"A COMPARATIVE STUDY OF FRACTIONAL CO2 LASER WITH TOPICAL BIMATOPROST 0.03% VERSUS FRACTIONAL CO2 LASER WITH TOPICAL TRIAMCINOLONE ACETONIDE IN THE TREATMENT OF ALOPECIA AREATA"

BY

Dr. HUSSAIN KOLSAWALA, M.B.B.S.

DISSERTATION SUBMITTED TO

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH,
TAMAKA, KOLAR, KARNATAKA,

IN PARTIAL FULFILLMENT OF THE REQUIREMENT FOR THE DEGREE OF

DOCTOR OF MEDICINE (M.D.)

IN

DERMATOLOGY, VENEREOLOGY AND LEPROSY

UNDER THE GUIDANCE OF

Dr. K. HANUMANTHAYYA.M.B.B.S., M.D.

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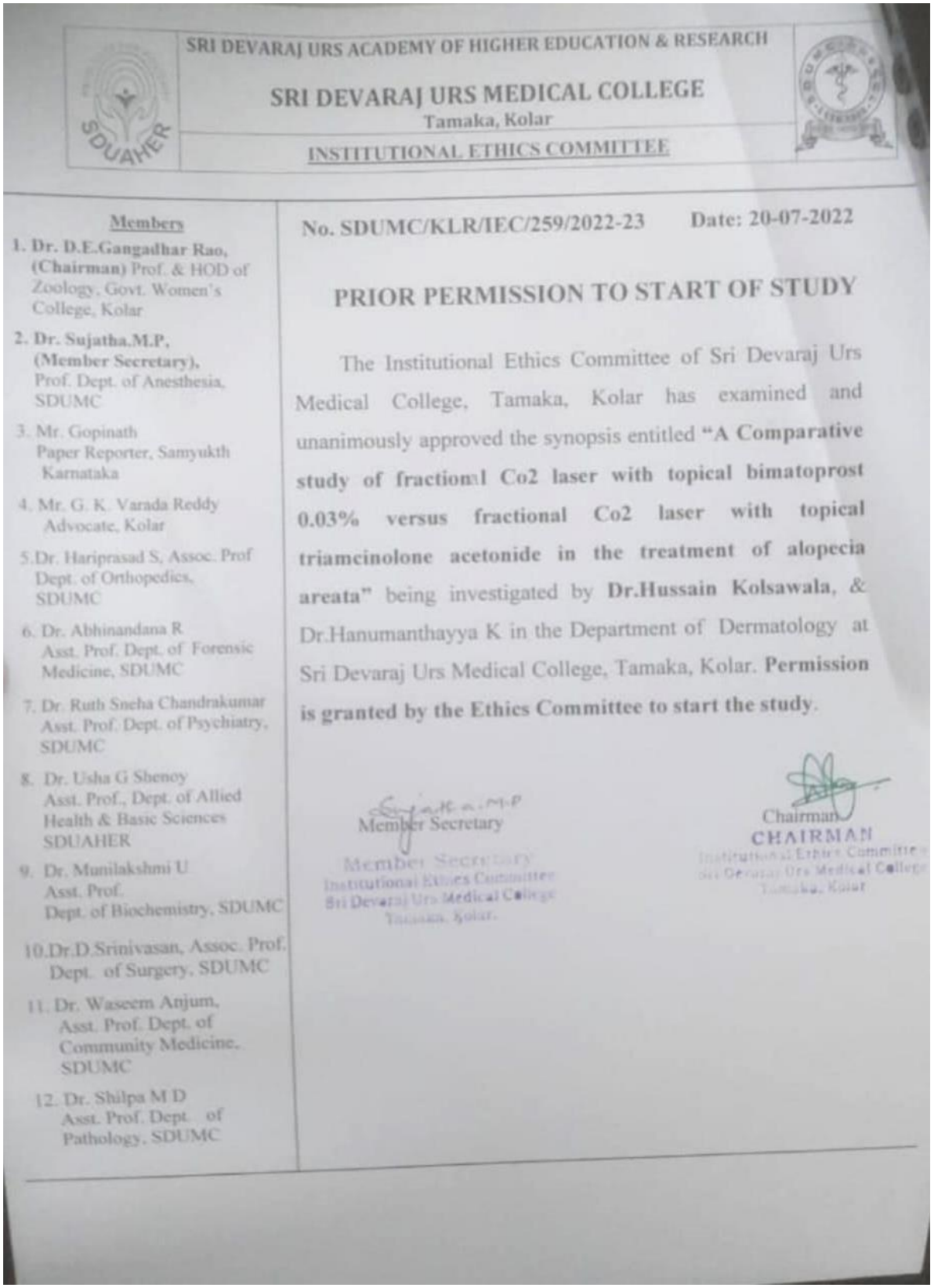

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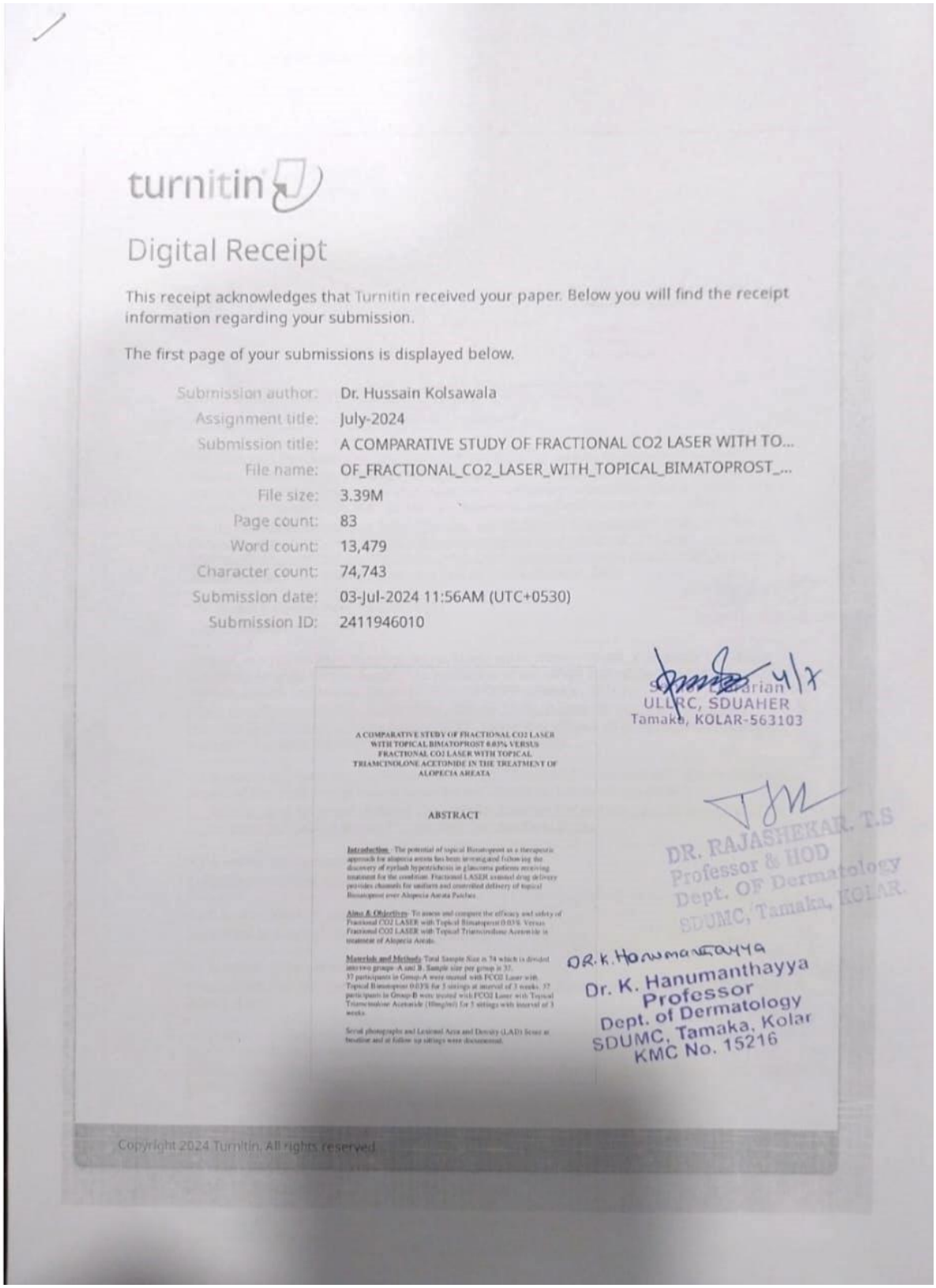

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ABSTRACT

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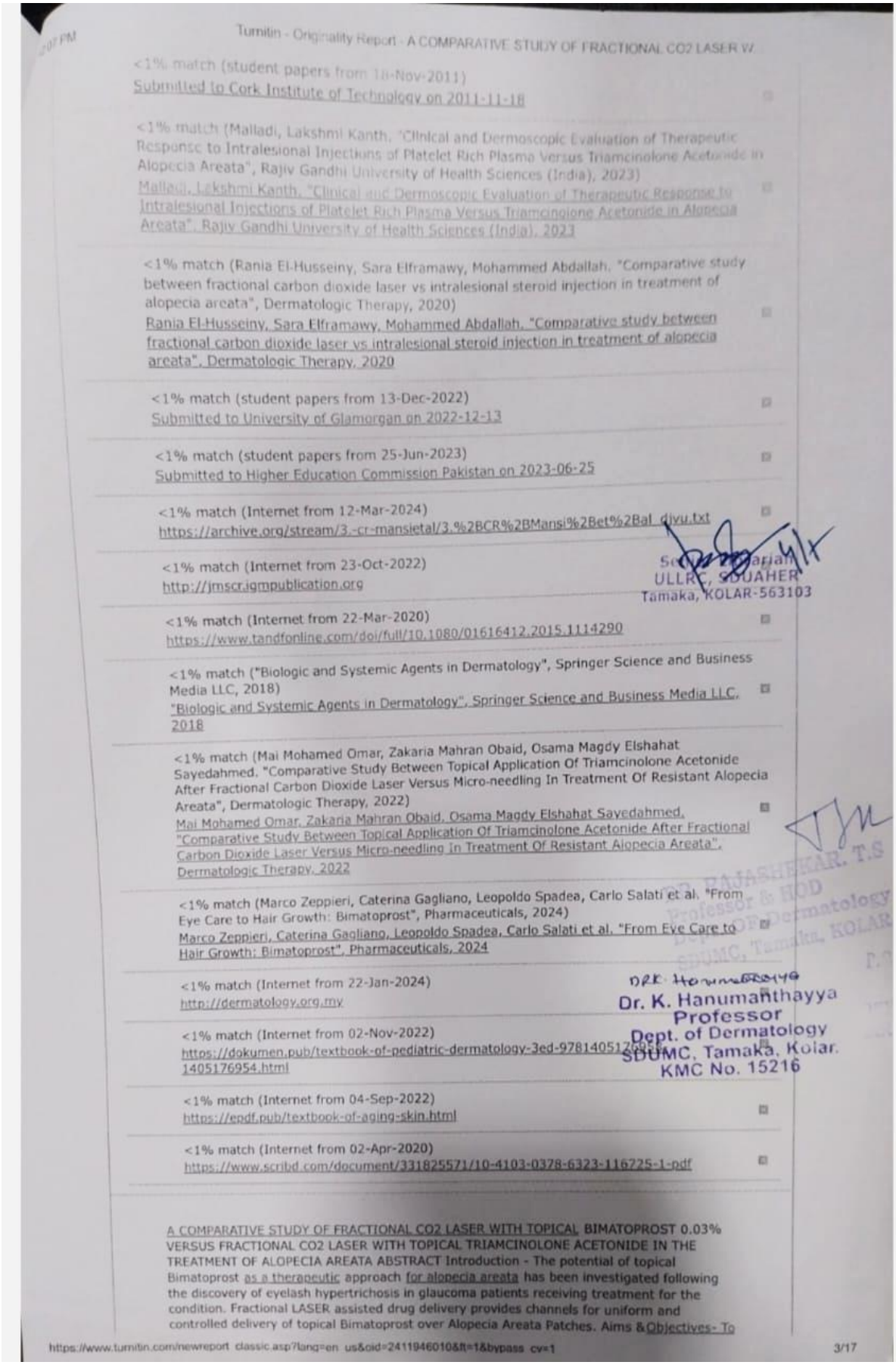

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I must express my gratitude to who have been my closest friends at every stage of my life, for teaching me the realities of both my personal and professional lives.

