalp

Name of PG student: Dr Warood Albadri, Department of Dermatology,

Name of Guide/Co-investigator: Dr Arun C Inamdar Professor & HOD Department of Dermatology,

DR RAGHVENDRA KULKARNI
CHAIRMAN
Institutional Ethical Committee
BLDEU's Shri B.M. Patil
Medical College, BIJAPUR-586103

Following documents were placed before Ethical Committee for Scrutinization:

1. Copy of Synopsis / Research project
2. Copy of informed consent form
3. Any other relevant documents.

19

CONSENT FORM

B.L.D.E. (Deemed to be University) SHRI B.M PATIL

MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE,

VIJAYAPURA-586 103

INFORMED CONSENT FOR PARTICIPATION IN DISSERTATION/RESEARCH

TITLE OF THE PROJECT:- STUDY OF INTRA-LESIONAL TRIAMCINOLONE ACETONIDE VERSUS AUTOLOGOUS PLATELET RICH PLASMA IN THE TREATMENT OF ALOPECIA AREATA OF SCALP

PG GUIDE **:-** DR ARUN C.INAMADAR

PG STUDENT **:-** DR WAROOD ALBADRI

PURPOSE OF RESEARCH:-

I have been informed that this project will comparethe efficacy of intralesional injection of autologous platelet rich plasma with intralesional injection of triamcinolone acetonide in the treatment for alopecia areata among patients attending skin OPD at SBMP Medical College and Hospital

BENEFITS:-

I understand that my participation in this study will help the investigator to know the efficacy of intralesional injection of autologous platelet rich plasma versus intralesional injection of triamcinolone acetonide in the treatment for alopecia areata among patients attending skin OPD at SBMP Medical College and Hospital.

PROCEDURE:-

I understand that relevant history will be taken and I will undergo detailed clinical examination after which treatment will be given.

RISK AND DISCOMFORTS:-

I understand the possible complications that may occur during and after the procedure, i.e., post procedure pain, swelling, erythema and atrophy at the site of injection.

CONFIDENTIALITY:-

I understand that medical information produced by this study will become a part of my hospital records and will be subjected to the confidentiality and privacy regulation of the said hospital. Information of a sensitive personal nature will not be a part of the medical records, but will be stored in the investigator's research file.

If the data are used for publication in the medical literature or for teaching purposes no names will be used and other identifiers such as photographs and audio or videotapes will be used only with my special written permission. I understand I may see the photographs, videotapes and hear the audiotapes before giving this permission.

REQUEST FOR MORE INFORMATION:-

I understand that I may ask more questions about the study at any time concerned. DrWaroodAlbadri is available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during the course of this study, which may influence my continued participation.

REFUSAL OR WITHDRAWAL OF PARTICIPATION:-

I understand that my participation is voluntary and I may refuse to participate or may withdraw consent and discontinue participation in this study at any time without prejudice. I also understand that DrWaroodAlbadri may terminate my participation in this study at any time after she has explained the reasons for doing so and has helped arrange for my continued care by my own physician, if this is appropriate.

INJURY STATEMENT:-

I understand that in the unlikely event of injury to me resulting directly from my participation in this study and if such injury were reported promptly, then medical treatment will be available to me, but no further compensation will be provided. I understand that by my agreement for my participation in this study, I am not waiving any of my legal rights.

I have explained to _____ the purpose of the research, the procedures required, and the possible risks and benefits to the best of my ability in patient's own language.

_____ _____

Investigator / P. G. Guide Date

I confirm that …….(Name of the PG guide/chief researcher) has explained to me the research, the study procedures that I undergo and the possible risks and discomforts as well as benefits that I may experience. I have read and I understand this consent form. Therefore, I agree to give my consent for my participation as a subject in this research project.

_____ _____

Participant / guardian Date

_____ _____

Witness to signature Date

PROFORMA

B.L.D.E. (Deemed to be University)

SHRI B. M. PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE,

VIJAYAPURA.