No words can express the gratitude I feel towards my beloved parents, ***Mr. Tafazzul Kolsawala*** *and* ***Mrs. Sakina Kolsawala****, and I also want to thank my brother Hassan Kolsawala and sister Farida Teta whose countless sacrifices and endless love has made me who I am today in life.*

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I am truly blessed in having the most wonderful friends and would like to thank them for their endless support.

I will be failing my duty if I do not thank all my patients involved in this study, without whose co-operation and patience this study would have been impossible.

Last, but not the least, I would like to express my gratitude to the Almighty for all his blessings.

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ABSTRACT

Introduction -Discovery of eyelash hypertrichosis induced by topical Bimatoprost in patients treated for glaucoma has led to exploring the potential of this therapeutic modality in alopecia areata. Fractional LASER assisted drug delivery provides channels for uniform and controlled delivery of topical Bimatoprost over Alopecia Areata Patches.

Aims &Objectives-To assess and compare the efficacy and safety of Fractional CO2 LASER with Topical Bimatoprost 0.03% Versus Fractional CO2 LASER with Topical Triamcinolone Acetonide in treatment of Alopecia Areata.

Materials and Methods-Total Sample Size is 74 which is divided into two groups–A and B. Sample size per group is 37.

37 participants in Group-A were treated with Fractional CO2 Laser with Topical Bimatoprost 0.03% for 5 sittings at interval of 3 weeks. 37 participants in Group-B were treated with Fractional CO2 Laser with Topical Triamcinolone Acetonide (10mg/ml) for 5 sittings with interval of 3 weeks.

Serial photographs and Lesional Area and Density (LAD) Score at baseline and at follow up sittings were documented.

Results-Average percentage of improvement in LAD score calculated for 37 patients in each group after 5 sittings of respective treatment modality was 95.3% and 75.6% in Group-A and Group-B respectively. Except transient erythema, Group-A had lesser or no adverse effects like edema, atrophy of skin and post inflammatory hypo/hyperpigmentation when compared with Group-B.

Conclusion-Efficacy and safety of treatment assessed using percentage improvement in LAD Score and Global photographic assessment showed topical Bimatoprost with FCO2 LASER to be a novel and more efficacious treatment modality with better safety profile for treatment of Alopecia Areata.

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LIST OF ABBREVIATIONS

S.NO	ABBREVIATIONS	FULL FORMS
1	AA	Alopecia Areata
2	LASER	Light Amplification by Stimulated Emission of Radiation
3	CO2	Carbon dioxide
4	GPA	Global Photograph Assessment
5	LAD Score	Lesional Area Density Score
6	LAD Score Improvement %	Lesional Area Density Score Percentage of Improvement
7	VDS	Visual Discomfort scale
8	VAS	Visual Analogue Scale
12	ULBP	UL16 Binding Protien
13	ILS	Intra Lesional Steroids
14	CTLA-4	Cytotoxic T-Lymphocyte associated protein 4
15	GWAS	Genome Wide Association Studies
16	IFN	Interferon
17	ROS	Reactive Oxygen Species
18	MHC	Major Histocompatibility Complex
19	JAK – STAT PATHWAY	Janus Kinase Signal Transducers and Activators of Transcription Pathway
20	CD	Clusters of Differentiation

21	HLA	Human Leukocyte Antigen
22	AGA	Andro Genetic Alopecia
23	AIRE	Autoimmune Regulator
24	SALT SCORE	Severity of Alopecia Tool Score
25	AAPI	Alopecia Areata Progression Index
26	OMP	Oral Mini Pulse
27	OS	Oxidative Stress
28	JAKis	Janus Kinase Inhibitors
29	IL	Inter Leukin
30	GARP	Glycoprotein-A Repetitions Predominant
31	NKG2D	Natural Killer Group 2D
32	SNPs	Single-Nucleotide Polymorphisms

INTRODUCTION

INTRODUCTION

Alopecia areata (AA) is a long-term, immune-mediated condition marked by sudden, non-scarring hair loss that can range from small, localized scalp patches to whole body and scalp hair loss. Until recently, there was little knowledge available on the pathophysiology of adult-onset and childhood-onset AA. As of right now, the theory is that the disease starts when the immune privilege (IP) of the hair follicle (HF) collapses, maybe as a result of both genetic and environmental causes.[1]

The Greek word alopekia, which means fox mange, is credited to Celsus for describing patterns of hair loss; Sauvages is credited with coining the name "alopecia areata." Alopecia areata is a type of non-cicatricial alopecia that is thought to be an autoimmune illness specific to hair, with hereditary variables influencing the disease's severity and susceptibility.[2]

Alopecia areata is a type of non-cicatricial alopecia that is thought to be an autoimmune illness specific to hair, with hereditary variables influencing the disease's severity and susceptibility.[3]

Therapeutically, there has been a glaring gap in the literature for a medication that can bring about a long-lasting, if not permanent, remission. The majority of the available symptom-based topical and/or systemic treatments use either immune modifying or non-specific immunosuppressive strategies.[4]

The JAK-signal transducer and activator of transcription (STAT) signaling pathway has been identified as a potential therapeutic target, opening new avenues in the understanding and management of this disease. This recognition has been made possible by the results of

genome wide association studies (GWAS) and recent evidence that suggests Janus kinases (JAKs) are important in the pathogenesis of AA.[5,6]

The unpredictable prognosis of alopecia areata, which frequently involves relapses and remissions over an extended period of time, adds significantly to the psychosocial load experienced by AA patients. Consequently, it is crucial that we increase our knowledge about the illness.[7]

AIMS & OBJECTIVES

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AIM OF THE STUDY:

- To assess and compare the efficacy and safety of Fractional CO2 Laser with Topical Bimatoprost 0.03% versus Fractional CO2 Laser with Topical Triamcinolone Acetonide in the treatment of Alopecia Areata.

Objectives of the Study:

1) To assess and compare the efficacy of Fractional CO2 Laser with Topical Bimatoprost 0.03% versus Fractional CO2 Laser with Topical Triamcinolone Acetonide in the treatment of Alopecia Areata.
2) To document the post procedural adverse effects of Fractional CO2 with Topical Bimatoprost 0.03% and Fractional Co2 Laser with Topical Triamcinolone Acetonide in the treatment of Alopecia areata.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

DEFINITION:

A non-cicatricial alopecia that affects the anagen hair follicles on the scalp, beard, or any other part of the body with a chronic autoimmune aetiology of heterogeneous severity is referred to as AA.[8]

SYNONYMS:

• PELADE • AREA CELSI

EPIDEMIOLOGY:

In the general population, the estimated lifetime risk of getting alopecia areata is 2%, with a prevalence of roughly 1 in 1000. Patients with Asian, Black, and Hispanic ancestry are more likely to experience alopecia areata, despite the absence of evidence supporting a sex predilection.[9]

It accounts for 0.7% of the new dermatology cases in India with prevalence of 0.1-0.2% and overall lifetime risk of 1.7%. Both adults and children can have alopecia areata, and the condition's incidence increases with age. For males, the typical age of onset is 32, while for females, it is 36.[10]

ETIOPATHOGENESIS:

Pathogenesis is largely influenced by genetic, environmental, and immunological variables, such as cytokines and T lymphocytes. An anticipated histological characteristic of AA is a peribulbar lymphocytic infiltration, which is indicative of activated T cells.[11]

Nonetheless, there is ongoing debate on the general AA etiology, leading to vague and inadequate treatment approaches. While there are numerous treatment options for AA, including systemic, topical, and phototherapies, only 30% of patients achieve long-lasting remissions, making AA a clinical challenge.[12]

Although the precise cause of the condition is unknown, autoimmunity, an atopic state, the patient's genetic makeup, non-specific immune responses, mental stress, and a variety of psychological factors may be to blame.[13]

Patients may have one or more patches at any body part where hair grows. Other autoimmune diseases such atopy, diabetes mellitus, hypertension, and asthma have been linked to AA. Ikeda divided AA into four categories in 1989: common, atopic, pre-hypertensive, and autoimmune type, depending on the illness's progression and related disorders.[14]

One representative part of the scalp that is heavily impacted by immunological cells and hormones is the hair follicle. The majority of hair follicles are found at the scalp's head, which is anticipated to be impacted by a number of variables, including temperature and UV radiation exposure. One typical hair follicle condition is alopecia areata, and it is well recognized that environmental factors can affect the course of the disease.[15]

A comprehensive review and meta-analysis of eighteen papers regarding OS in patients with AA was carried out by Acharya et al. in 2019. They found that whilst antioxidant indices were decreasing, pro-oxidative indices were increasing. Moreover, a relationship was shown between the severity of the condition and the rise in oxidation levels.[16,17]

Daily Lifestyle Factors Related to Alopecia Areata[18]

Smoking

Alcohol Consumption

Sleep disturbance

Obesity

Fatty acids

Gluten

GENETIC FACTORS:

Studies using observational data reveal a strong (10%–42%) association between AA and family history. Single-nucleotide polymorphisms (SNPs) linked to AA have been found in large numbers through genome-wide association studies(GWAS). Human leukocyte antigen-DR (HLA-DR) on chromosome 6 seems to be the main risk factor for AA, according to a recent meta-analysis. The CD4+ and CD8+ T-cells, which are significant effector cells in AA, are closely associated with these HLA class II genes.[19]

Furthermore, BCL2-like protein 11, or BIM, which aids in controlling autophagy, was also linked in the etiology of the disease in this investigation. AA susceptibility is also influenced by genes encoding downstream effectors of the JAK pathway and natural killer cell receptor D ligands. T-regulatory cells (Tregs), autophagy, and apoptosis are additional pathways that have been linked to the process; however, more data is required to pinpoint the precise mechanisms.[20]

ENVIRONMENTAL FACTORS:

Environmental variables most likely cause or worsen AA. Although stress is frequently suggested as a cause of AA, there is conflicting evidence in human study literature. On the other hand, compared to normal mice, the central and peripheral hypothalamic pituitary adrenal axis activity was higher in a mouse model. Pro-inflammatory cytokine levels in the skin were positively linked with higher levels of adrenocorticotropic hormone, corticosterone, and oestradiol, indicating a possible role for physiological and psychological stressors in the development of AA.[21]

Although their precise effects are unknown, illnesses, immunizations, variations in hormone levels, and food are some possible environmental stresses that may be linked to AA. Soy products have been linked to AA in the mouse model, and recent research has highlighted a relationship between AA and levels of vitamins A and D. Numerous environmental elements most likely have an impact on the course of the disease.[22]

FOLLICULAR IMMUNE PRIVILEGE IN AA:

A zone of immune privilege exists in a normal hair follicle as a result of decreased activity of antigen-presenting cells, synthesis of immunosuppressive molecules including α-melanocyte-stimulating hormone and transforming growth factor-β (TNF-β), and downregulation of MHC I and β2 macroglobulin molecules. It is postulated that an unidentified autoantigen in AA is responsible for the immunological privilege zone's collapse. The immunological privilege zone can then be reached by CD8+, CD4+, and other inflammatory cell infiltration caused by interferon-γ (IFN-γ) and interleukin (IL)-2. Hair follicle inflammation is a result of all of these changes, and it can lead to hair loss.[23]

A hallmark of immunological privilege collapse is the overexpression of MHC-I and -II. A growing body of research suggests that the proinflammatory cytokine interferon-γ (IFNγ) enhances MHC I and II expression, which in turn causes HFIP collapse and immune-mediated eHFSC destruction. Increased MHC-I expression has been suggested to help CD8+ T cells launch an autoimmune assault in AA. Anagen hair bulbs from murine back skin have demonstrated that IFNγ functions as a strong upregulator of MHC-I in vivo when compared to other cytokines (e.g., IL-1β, TNF-α) thought to promote MHC-I. Additionally, Ito et al. administered IFNγ to human scalp HFs; lower doses were enough to cause ectopic MHC class 1 expression, whereas higher doses resulted in an early induction of catagen in vitro.[24]

NK cells have been the subject of current research about their possible involvement in HFIP breakdown, specifically in AA. MHC class I chain-related A gene (MICA), a stress-induced ligand that activates NKG2D recognition receptors on NK and CD8+ T cells, is hardly expressed in healthy HFs. Other autoimmune conditions like type I diabetes and rheumatoid arthritis have been linked to NKG2D. Lesional AA HFs are found to be surrounded by NKG2D+ NK and CD8+T cells, and they have strong MICA immunoreactivity. Atypically elevated MICA expression could potentially enable HF assault through triggered NKG2D+ cells, resulting in anagen phase disruption and hair loss.[25]

CLINICAL FEATURES:

Although AA can affect almost any part of the body that bears hair, 90% of cases treated in dermatological clinics involve the scalp. The degree or pattern of hair loss might be used to categorize the condition.Depending on how much hair has been lost, several clinical forms of AA can be seen.The most prevalent type of AA, which affects up to 75% of individuals, is patchy.[26]

Alopecia reticularis is characterized by a number of regrowing, stable, or active patches that may combine to create a reticulate or mosaic pattern. This type of alopecia is linked to prehypertensive and atopic variants of Ikeda's disease and has a worse prognosis.[27]

Alopecia subtotalis, or almost total hair loss on the head, and complete alopecia on the scalp affect a tiny minority of patients (10–20%). The name for the AA kind of alopecia, in which all of the hair on the body and scalp is lost, is alopecia universalis. Loss of the nose, ear hairs, eyelashes, and eyebrows is also included in this. Patients with Down syndrome, atopic individuals, and children are frequently affected by this severe type. Ten percent of cases involve one or more afflicted areas other than the scalp. It is possible for all body hair to be gone, either suddenly or gradually. Of patients with localized AA, 1%–5% develop alopecia totalis.[28]

The term "ophiasis" (Greek for snake) refers to a unique band-like pattern of AA that coils around the occipital hairline and extends toward the temples. It frequently responds poorly to treatment and typically has a more challenging prognosis. It is more prevalent among atopic individuals and children.A band-like pattern on the frontal hairline is even more unusual; this is not the same as frontal fibrosing alopecia. The reverse of ophiasis, known as sisaipho, is characterized by core hair loss and sparing at the scalp's edges.It might resemble alopecia androgenetic.[29]

Rebora provided the initial description of alopecia areata incognita (AAI) in 1987. Within a few weeks, the disorder's extremely abrupt beginning gives way to generalized hair loss. Short regrowing hairs and yellow specks are visible on dermoscopy. Perinevoid alopecia areata is a peculiar and uncommon type characterized by patches surrounding a nevus. A variation of acute diffuse and complete baldness has been reported recently. It has a good prognosis despite its rapid development and widespread hair loss.[27,30]

While diffuse AA covers the entire scalp, not every hair is typically affected. The diagnosis is made possible by a very positive pull test, the extra patch existence, and dermoscopic evidence of AA. Some individuals have patches on their bodies or beards, eyelashes, or eyebrows, either with or without scalp baldness.[31]

A hairdresser or relative usually notices the initial patch, which is typically asymptomatic. It's a perfectly defined, smooth, round or oval area of complete baldness. Occasionally, there is minor erythema or paresthesia along with the beginning.[32]

The distinctive hair with an exclamation point can be spotted around the edges of a newly formed or growing patch. They resemble broken hair that is 2-4 mm long, with progressively thinner and less pigmented shafts near the bulb end. Their free ends are spread, producing the appearance of a "frayed rope," as seen by a hand lens. It is likely to spread since some telogen hair can be easily pulled out surrounding such an area. If terminal hair is discovered within a patch, it may appear to be unharmed but may exhibit one or more shaft constrictions.[33]

Shuster explained how to distinguish diffuse AA from other diffuse alopecias using the "Coudability" indicator. As a result, in AA, a typical-looking hair kinks 5–10 mm above the surface when twisted or pressed inward. Forme fruste exclamation mark hair is created when a portion of the terminal hair taper at the proximal end.[34]

The original patch may grow back in a few months, or additional patches may appear 3-6 weeks apart and then cyclically. The length of these periods varies.When neighboring patches confluence, alopecia totalis, or near total alopecia, may result.[35]

The sparing of white hairs is an interesting characteristic of alopecia areata. White or non-pigmented hair appears to be protected from the illness process, which tends to preferentially attack pigmented hair. If the alopecia advances quickly, this could lead to a noticeable change

in hair color, which is most likely the reason behind historical reports of persons "going white overnight." White hairs are not immune to the disease, while being less vulnerable to it.Regrowth in AA often occurs fine and unpigmented, progressively increasing in quality and color. Heterochromia, or variations in pigmentation across a single hair's length or among a group of hairs, is the outcome of this.[36]

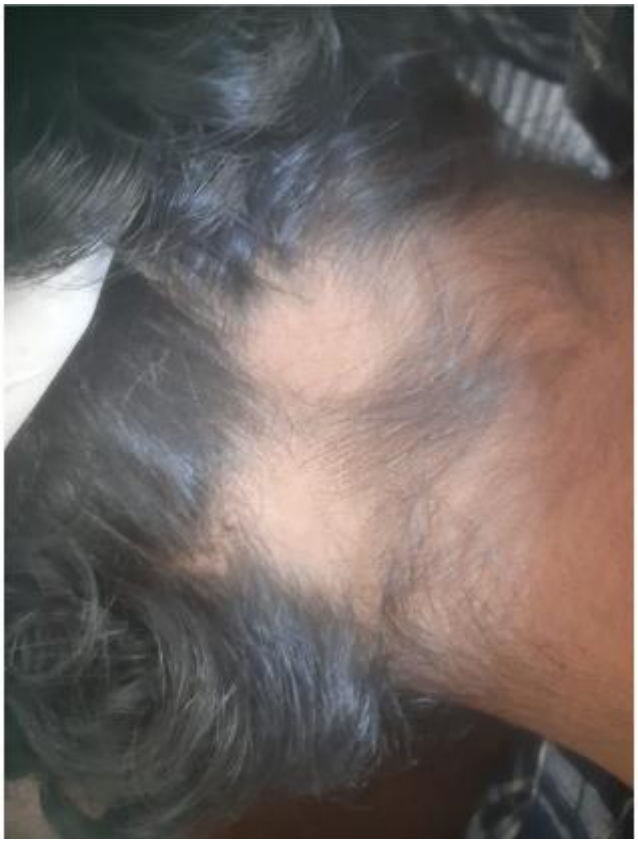

FIGURE-1 LOCALISED PATCH OF ALOPECIA AREATA OVER THE SCALP

CLINICAL CLASSIFICATION/VARIANTS OF AA:[37,38]

Based on Extent

Patchy AA

Alopecia Subtotalis

Alopecia Totalis

Alopecia Universalis

Based on pattern

Reticular

Ophiasis

Sisaipho (Ophiasis Inversus)

Linear

Perinevoid

Miscellaneous variants

AA Incognito (AAI)

NAIL CHANGES:

The different nail changes are as follows:[39,40,41,42,43,44]

1. Pitting

2. Longitudinal striations

3. Lomellar solittina

4. Trachyonychia

5. Distal notching

6. Ragged cuticles

7. Beau′s lines

DIFFERENTIAL DIAGNOSIS OF AA: [45]

Trichotillomania,

Traction alopecia,

Tinea capitis,

Secondary syphilis,

Androgenetic alopecia,

Aplasia cutis,

Temporal triangular alopecia.

AA ASSOCIATIONS: Related illnesses [46,47,48,49]

• Atopy (atopic dermatitis, asthma, and allergic rhinitis); >40% in certain studies; • Vitiligo, inflammatory bowel disease, and autoimmune thyroid disease (e.g., Hashimoto thyroiditis).

• Type 1 diabetes is more common in relatives of patients with alopecia areata (autosomal recessive, caused by mutations in the autoimmune regulator gene [AIRE]; up to 30% of patients have alopecia areata).

HLA associations

• HLA-DQB1*03 appears to be a susceptibility HLA marker for all forms of alopecia areata, but the HLA alleles DRB1*0401 (DR4) and HLA-DQB1*0301 (DQ7) are thought to be markers for severe long-term alopecia totalis/universalis. HLA-DQB1*03, HLA-DQB1*03 (DQ3), and HLA-DRB1*1104 (DR11).

• Treg cell functions include CTLA4, IL-2/IL-21, IL2RA, Eos, and GARP.

• The ULBP gene cluster encodes ligands that activate the NKG2D NK cell receptor.