DEPARTMENT OF DERMATOLOGY,VENEREOLOGY AND LEPROSY

SCHEME OF CASE TAKING

STUDY OF INTRA-LESIONAL TRIAMCINOLONE ACETONIDE VERSUS AUTOLOGOUS PLATELET RICH PLASMA IN THE TREATMENT OF ALOPECIA AREATA OF SCALP

Serial no :

Name : Sex :

Age : OPD No:

Address :

Occupation :

Socio economic status:

HISTORY:

Present history:

Duration of the illness:

Past history :

History of Prior Treatment Taken:

Family History:

Similar history in family members:

Diabetes mellitus:

Bronchial asthma/HIV/TB:

Others:

EXAMINATION:

Height :

Body Weight :

Pallor :

Nutritional status: well nourished / poorly nourished:

Cutaneous examination:

Site of the lesion and number:

- Number of lesions:
- Measurement of lesion:

  Length =

  Width =

- Grading :

	Patch A	Patch B
Baseline		
1st follow up		
2nd follow up		
3rd follow up		
4th follow up		

- Trichoscopy

Patch A	Black dots	Yellow dots	Exclamation hair	Short vellus hair	Terminal hair
Baseline					
1st follow up					
2nd follow up					
3rd follow up					
4th follow up					

Patch B	Black dots	Yellow dots	Exclamation hair	Short vellus hair	Terminal hair
Baseline					
1st follow up					
2nd follow up					
3rd follow up					
4th follow up					

KEY TO MASTERCHART

MDHN – Mac Donald Hull and Norris grade

A – Absent

P – Present

BD – Black dots

SVH – Short vellus hair

YD – Yellow dots

EH – Exclamation hair

TH – Terminal hair

MASTERCHART

Sr No	Age	Sex	Duration	Patch A																																			
				Site	Size		Baseline						1st F/U						2nd F/U						3rd F/U						4th F/U									
					Length (cm)	Width (cm)	MDHN	Trichoscopy					MDHN	Trichoscopy					MDHN	Trichoscopy					MDHN	Trichoscopy					MDHN	Trichoscopy								
								BD	SVH	YD	EH	TH		BD	SVH	YD	EH	TH		BD	SVH	YD	EH	TH		BD	SVH	YD	EH	TH		BD	SVH	YD	EH	TH			
1	15Y	M	2 months	Parietal	3	3.2	0	A	P	P	A	A	1	A	P	P	P	A	2	P	P	P	P	P	2	P	P	A	P	P	3	P	P	A	P	P			
2	31Y	M	1 week	Parietal	1.5	2	1	P	P	A	A	A	1	P	P	A	A	A	2	P	P	A	A	P	3	P	P	A	P	P	3	P	P	P	A	P			
3	15Y	F	6 months	Occipital	4	2	0	A	A	P	P	A	1	A	P	P	P	A	2	P	P	P	P	P	3	A	P	P	A	P	4	A	P	A	A	P			
4	2	M	1	Fr	2.	2	0	P	P	P	A	A	1	P	P	P	P	A	2	P	P	P	P	P	3	P	P	A	P	P	3	P	P	A	P	P			

	8 Y		month	ontal	5																																
5	24 Y	M	2 months	Occipital	3	5	1	P	P	A	P	A	1	P	P	A	A	A	2	P	P	A	A	P	4	P	P	A	A	P	4	P	P	P	A	P
6	17 Y	M	1 month	Parietal	1	1	1	P	P	P	P	A	2	P	P	P	P	P	3	A	P	P	P	P	4	P	P	P	A	P	4	A	P	A	A	P
7	30 Y	F	1 month	Frontal	2	2.5	0	A	A	P	P	A	1	P	P	P	P	A	2	P	P	P	A	P	3	P	P	A	P	P	3	P	P	P	P	P
8	39 Y	M	2 months	Occipital	2	2	0	P	P	A	A	A	1	P	P	A	P	A	1	P	P	A	P	A	2	P	P	A	P	P	3	P	P	A	P	P
9	18 Y	M	1 week	Parietal	1	2	1	A	P	P	P	A	2	A	P	P	P	P	3	P	P	P	P	P	3	P	P	P	A	P	3	P	P	A	A	P
10	34 Y	M	6 months	Parietal	1.5	1.5	1	A	P	P	P	A	1	P	P	A	P	A	2	A	P	A	P	P	2	P	P	P	P	P	3	P	P	P	A	P
11	27 Y	M	6 months	Parietal	2.5	2	0	P	A	P	P	A	1	P	P	P	P	A	1	P	P	A	A	A	2	A	P	A	P	P	3	A	P	P	A	P
12	21 Y	M	2 year	Occipit	2	2	0	P	P	A	A	A	1	P	P	A	P	A	1	A	P	A	P	A	2	P	P	A	A	P	3	P	P	A	P	P