COURSE AND PROGNOSIS:

People who have alopecia areata still have the ability to develop new hair since between 34 and 50 percent of people who have patchy hair loss recover on their own in a year. Less than 10% of patients will fully heal, and the majority will experience recurrence. Alopecia totalis or universalis affects roughly 10% of patients with patchy illness.[50]

The degree of hair loss and the patient's age at diagnosis are important prognostic variables. A young diagnosis and an ophiasis distribution point to a poor prognosis. A bad prognosis may also be indicated by atopy, nail dystrophy, alopecia areata in the family, severe disease, duration of more than a year, or concomitant autoimmune disease.[51]

DIAGNOSIS:

Clinical diagnosis of alopecia areata usually depends on the patient's medical history and physical examination. Individual skin alopecia patches that appear quickly and are frequently accompanied by mild erythema should arouse clinical suspicions. [52]

HISTOPATHOLOGY:

Alopecia Areata

Depending on the stage of the disease, the histology will change. A "swarm of bees"-like peribulbar lymphocytic infiltration, consisting of CD4+ and CD8+ T-cells around anagen follicles, is seen during the acute and subacute phases. Furthermore, follicle shrinking occurs as the hair grows from the catagen stage to the telogen phase, or resting phase. The area surrounding the hair follicles may exhibit edema, micro-vesiculation, apoptosis, macrophages, and large cells from the foreign body. In the chronic stage, pigmentary incontinence occurs, the amount of catagen or telogen hairs grows, and the inflammation may or may not go away. Anagen hair, or hair that is actively developing, is more prevalent and there is less inflammation during the recovery phase.[53]

Early, progressive disease ("acute" and "subacute")

• Normal total number of hairs; an infiltration of peribulbar mononuclear cells, primarily affecting terminal anagen and catagen hair bulbs, occasionally accompanied by eosinophils; Periodically, inflammatory cells are exocytosed into the bulbar epithelium; hair matrix degenerative changes occur; the number of terminal catagen and telogen hairs increases; the number of miniaturized hairs increases; trichomalacia and noticeable hair shaft constriction occur. Prolonged and steady illness referred to as "chronic".[53,54]

The majority of hairs are in the catagen or telogen phases. A large number of tiny, "arrested," quickly cycling hairs, known as nanogen hairs, are present. A mild peribulbar mononuclear cell infiltration surrounds those nanogen hairs that have bulbs that resemble anagen or catagen.[55]

In the early stages, there is arterial thrombosis, inflammation in the dermis, and superficial fat.When severe, eccrine coils and the epidermis will die. Synchronized transition of the majority or all of the terminal hair follicles to the catagen/telogen phase (without hair shrinkage) in association with alopecia Trichomalacia (but not altered follicular anatomy), pigment casts, and incontinent pigment within collapsed root sheaths.[53,54]

FIGURE-2 Histopathology of Alopecia Areata[53]

DERMOSCOPY/TRICHOSCOPY:

A noninvasive, simple and easy technique which helps in the accurate diagnosis of AA in challenging cases.[56,57,58]

The characteristic features of AA in Trichoscopy are as follows:

• Exclamation Mark (!) Hair – also called as Tapering Hair

• Black Dots – also called as Cadaver Hair

• Broken Hair

• Yellow Dots (indicates dystrophy of follicular epithelium and sebaceous glands) increased more than in Androgenetic Alopecia.

• Coudability sign can be appreciated even clearly as the trichoscope is placed over the terminal hairs.

• Increased number of yellow dots and black dots indicates the disease severity.

• Ratio of terminal to vellus hair is 1:1 indicating severe miniaturization exclusively in chronic cases.

• “i Hair” – in resolving lesions.

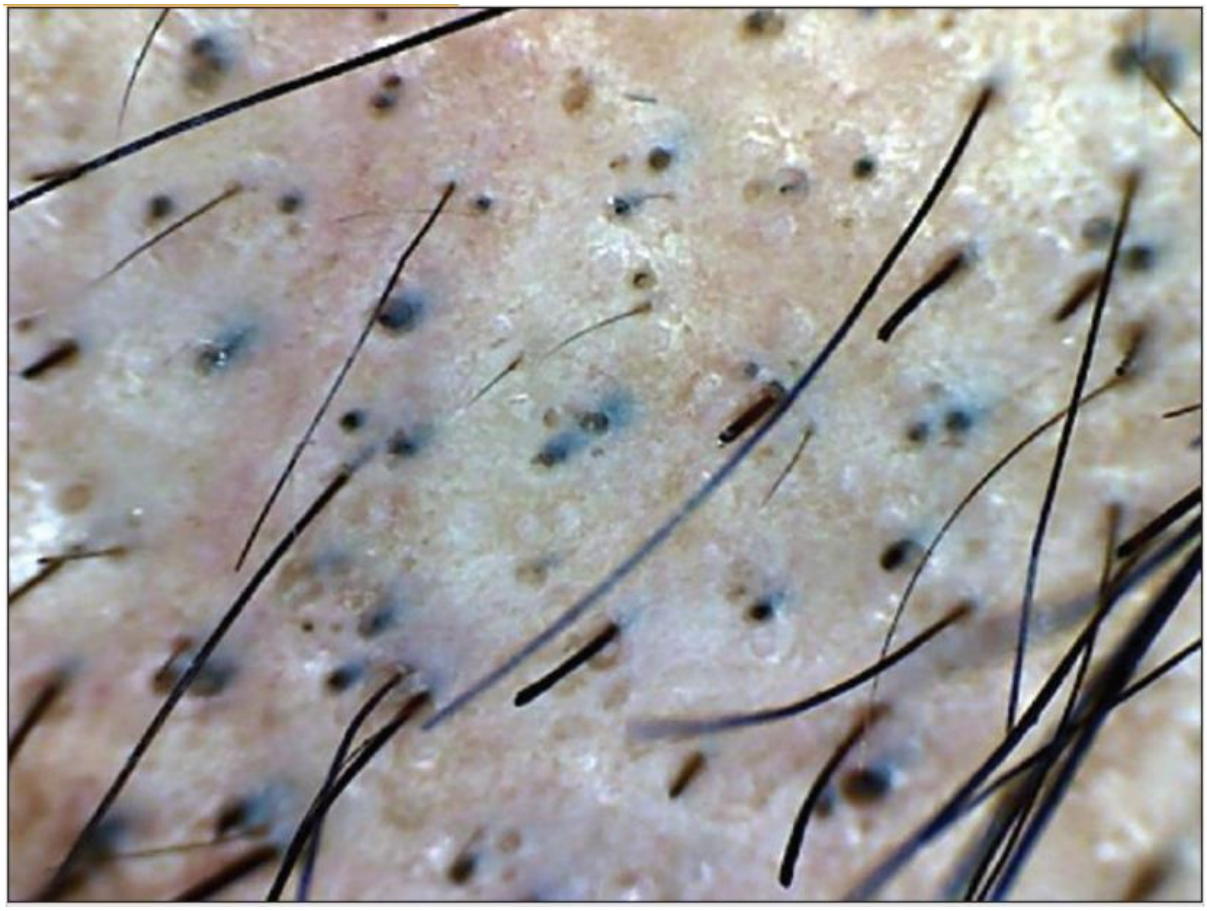

FIGURE-3 Dermoscopy of AA, showing-yellow dots, black dots, broken hair and tapering hair [56]

SEVERITY SCORING:

SEVERITY OF ALOPECIA TOOL (SALT) SCORE:[59]

- The SALT score is helpful in determining the quantitative evaluation of hair loss on the scalp.
- Based on surface area, the entire scalp was split into four sections: the right side (18% - 0.18), left side (18% - 0.18), posterior (24% - 0.24), and top (40% - 0.4).
- The percentage of scalp covered in each location is multiplied by the percentage of hair loss in that part of the scalp, and the results of adding the products of each area yield the SALT score.

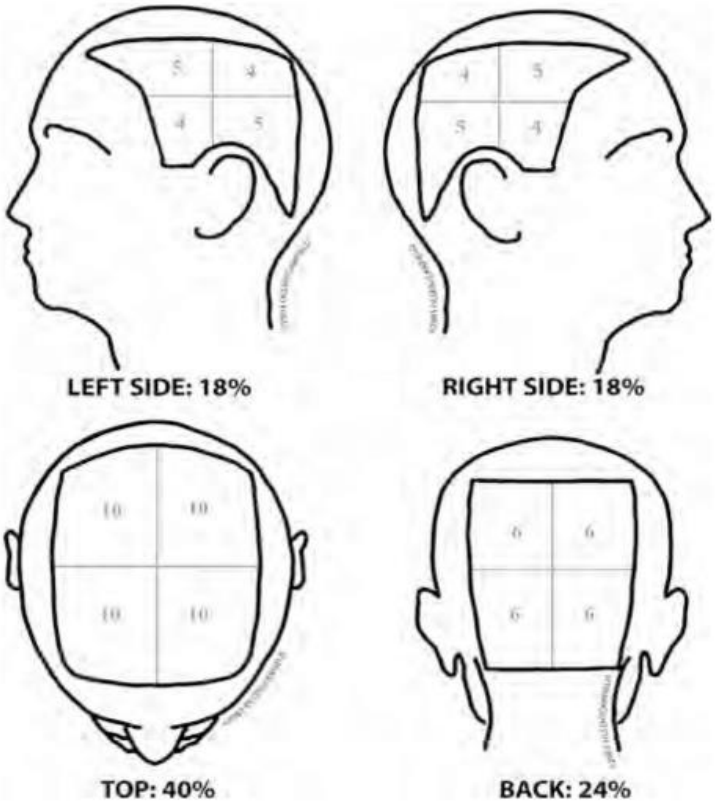

FIGURE-4: SEVERITY OF ALOPECIA TOOL (SALT SCORE) CALCULATION[60]

ALOPECIA AREATA PROGRESSION INDEX (AAPI):

Alopecia Areata Progressive Index[61]

- This approach was introduced in 2016 and involves using the SALT I visual aid to determine the percent scalp hair loss per quadrant, multiplying this amount by a hair loss activity score, and then adding the products of each quadrant.
- The evaluation of both exclamation point hairs and a mild hair pull at the edge of many patches of hair loss can help to determine active progressive hair loss. This hair loss activity score is based on (1) Activity of hair loss.

(2) Exclamation point hairs, broken hairs, and black dots were observed dermoscopically in a representative region of each quadrant.

LESIONAL AREA AND DENSITY (LAD) SCORE PERCENTAGE OF

IMPROVEMENT:

The score for Lesional Area and Density (LAD). This adds the area of every target lesion to a density score calculated on a 100-point density scale relative to normal. The total LAD score, if more than one target region is used, is the sum of the individual LAD scores.The long axis multiplied by the perpendicular axis yields the area of each patch. If the alopecia patch is completely round, πr 2 may be utilized in its place. For this approach, it will be necessary to figure out how to recognize these alopecia spots in photos taken during follow-up visits.[62]

TREATMENT:

Topical steroid

Corticosteroids are known to have a potent inhibitory effect on the activation of T-lymphocytes, which is why AA is a T-cell-mediated response. Tachyphylaxis, local folliculitis, and other side effects are possible.[63]

Topical minoxidil

The duration of the anagen growth phase is extended by minoxidil. It also causes the potassium channels to activate. Irritating dermatitis and allergic contact dermatitis are among the side effects of minoxidil.[64,65]

Anthralin

It is advised to avoid irritation with short contact therapy, yet irritation to some extent could be required for a therapeutic response.[66,67]

As a first-line systemic therapy for children, few clinicians favour using oral prednisolone and oral Mini pulse (betamethasone 0.1 mg/kg/day) two days in a row. Prednisolone has been used to treat AA at different doses, ranging from 0.1 mg/kg/day to 1 mg/kg/day. Its usage in the pediatric population should be postponed due to the possibility of stunted growth. Regarding the direction of systemic steroid therapy, there is no worldwide agreement. The majority of patients need ongoing care to preserve hair growth, and the majority of the time, the side effects of treatments are not worth the dangers.[68]

JAK-STAT inhibitors

JAK-STAT inhibitors have demonstrated potential advantages over traditional systemic treatment. A positive feedback loop plays a role in the pathophysiology of AA. Skin-infiltrating CD8 + NKG2D + T cells release IFN-gamma, which stimulates the synthesis of IL-15 and 1L-15 R alpha by follicular epithelial cells. This, in turn, activates and maintains the autoimmunity and response of CD8 + NKG2D + T cells. Tofacitinib, a JAK 1 and 3 inhibitors, is one JAK-STAT inhibitor that blocks this positive feedback loop, which in turn blocks the autoimmune process.[69]

Regrowth of hair has been observed in numerous case series while receiving tofacitinib medication. Adults should take 5 mg BD, while smaller children can take 5 mg OD or 5 mg every other day.[70]

Baricitinib (JAK 1 and 2 inhibitor) at a dose of 2-4 mg daily in a case report of long-standing AA has also demonstrated efficacy. Baseline investigations such as complete blood count, liver function tests, serum lipids, chest X-ray, and interferon-gamma release assay should be performed prior to initiating JAK inhibitors. Adverse effects may include elevated transaminases levels, hyperlipidemia, pancytopenia, increased susceptibility to infections, and increased risk for thromboembolic events. Regular monitoring of blood parameters such as complete blood count, liver function tests, and serum lipids is recommended.[71]

JAK inhibitors have already shown promise in treating a number of inflammatory conditions, including vitiligo, rheumatoid arthritis, and psoriasis. Additionally, there have been recent reports of JAK inhibitors being effective in treating TH2-driven conditions, such as atopic dermatitis.39–43 Preclinical research with JAK inhibitors in C3H/HeJ mice demonstrated dramatic hair regeneration, which strongly supported testing these drugs in humans.44 The first studies showing the effectiveness of a JAK inhibitor in AA included a patient with AU

who received tofacitinib and had significant hair growth, as well as three patients who received ruxolitinib and showed biomarker changes that resembled those of healthy controls.[72,73]

In the second open-label research, 66 patients with different types of AA were treated with oral tofacitinib (5 mg twice daily). According to this study, 32% of patients saw a 50% improvement in their Severity of Alopecia Tool (SALT) score after three months of medication, and 64% of patients responded to treatment overall. The normalization of gene expression indicators in scalp biopsy specimens following treatment further supported these findings. During a three-month follow-up period without treatment, patients were reevaluated to determine the durability of response. Just 20 patients, however, were available for follow-up, and all of them had hair loss (median 8.5 weeks after therapy cessation).[74]

ORAL CORTICOSTEROIDS

Systemic corticosteroids are commonly used in the treatment of autoimmune illnesses and have shown promise in the majority of clinical forms of AA, albeit their effectiveness is diminished in cases of ophiasis and alopecia universalis (AU).[75]

In most cases, AA can be treated with a brief course of steroids; however, some studies have reported similar outcomes in individuals treated for three to five months at a dose of 20 to 30 mg per day.4 The effects of pulse steroids, however, might not last long, and many patients experience relapses 4–9 weeks after stopping the medication.22 Steroids typically have adverse consequences that make long-term use impossible. Pituitary-adrenal axis suppression, impacts on bone growth or integrity, ocular abnormalities, and exacerbation of hypertension or diabetes are a few of these.[76]

IMMUNOSUPPRESANTS

Steroid-sparing medications include methotrexate (5–15 mg/week), azathioprine (2–2.5 mg/kg/day), and cyclosporine (2.5–5 mg/kg/day) can be used as stand-alone treatments or in conjunction with systemic steroids. According to accepted practices, every medication should be regularly monitored. Rather than being used as monotherapy to start AA regeneration, these medicines seem to work better when used as steroid-sparing treatments to stop relapses. Regarding which steroid-sparing agent is preferable to another, experts cannot agree on anything.[66]

METHOTREXATE

In a different trial, 14 children with severe AA who had not responded to traditional treatment were studied by Royer et al. (25). A mean dose of 18.9 mg methotrexate (range: 15–25 mg) was administered to these individuals, and 8 out of 14 additionally got oral corticosteroids. Out of the 13 kids who could be evaluated, 5 showed a good response with 50% regrowth (4 of these were also given short-term corticosteroids) and began showing symptoms about 4.4 months later.These findings imply that methotrexate would be a good choice for treating severe and refractory AA.[77]

CYCLOSPORINE:

Cyclosporine inactivates the T-Cell activation and suppresses Interferon – Gamma. It is given orally at a dose of 6 mg/kg/day. Their use is limited because they have high relapse rates and serious side effects.[78]

AZATHIOPRINE:

Azathioprine has actions on Langerhan's cells and CD4/CD8 lymphocytes by their immunosuppressive and immunomodulator effect. The daily dosage of Azathioprine is 2mg/kg/day for 6 months is usually recommended.[66,77,78]

TOPICAL IMMUNOTHERAPY

Topical immunotherapy, such as diphenylcyclopropenone (DPCP) and squaric acid dibutylester (SADBE), induces allergic contact dermatitis and may alter the immune cell milieu surrounding hair follicles by inducing antigenic competition through a mechanism that is yet not fully understood.[79]

Even for pediatric patients [10 years of age], there is evidence to support its usage in severe AA (Fig. 6). Prior to receiving DPCP treatment, patients should be sensitized to a 4 cm circular region using 2% DPCP. You should get DPCP from a pharmacy that has experience compounding it in acetone.Subsequently, 0.001% DPCP is administered unilaterally starting one week later, with the concentration being increased every week until the patient experiences the desired 36-hour erythema and pruritus associated with moderate tolerable dermatitis. The doctor or nurse should administer the patient's effective concentration once it has been identified on a weekly basis.[80]

To avoid light exposure, which breaks down the molecule, DPCP should be kept on the scalp and covered for the full 48 hours following treatment. Both sides are treated once one exhibits a trichogenic response. Patients should be informed that stopping DPCP treatment abruptly increases the risk of relapse. This is because failing to decrease the dose may result in relapses.[79]

BIMATOPROST:

A synthetic prostamide F2a homologue is called bimatoprost. Prostaglandins and prostaglandin analogs differ from prostamides and their structural analogs in terms of structure, pharmacology, and function. With the exception of a double bond at the carbon 13–14 locations rather than a single one, bimatoprost free acid is similar to latanoprost's. The prostamide receptor, which is pharmacologically different from F prostanoid (FP) receptors, is stimulated by bimatoprost to produce its effects. Prostaglandin receptors have been found throughout the hair follicle, especially in the dermal papilla and outer root sheath, where they are thought to play a role in the growth and regeneration of the hair follicle.[81]

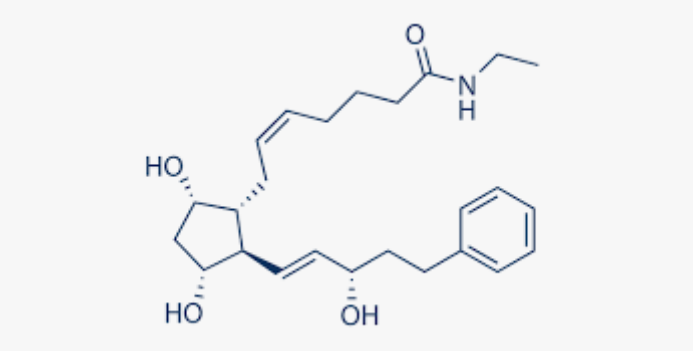

FIGURE-5: Chemical Structure of Bimatoprost[82]

Following the coincidental finding that latanoprost causes eyelash hypertrichosis in glaucoma patients receiving treatment, numerous clinical and animal model studies have been conducted to investigate the therapeutic potential of this approach for alopecia areata.[83] Furthermore, compared to latanoprost, bimatoprost has been shown to induce hypertrichosis earlier and more severely. Compared to latanoprost, bimatoprost does not require conversion into an active metabolite in order to have pharmacological activity, which may account for its

better efficacy. Furthermore, rather than discussing full hair restoration, Zaher et al. have merely commented on the proportion of hair regrowth.[84]

The majority of patients tolerated latanoprost well; the sole side effect was erythema at the application site. Because vasodilatation acts on PGF2α receptors on dermal vessels, this is linked to it. Numerous more investigations have shown that the only unfavorable effect at the latanoprost-treated locations is an erythematous reaction, or that there are no side effects at all. This contrasts with the steroid treatment, which has a number of negative side effects, including modest local atrophy, telangiectasia, pustules, and acneiform facial eruptions.[85]

Eyelash alopecia areata was initially treated with topical latanoprost. Dermal papilla and outer root sheath prostaglandin F2α receptors are expressed by eyelash hair follicles. By binding to these receptors, latanoprost causes the telogen follicles to enter the anagen phase. Additionally, it lengthens the hair cycle's anagen phase.[84]

In 2001, bimatoprost was first authorized for the treatment of ocular hypertension and open-angle glaucoma. Clinical investigations revealed eyelash growth as an adverse event (AE), which prompted assessments of eyelash hypotrichosis therapy options.7–10 In adults with idiopathic and chemotherapy-induced eyelash hypotrichosis, once-nightly application of bimatoprost 0.03% to the upper eyelid margin enhanced eyelash length, thickness, and blackness compared with vehicle.[81,82]

In 2008, bimatoprost ophthalmic solution 0.03% was authorized for the treatment of adult eyelash hypotrichosis. By conducting this study, the Food and Drug Administration satisfies a postmarketing obligation to furnish information regarding the safety of bimatoprost 0.03% in treating eyelash hypotrichosis in children. [83,85]

Zaher et al. recently reported on the first randomized controlled trial investigating the use of bimatoprost in the treatment of scalp AA (4). Even though mometasone furoate cream and topical bimatoprost were shown to be equally effective, the patches treated with the former showed a higher percentage of hair regrowth, quicker response, less resistance, and less relapse.[84]

FDA Approved Indications of Bimatoprost: [81,83]

1. Eyelash hypotrichosis

Other off label uses:

2. Eyebrow hypotrichosis

3. Androgenetic alopecia

4. Alopecia areata (AA) Others with minimal evidence

5. Vitiligo

The prostaglandin F2 alpha analog bimatoprost has been shown to stimulate hair development by raising the proportion of anagen follicles, encouraging telogen hair follicles to re-enter the anagen phase, and enlarging hair bulb diameter. Moreover, melanogenesis is increased and human melanocyte dendricity is potently stimulated upon activation of the prostaglandin F receptor.[81,82]

Conjunctival hyperemia, ocular discomfort, iris pigmentation, periorbital pigmentation, and fat atrophy were the most commonly reported side effects of using bimatoprost solution for ophthalmic disorders and eyelash hypotrichosis. In contrast to ocular disorders, bimatoprost treatment for eyebrows was linked to sporadic complaints of skin irritation, pruritis, and mild

skin pigmentation, all of which disappeared after the medication was stopped. There were extremely few side effects recorded in the current trial.[81,83]

Complications of Alopecia Areata

The following are a few possible alopecia areata complications:[86,87]

- Anxiety and depression, sunburn, irregular nails, recurrence, irreversible hair loss, and skin damage.
- Variable hair regrowth in terms of pattern, texture, and rate.
- A higher chance of systemic and dermatological conditions such vitiligo, psoriasis, thyroid disease, lupus erythematosus, and atopic dermatitis coexisting.
- Medication side effects such as thrombosis, dermatitis, infections, cancer, and skin atrophy.
- A three-fold increased risk of retinal disorders, including retinopathy, retinal vascular occlusion, and retinal detachment.

FRACTIONAL CO2 LASER

To increase the amount of medication that reaches the intended tissue, TED can be utilized in combination with mechanical, chemical, or physical methods. An ablative non-fractional device was used in the 1987 publication of the first report on the usage of TED lasers. Fractional lasers were first introduced for TED in 2004 by Manstein et al. The MTZ channels produced by fractional lasers provide uniform and regulated drug delivery. The consistent dispersion of the channels throughout the target area results in homogeneous drug deposition. Fractional lasers can penetrate the dermis up to 2-3 mm and deposit heat energy where the dermal papilla resides, which is the location where the capillaries wrap the hair germ cells.[88]

TED is a technique that can be used in conjunction with mechanical, chemical, or physical approaches to maximize the penetration of medicines into the target tissue. The first report of TED laser use was published in 1987, utilizing an ablative non-fractional device. Manstein et al. introduced fractional lasers for TED in 2004.Drug delivery can be made consistent and controlled thanks to the channels created by fractional lasers' creation of MTZ. The drug deposition becomes homogeneous as the channels are dispersed uniformly throughout the target area. Fractional lasers can deposit heat energy where the dermal papilla is, which is the area where the capillaries encircle the hair germ cells, by penetrating the dermis up to 2-3 mm.