			s	al																																	
13	28Y	M	5 months	Frontal	1.5	2	0	P	A	P	A	A	1	P	P	P	P	A	1	P	P	P	A	A	2	P	P	P	P	P	3	P	P	A	P	P
14	42Y	M	1 month	Occipital	1.5	1	1	P	P	A	P	A	1	P	P	A	A	A	2	P	P	A	P	P	2	P	P	A	P	P	3	P	P	P	A	P
15	18Y	M	2 months	Parietal	2	2.5	1	P	P	P	P	A	1	P	P	P	P	A	2	P	P	P	P	P	2	P	P	P	A	P	3	P	P	P	P	P
16	23Y	M	1 month	Frontal	2.5	1.5	1	A	P	A	P	A	1	P	P	A	P	A	2	A	P	A	A	P	2	A	P	A	P	P	3	A	P	A	A	P
17	29Y	M	1 month	Occipital	2	3	1	A	P	P	A	A	1	A	P	P	P	A	2	P	P	P	P	P	3	P	P	P	P	P	3	P	P	P	A	P
18	22Y	M	2 weeks	Parietal	2	1.5	1	P	P	A	P	A	2	P	P	A	A	P	2	A	P	P	P	P	3	P	P	P	P	P	3	P	P	A	A	P
19	45Y	F	1 month	Occipital	2	3	0	A	A	P	A	A	1	A	P	P	P	A	2	P	P	A	P	P	2	A	P	A	A	P	3	A	P	A	P	P
20	22Y	F	1 month	Parietal	2.5	2	1	P	P	A	P	A	1	P	P	A	A	A	2	P	P	P	P	P	3	A	P	P	A	P	3	P	P	P	P	P
21	28	M	1 m	Parie	2	1.	1	P	P	A	P	A	2	P	P	A	P	P	2	P	P	A	P	P	3	P	P	A	P	P	3	P	P	A	A	P

	Y		onth	tal		5																														
22	36Y	M	2 months	Parietal	2	2.5	0	P	A	A	A	A	1	A	P	A	A	A	2	A	P	A	A	P	3	A	P	A	A	P	4	P	P	A	A	P
23	28Y	M	1 month	Occipital	1.5	2	0	P	A	A	P	A	1	P	P	P	P	A	2	A	P	P	A	P	3	A	P	P	A	P	3	A	P	A	A	P
24	42Y	M	3 months	Parietal	2	3	0	A	A	P	A	A	1	A	P	P	A	A	2	A	P	A	A	P	2	A	P	A	A	P	3	A	P	A	A	P
25	22Y	F	1 month	Frontal	2	1	0	P	A	P	A	A	1	P	P	P	A	A	1	A	P	A	A	A	2	A	P	A	A	P	3	A	P	A	A	P
26	26Y	M	1 month	Parietal	1	1	1	P	P	P	P	A	2	P	P	A	P	P	3	P	P	P	A	P	3	P	P	A	A	P	4	P	P	A	A	P
27	30Y	M	2 weeks	Parietal	2	1.5	0	P	A	P	P	A	1	P	P	A	P	A	2	P	P	P	A	P	3	P	P	A	A	P	3	P	P	A	A	P
28	24Y	F	1 month	Occipital	2	2	0	P	A	A	A	A	1	P	P	A	A	A	2	A	P	A	A	P	3	A	P	A	A	P	4	A	P	A	A	P
29	18Y	M	2 weeks	Parietal	1.5	2	0	P	A	P	A	A	1	P	P	P	A	A	1	P	P	A	A	A	2	A	P	A	A	P	3	A	P	A	A	P
30	22	M	2 m	Fron	1.5	2	1	P	P	P	A	A	2	P	P	P	A	P	2	P	P	A	A	P	3	A	P	A	A	P	4	A	P	A	A	P