Fractional laser therapy has been proposed as a means of encouraging telogen to anagen transitions, enhancing blood flow, and triggering T cell apoptosis.[89]

Compared to needle injection, this innovative method of delivering triamcinolone using a fractional CO2 laser as TED system allows for more uniform drug delivery in the target area, which promotes a more favorable therapeutic response. Furthermore, by switching to fractional laser injections rather than several intradermal pricks, the discomfort connected with repetitive injections can be avoided. A potential benefit of utilizing fractional lasers could be the decreased occurrence of skin atrophy while using topical triamcinolone.[90]

It is assumed that the dermal heat generated by fractional laser therapy will promote hair growth. Fractional lasers have a direct therapeutic impact, but they also work by delivering the topical steroid into the hair follicle through transepidermal drug delivery (TED). This characteristic of fractional lasers is commonly referred to as "laser-assisted drug delivery," and it has been used to treat a variety of dermatological conditions, including lichen planus, burn scars, and skin cancers. Dermatological therapy has introduced a novel concept:

fractional laser-assisted medication delivery of corticosteroids for refractory alopecia areata.[88,89,90]

MECHANISM OF ACTION OF FRACTIONAL CO2 LASER:

The stimulation of T cell apoptosis, stopping hair follicles in the telogen stage and stimulating anagen stage and denovo neogenesis of hair follicles from non-hair follicle stem cells are considered to be the mechanisms of action of fractional CO2 laser in the treatment of AA.

Anagen inductions may also be connected to the Wnt/β-catenin signaling pathways that have been observed following fractional laser in a study using a mouse model.[91]

In the meantime, heat-induced laser stimulation of the papillary dermis promotes hair growth. Moreover, fractional laser-generated microthermal therapy zones can offer routes for consistent and regulated transepidermal drug delivery of conventional topical drugs like corticosteroids.[92,93,94]

FIGURE-6: FRACTIONAL CO2 LASER

METHODOLOGY

MATERIALS & METHODS

Source of data:

This study will be conducted in outpatient clinic of Dermatology, Venereology and Leprosy in R L Jalappa Hospital and Research Centre attached to Sri Devaraj URS Medical College, Tamaka, Kolar from **October 2022 to March 2024.**

Study Design: Randomized Controlled Trial [RCT]

Sample size calculation:

- Sample Size is calculated based on the proptional value, the proportional value in group 1 as 56.7 and in group 2 as 83.3 from the research article Bimatoprost versus Mometasone Furoate in the Treatment of Scalp Alopecia Areata: A Pilot Study
- With the confidence interval of 90% and with power 80% the sample size has been calculated by using OPEN EPI data version 3.01
- Two-sided confidence interval (1-alpha) = 90
- Power (chance of detecting) = 80
- Ratio of two groups= 1:1
- Hypothetical proportion of group 1 with exposure =56.7
- Hypothetical proportion of group 2 with exposure =83.3
- Sample size of group 1= 37
- Sample size of group 2= 37
- Total sample size = 74
- Reference

  Kelsey et al; Methods in Observational Epidemiology 2nd Edition, Table 12-15

Statistical Analysis:

Data will be entered into a Microsoft Excel Data Sheet and will be analysed using SPSS 27 version software. Categorical data will be represented in the form of Frequencies and proportions. Chi-square will be used as test of significance. Continuous data will be represented as mean and standard deviation. p value <0.05 will be considered as statistically significant.

Method of collection of data (including sampling procedure)

Inclusion criteria:

- Patients with Localized Alopecia Areata over any part of the body.

Exclusion criteria:

- Patients with other systemic illness – cardiac, renal, hepatic illness,
- Severely Immunocompromised
- Pregnancy.
- Patients taking photosensitive drugs and photosensitivity
- Patients <10 years of age.
- Patient not willing for study

- **Randomization**: Computer generated block randomisation.

A Randomization Plan

from

http://www.randomization.com

1. A
2. B
3. B
4. A
5. B
6. B
7. A
8. A
9. B
10. B
11. B
12. A
13. B
14. A
15. A
16. B
17. A
18. B
19. A
20. A
21. A
22. B
23. B
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27. B
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67. B
68. A
69. B
70. A
71. A
72. B
73. B
74. B
75. B
76. A

To reproduce this plan, use the seed 14136
along with the number of subjects per block/number of blocks
and (case-sensitive) treatment labels as entered originally.

Methods of data collection:

All patients satisfying the inclusion criteria will be divided into two groups as follows:

GROUP A- participants were treated with Fractional CO2 Laser in combination with Bimatoprost 0.03% within 2 minutes immediately after laser for 5 sittings with an interval gap of 3 weeks with topical Bimatoprost 0.03% once daily application between each sitting.

GROUP B- participants were treated with Fractional CO2 Laser in combination with Topical Triamcinolone acetonide Aqueous solution (2.5 mg/ml to 10 mg/ml) within 2 minutes immediately after laser for 5 sittings with an interval gap of 3 weeks between each sitting.

Group A - A topical anaesthetic, containing a mixture of lidocaine-2.5% w/w + prilocaine-2.5% w/w in a cream base was applied for 1 hour on the treatment area. After satisfactory anaesthesia is achieved, the treatment area was cleaned with a mild cleanser. Eyes were protected with eye shields.

Fractional CO2 LASER was then delivered to the Alopecia Areata site at the fluence of 50-60 mJ/cm2(trans epidermal pores are created in the skin). Ice pack were applied immediately. Then within 2 minutes of LASER procedure, Topical Bimatoprost 0.03% was applied and asked to continue twice daily application till next sitting.

GROUP B – A topical anaesthetic, containing a mixture of lidocaine-2.5% w/w + prilocaine-2.5% w/w in a cream base was applied for 1 hour on the treatment area. After satisfactory anaesthesia is achieved, the treatment area was cleaned with a mild cleanser. Eyes were protected with eye shields.

Fractional CO2 LASER was then delivered to the Alopecia Areata site at the fluence of 50-60 mJ/cm2 (trans epidermal pores are created in the skin). Ice pack were applied immediately.

Then within 2 minutes of LASER procedure, Topical Triamcinolone Acetonide Aqueous solution (2.5 mg/ml to 10 mg/ml) was applied.

Participants were required to undergo serial photography at baseline and at subsequent sittings.

- Lighting and positioning was kept identical for all serial photographs.
- The serial photographs were assessed independently by a blinded third observer.
- The third observer gave the grading of the efficacy of the treatment modality based on the **Global Photograph Assessment (GPA)scale.**
- A score of 0, 1, 2 and 3 was given if the response was <25%, 25-50%, 51-75% and >75%, respectively by the third observer.
- **LAD Score (Lesional Area and Density Score)** of lesions was calculated at baseline and at subsequent follow up sittings.
- (LAD) score calculation is done by the following steps: LAD score is used for determination of the response to a given treatment, for example, in the assessment of response to intralesional steroids, the area(s) of alopecia areata is clearly demarcated from the surrounding hair. If the margins of the areas are instead less clear, the edges of the alopecia areata patch is marked directly on the scalp and photograph is taken so that there is a record of which margins were used at the initial assessment.
- The area of each patch is determined by multiplying the long axis by its perpendicular axis. If the patch of alopecia is totally circular, πr^2 will be used.
- The LAD Score is calculated by the formula:

LAD Score = Area of alopecia areata site x Density of hair loss

- LAD Score was calculated for all the participants of the study at the baseline presentation and at subsequent follow up sittings. The **percentage of improvement in LAD** was assessed at each sitting with the baseline presentation.

- To assess the safety of both interventions and to document the post procedural adverse effects of both interventions, following parameters were taken in to consideration:

- The **visual discomfort scale** (scoring from 0 to 10) was used to record the subjectory discomforts of the participants such as pain, burning sensation and any other peculiar discomforts following the procedure at each sitting.

- Serial photographs were taken to document the objectory post procedural adverse effects such as erythema, atrophy of skin, edema, post inflammatory hypo/hyperpigmentation and any other peculiar features and it were assessed independently by a blinded third observer.

- The **visual analogue scale** (scoring from 0 to 10) was used to record the satisfaction of the treatment modality as perceived by participants at the end of 5 sittings.

RESULTS

RESULTS

Table 1: Association of gender between two groups

Gender	Group A	Group B	Total	Test statistic	p value
Female	14(37.8)	14(37.8)	28(37.8)	0.000	1.000
Male	23(62.2)	23(62.2)	46(62.2)		
Total	37(100.0)	37(100.0)	74(100.0)		

Statistical test used: Chi Square test; n (%) is reported
*p value <0.05 is considered statistically significant

The study consists of 37 study participants in group A and group B. It is observed that there is equal distribution among females (37.8%) and males (62.2%) in group A and group B (p value =1.00). Overall, 46(62.2%) were males and 28 (37.8%) were females (figure 1).

Graph 1: Pie chart depicting gender wise distribution

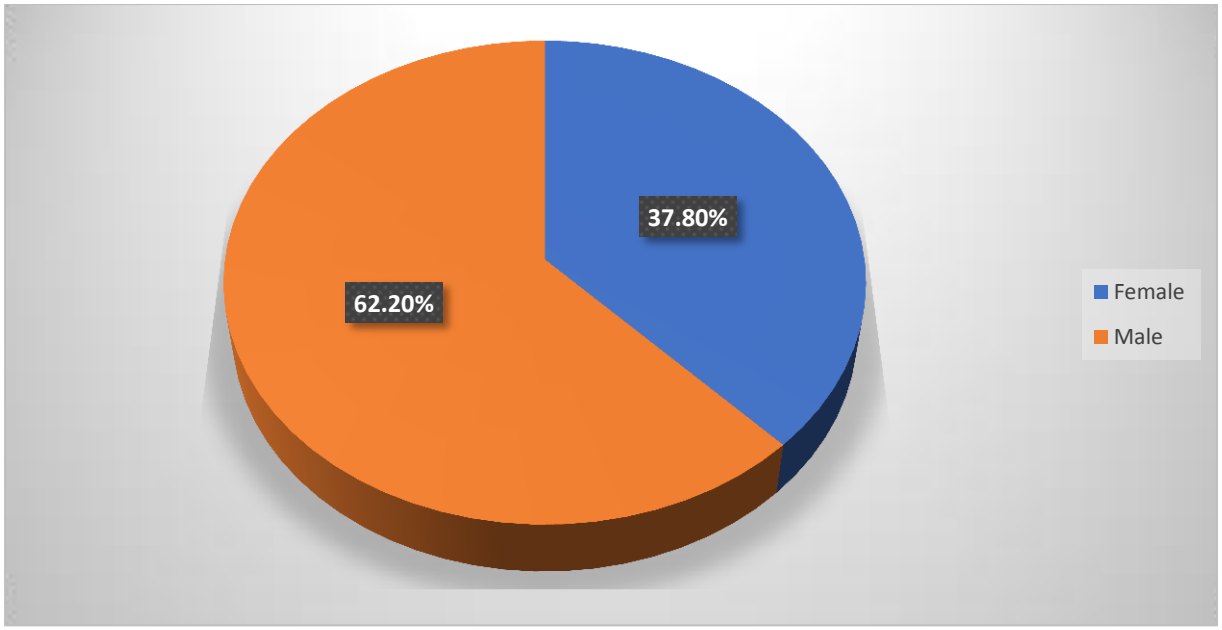


Table 2: Comparison of age between two groups

	Group A	Group B	Test statistic	p value
Age (in years)	29.973 ± 8.513	28.216 ± 9.396	0.843	0.402

Statistical test used: Independent sample t test

*p value <0.05 is considered statistically significant

The mean age of study participants in group A was 29.973 ± 8.513 years and group B was 28.216 ± 9.396 years with minimum age being 13 years and maximum age being 51 years. The study shows that there is no significant different difference in age between two groups. (p value =0.402)

Graph 2: Bar diagram depicting age between two groups

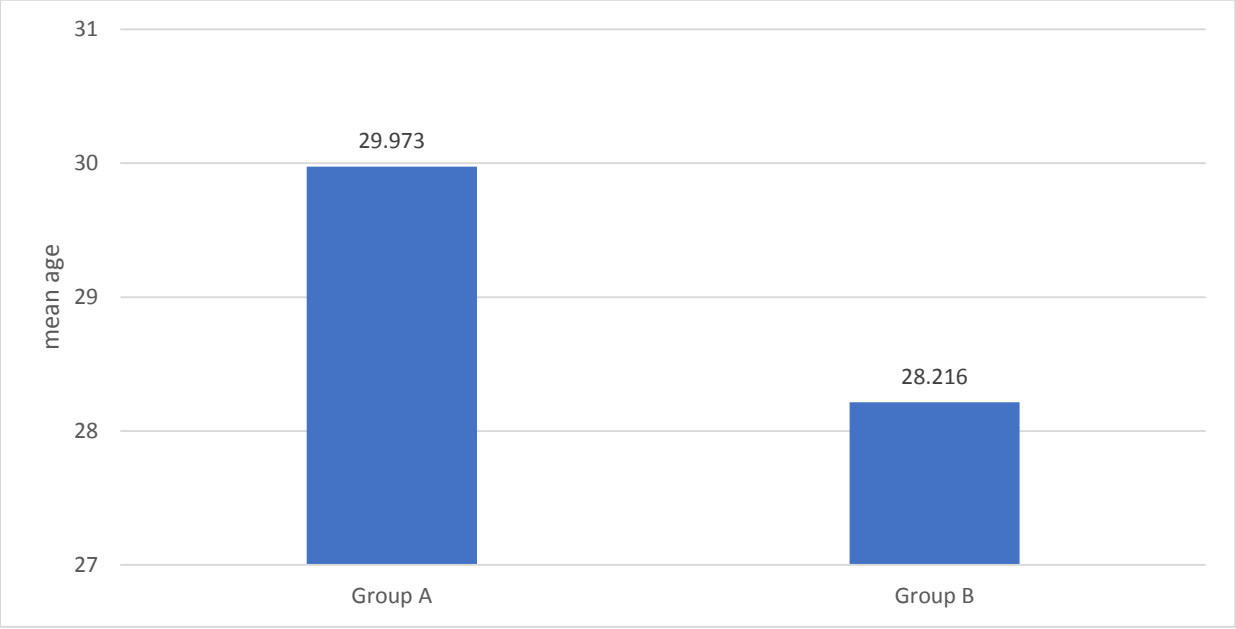


TABLE – 3: DISTRIBUTION OF CASES ACCORDING TO AGE GROUPS

AGE	GROUP – A	GROUP – B	TOTAL
10-18 YEARS	1	6	7
	1.3 %	8.1%	9.4 %
19 – 30 YEARS	22	18	40
	29.7%	24.3 %	54.0 %
31 – 40 YEARS	8	9	17
	10.8%	12.1%	22.9 %
41 – 50 YEARS	6	3	9
	8.1%	4.0%	12.1 %
> 50 YEARS	0	1	1
	0%	1.3%	1.3%
TOTAL	37	37	74
	100 %	100 %	100 %

GRAPH – 3: GRAPH SHOWING DISTRIBUTION OF CASES ACCORDING TO AGE GROUPS

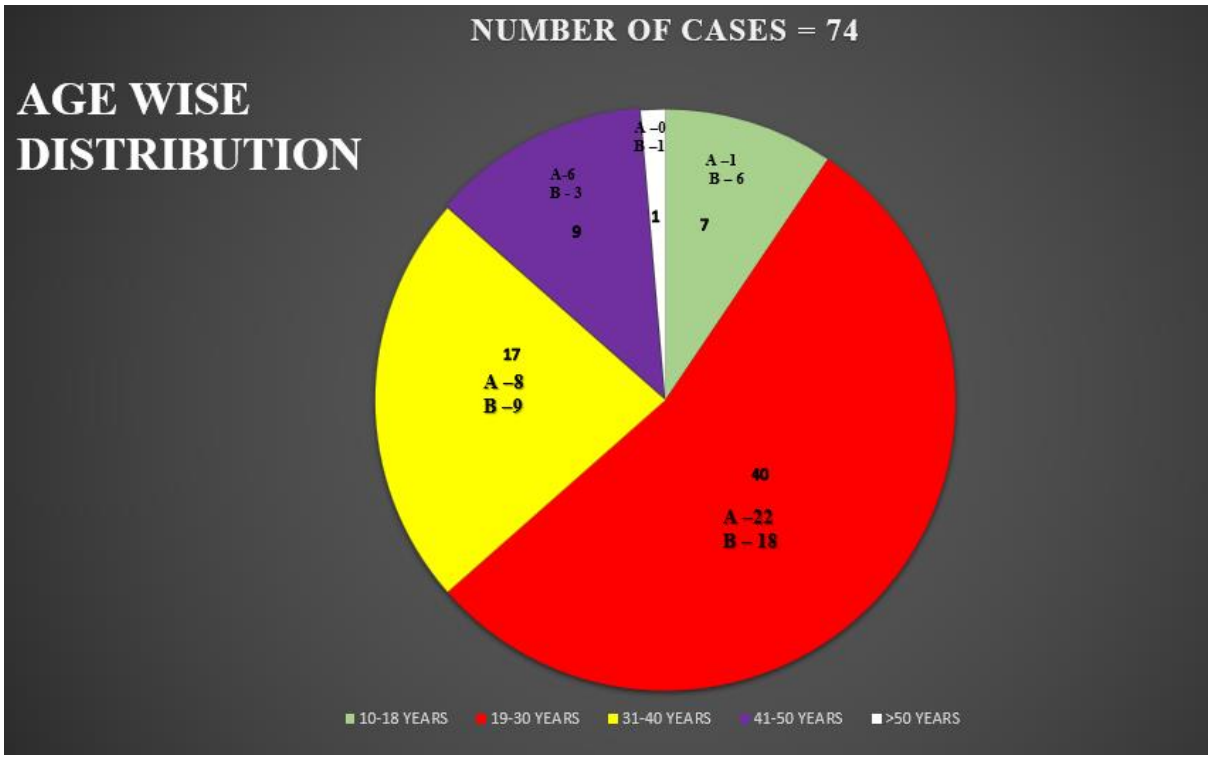


TABLE-4: DISTRIBUTION OF CASES BASED ON ASSOCIATED SYMPTOMS

ASSOCIATED SYMPTOMS	GROUP – A	GROUP – B	TOTAL
ASYMPTOMATIC	35	33	68
	94.59 %	89.18 %	91.89%
ITCHING	2	4	6
	5.4%	10.8%	8.10 %

GRAPH – 4: GRAPH SHOWING DISTRIBUTION OF CASES BASED ON ASSOCIATED SYMPTOMS

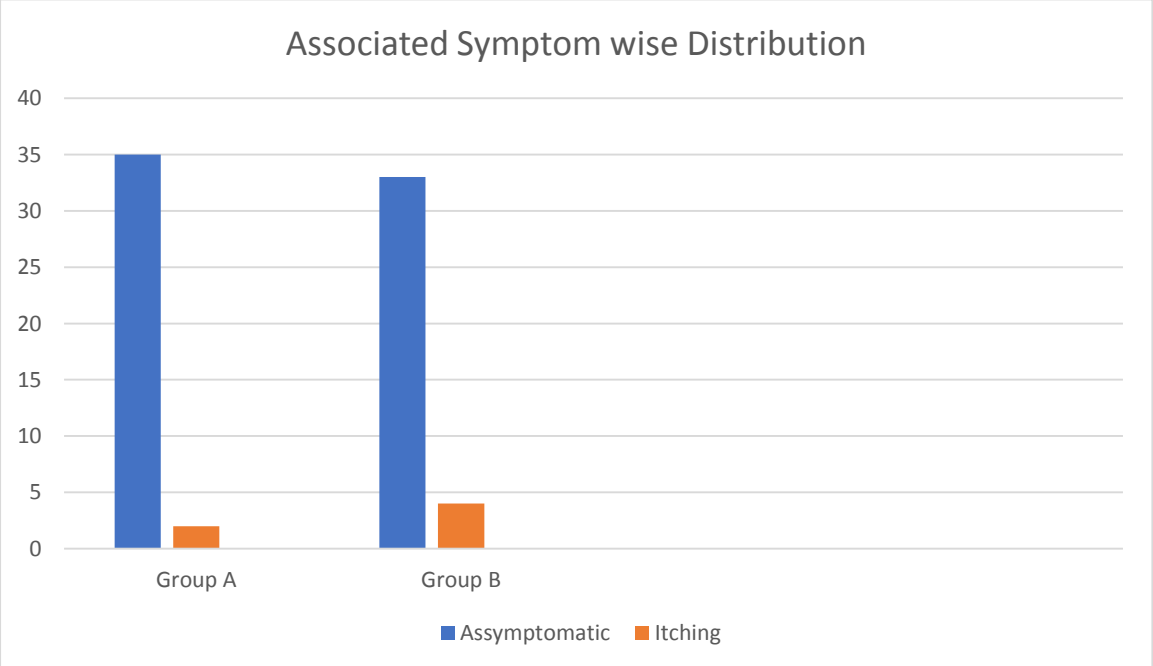


TABLE-5: DISTRIBUTION OF CASES BASED ON REASONS FOR TREATMENT

REASON FOR TREATMENT	GROUP – A	GROUP – B	TOTAL
COSMETIC REASONS	30	27	57
	81.08 %	72.97%	77.02 %
THERAPEUTIC BENEFIT	7	10	17
	18.91%	27.02%	22.97 %