	Y		onths	tal																																
31	35Y	M	1month	Parietal	2	2.5	0	P	A	P	P	A	1	P	P	P	A	A	2	P	P	P	A	P	3	P	P	A	A	P	4	P	P	A	A	P
32	28Y	M	1month	Parietal	2	1.5	0	P	A	P	A	A	1	P	P	P	A	A	1	P	P	P	A	A	2	P	P	A	A	P	3	A	P	A	A	P

Sr No	Age	Sex	Duration	Patch B																															
				Site	Size		Baseline						1st F/U						2nd F/U						3rd F/U						4th F/U					
					Length (cm)	Width (cm)	MDHN	Trichoscopy					MDHN	Trichoscopy					MDHN						MDHN	Trichoscopy					MDHN	Trichoscopy				
								BD	SVH	YD	EH	TH		BD	SVH	YD	EH	TH		BD	SVH	YD	EH	TH		BD	SVH	YD	EH	TH		BD	SVH	YD	EH	TH
1	15Y	M	2 months	Parietal	4.5	3.5	0	A	P	A	A	A	1	A	P	P	A	A	1	P	P	P	P	A	2	P	P	A	A	P	2	P	P	A	P	P
2	31Y	M	1 week	Parietal	2	2	1	P	P	A	P	A	1	P	P	P	P	A	2	P	P	P	P	P	2	P	P	P	P	P	3	P	P	P	A	P
3	15Y	F	6 months	Occipital	4	4	0	A	A	A	A	A	1	P	P	A	A	A	1	P	P	A	A	A	2	P	P	A	P	P	3	P	P	A	P	P
4	28Y	M	1 month	Frontal	2.5	3.5	1	P	P	P	P	A	1	P	P	P	P	A	2	A	P	P	A	P	2	P	P	P	P	P	2	P	P	P	P	P

5	24Y	M	2 months	Occipital	2.5	3	1	A	P	P	P	A	1	A	P	P	P	A	2	P	P	P	P	P	2	P	P	P	A	P	2	P	P	A	A	P
6	17Y	M	1 month	Parietal	1	2	1	P	P	P	P	A	2	P	P	A	P	P	3	P	P	A	P	P	4	A	P	A	P	P	4	A	P	A	A	P
7	30Y	F	1 month	Occipital	4	5	0	A	P	A	P	A	1	P	P	P	P	A	2	A	P	P	P	P	2	P	P	P	P	P	3	P	P	P	A	P
8	39Y	M	2 months	Occipital	3	2	0	P	A	P	A	A	0	P	A	P	P	A	1	P	P	P	P	A	2	P	P	P	A	P	2	P	P	P	A	P
9	18Y	M	1 week	Parietal	3	2	1	P	P	A	P	A	2	P	P	A	P	P	2	P	P	A	P	P	2	P	P	A	P	P	3	P	P	A	A	P
10	34Y	M	6 months	Frontal	3	2	1	A	P	P	P	A	1	A	P	A	P	A	2	P	P	A	A	P	2	P	P	P	P	P	2	P	P	P	P	P
11	27Y	M	6 months	Parietal	3	2.5	0	P	P	P	P	A	1	P	P	P	A	A	1	P	P	A	P	A	2	P	P	P	P	P	2	P	P	A	A	P
12	21Y	M	2 years	Parietal	1.5	2	0	P	P	P	A	A	1	P	P	P	P	A	1	P	P	P	A	P	1	P	P	P	P	A	2	P	P	A	P	P
13	28	M	5 m	Parie	2.5	2	0	A	A	A	A	A	1	P	P	A	P	A	1	P	P	A	P	P	2	A	P	A	P	P	3	P	P	A	A	P

	Y		onths	tal																																
14	42Y	M	1month	Parietal	2	1.5	1	A	P	P	P	A	1	A	P	P	A	A	1	A	P	A	P	P	1	P	P	A	A	P	2	P	P	A	A	P
15	18Y	M	2months	Parietal	2	2	1	P	P	P	P	A	1	P	P	P	P	A	1	P	P	A	P	A	2	P	P	A	P	P	2	P	P	P	P	P
16	23Y	M	1month	Occipital	2	2	0	P	P	P	P	A	0	P	P	P	P	A	1	P	P	P	P	A	2	P	P	A	P	P	2	P	P	A	A	P
17	29Y	M	1month	Parietal	2	1.5	1	P	P	P	P	A	1	P	P	P	P	A	2	P	P	P	P	P	2	P	P	P	A	P	3	A	P	A	P	P
18	22Y	M	2weeks	Occipital	2	2	1	P	P	A	P	A	1	P	P	A	P	A	2	P	P	A	A	P	2	A	P	A	P	P	3	P	P	A	P	P
19	45Y	F	1month	Occipital	2	2	0	A	A	P	A	A	1	A	P	P	A	A	1	P	P	P	P	A	2	P	P	P	P	P	3	P	P	P	P	P
20	22Y	F	1month	Parietal	2.5	2.5	1	A	P	A	P	A	1	A	P	P	P	A	2	A	P	P	A	P	2	P	P	A	P	P	2	P	P	A	A	P
21	28Y	M	1month	Frontal	1.5	2.5	1	P	P	A	P	A	2	P	P	A	P	P	3	P	P	A	P	P	3	A	P	A	A	P	3	P	P	A	A	P
22	36	M	2m	Occi	1	2	0	P	A	A	A	A	0	P	A	P	A	A	1	P	P	P	A	A	2	P	P	A	A	P	2	P	P	A	A	P

	Y		onths	pital																																
23	28Y	M	1month	Parietal	2	2	0	P	A	A	P	A	1	P	P	P	A	A	2	P	P	A	A	P	2	A	P	A	A	P	3	A	P	A	A	P
24	42Y	M	3months	Parietal	1.5	2	0	A	A	A	A	A	1	A	P	A	A	A	1	A	P	A	A	A	2	A	P	A	A	P	2	A	P	A	A	P
25	22Y	F	1month	Parietal	1	1	0	P	A	P	A	A	1	P	P	P	A	A	1	A	P	A	A	A	2	A	P	A	A	P	2	A	P	A	A	P
26	26Y	M	1month	Parietal	1	2	1	P	P	P	P	A	2	P	P	P	A	P	2	A	P	A	A	P	3	A	P	A	A	P	4	A	P	A	A	P
27	30Y	M	2weeks	Occipital	2	1	0	P	A	P	A	A	1	P	P	P	A	A	1	A	P	A	A	A	2	P	P	A	A	P	3	P	P	A	A	P
28	24Y	F	1month	Parietal	1	2	0	P	A	A	A	A	1	P	P	A	A	A	2	A	P	A	A	P	2	A	P	A	A	P	3	A	P	A	A	P
29	18Y	M	2weeks	Occipital	2	2	0	P	A	P	A	A	1	P	P	P	A	A	1	P	P	A	A	A	2	A	P	A	A	P	2	A	P	A	A	P
30	22Y	M	2months	Parietal	2	1	1	P	P	P	A	A	2	P	P	P	A	P	2	P	P	A	A	P	3	A	P	A	A	P	4	A	P	A	A	P

31	35Y	M	1month	Frontal	2	2	0	P	A	P	P	A	1	P	P	P	A	A	2	P	P	A	A	P	2	P	P	A	A	P	2	P	P	A	A	P
32	28Y	M	1month	Occipital	2	2	0	P	A	P	P	A	1	P	P	P	A	A	2	A	P	A	A	P	2	P	P	A	A	P	3	P	P	A	A	P