GRAPH – 5: GRAPH SHOWING DISTRIBUTION OF CASES BASED ON REASONS FOR TREATMENT

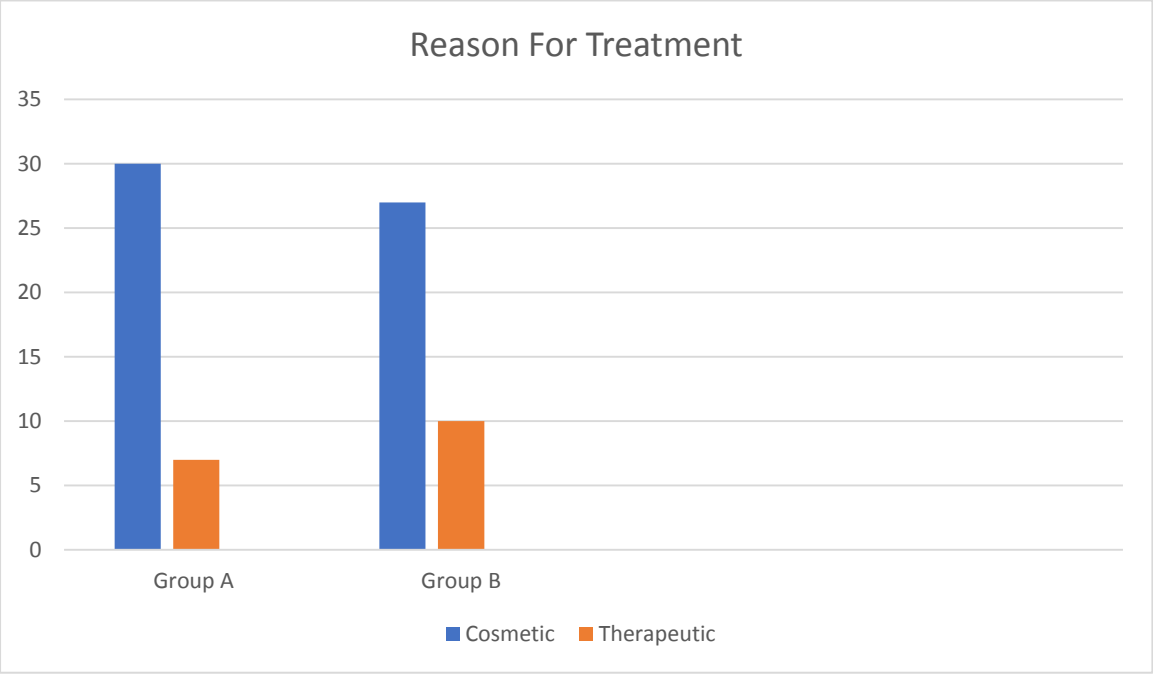


TABLE – 6: DISTRIBUTION OF CASES BASED ON THE SITE OF AA PATCHES

SITE OF AA PATCHES	GROUP – A	GROUP – B	TOTAL
SCALP	35	31	66
	94.5 %	83.7 %	89.1%
BEARD	1	5	6
	2.7 %	13.5 %	8.1 %
MOUSTACHE	1	1	2
	2.7 %	2.7 %	2.7 %
EYEBROW / ANY OTHER PART OF BODY	-	-	-
	-	-	-

GRAPH – 6: GRAPH SHOWING DISTRIBUTION OF SITES OF AA PATCHES

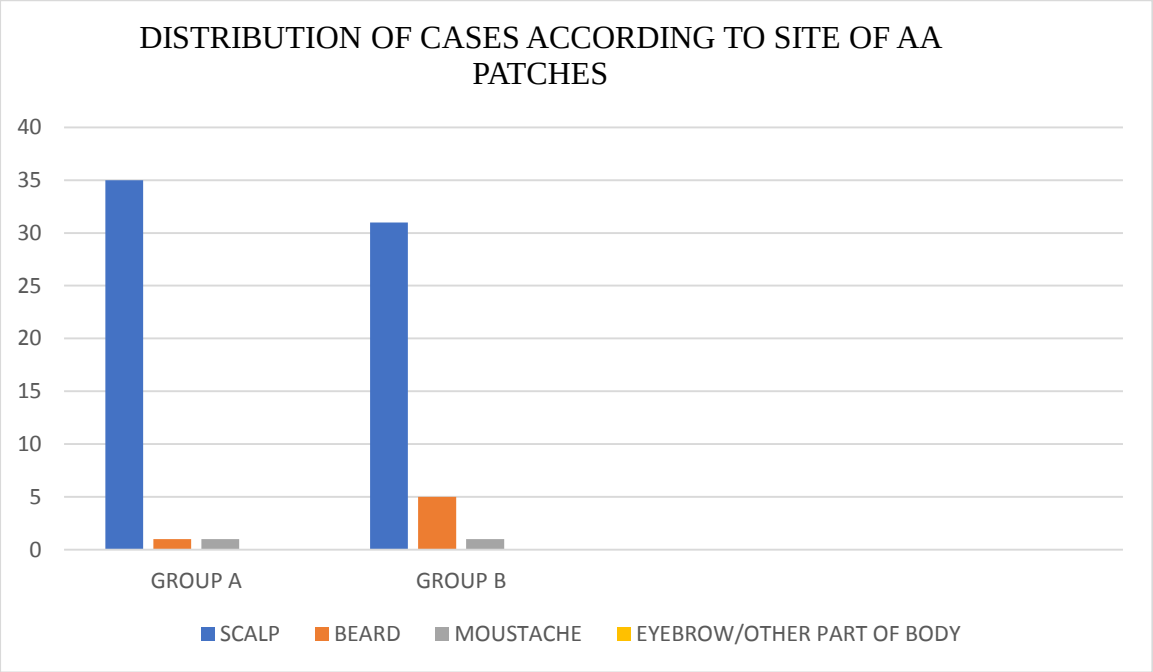


Table-7: Comparison of LAD score between two groups

LAD Score	group	N	Mean	SD	Percentiles			Test statistic	p value
					25th	50th	75th		
Baseline	Group A	37	12.554	8.144	8.9	9.2	15.9		
	Group B	37	19.970	8.856	15.9	16.6	25.2	341.5	0.0001*
1st setting	Group A	37	9.836	6.462	6.9	7.1	12.6		
	Group B	37	14.415	6.578	11.4	11.7	18.2	441	0.009*
2nd setting	Group A	37	6.819	4.563	4.8	4.9	8.6		
	Group B	37	10.43	4.897	8.1	8.6	13.2	394	0.002*
3rd setting	Group A	37	4.219	2.71	2.9	3.2	5.4		
	Group B	37	8.589	4.014	6.7	7.2	10.7	252	0.0001*
4th setting	Group A	37	2.217	1.357	1.5	1.7	2.4		
	Group B	37	6.345	2.811	4.9	5.7	7.7	177.5	0.0001*
5th setting	Group A	37	0.653	0.638	0.2	0.45	0.8		
	Group B	37	4.870	2.104	3.6	4.3	6.5	28.5	0.0001*

Statistical test used: Mann Whitney U test
*p value <0.05 is considered statistically significant

The study shows that average LAD score was higher (median =16.6) in group B compared to group A (median = 9.2) at the baseline. In the 1st setting average LAD score was 7.1 in group A and 11.7 in group B (p value =0.009). In the 1st setting average LAD score was 7.1 in group A and 11.7 in group B (p value =0.002) and in 2nd setting average LAD score was 8.6 in group B and 4.9 in group A (p value = 0.003). It is observed that 3rd, 4th and 5th setting there is a dip in LAD in both the groups and difference is statistically significant (p value =0.0001). The study shows that Group A consistently has lower LAD compared to Group B. In general, there is decreasing trend in LAD score from baseline to 5th settings in both the groups.

GRAPH-7 : Line diagram depicting LAD score between two groups

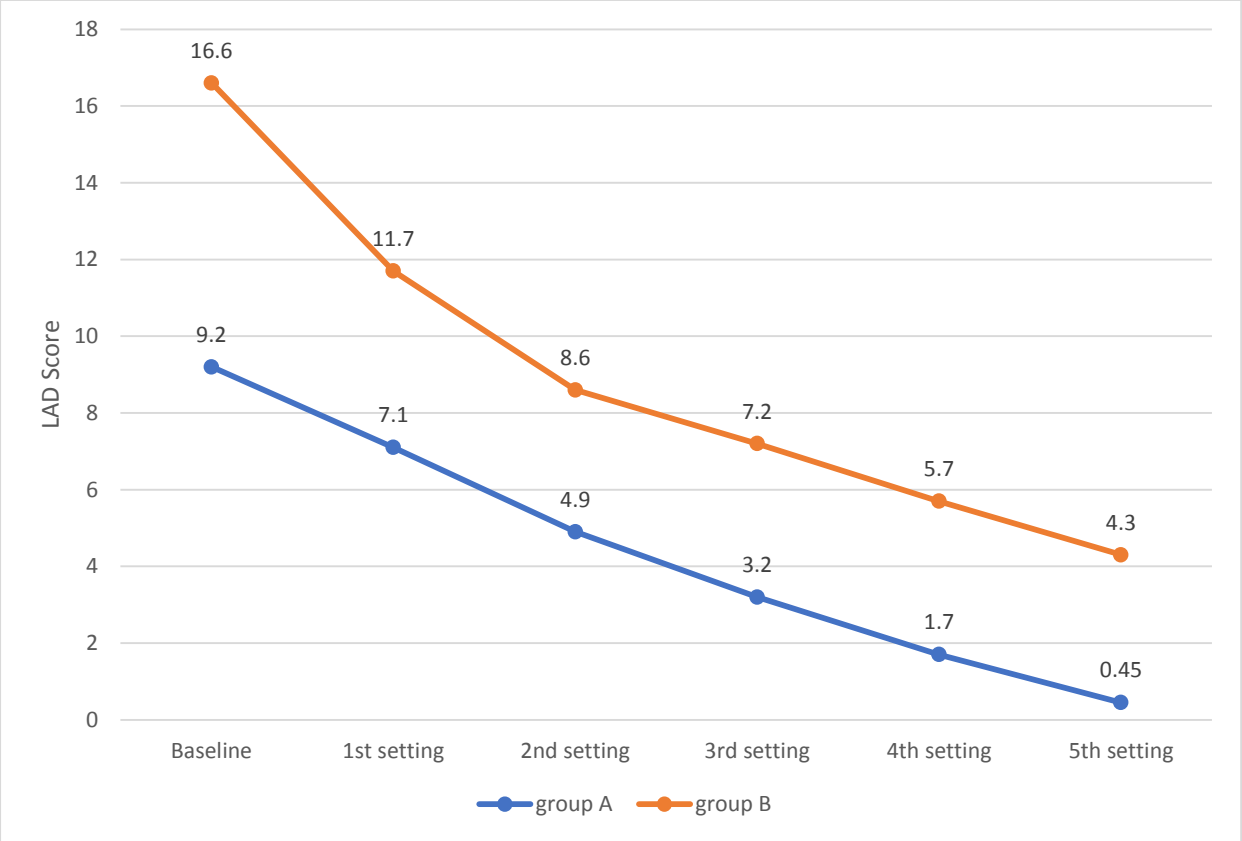


Table-8: Comparison of percentage LAD improvement between two groups

percentage LAD improvement	group	N	Mean	SD	Percentiles			Test statistic	p value
					25th	50th	75th		
1st setting	Group A	37	21.949	2.987	20	22.472	23.81	71	0.0001*
	Group B	37	28.505	2.711	27.424	28.916	29.762		
2nd setting	Group A	37	46.324	3.706	45	46.914	48.298	419.5	0.004*
	Group B	37	48.391	2.391	46.988	48.133	50.307		
3rd setting	Group A	37	66.678	2.686	64.835	66.304	69.167	1	0.0001*
	Group B	37	57.399	2.428	55.556	57.708	59.639		
4th setting	Group A	37	82.032	2.601	80.881	81.915	84.043	0	0.0001*
	Group B	37	68.298	1.662	67.901	68.817	69.524		
5th setting	Group A	37	95.344	1.629	94.186	95.109	96.774	0	0.0001*
	Group B	37	75.641	1.776	74.214	74.843	77.143		

Statistical test used: Mann Whitney U test
*p value <0.05 is considered statistically significant

At the 1st setting and 2nd setting, Group B (28.916%, 48.133%) shows a higher improvement compared to group A (22.472%, 46.914%) and the difference is statistically significant (p value =0.0001). At 3rd, 4th and 5th setting LAD improvement was higher in group A (66.604%, 81.915% and 95.109%) compared to group B (57.708%, 68.817% and 74.843%) and the difference is statistically significant (p value =0.0001). Overall, at initial setting Group B showed higher improvement and later settings Group A showed higher improvement. The differences between groups across settings are statistically significant.

GRAPH-8: Line diagram depicting percentage LAD improvement

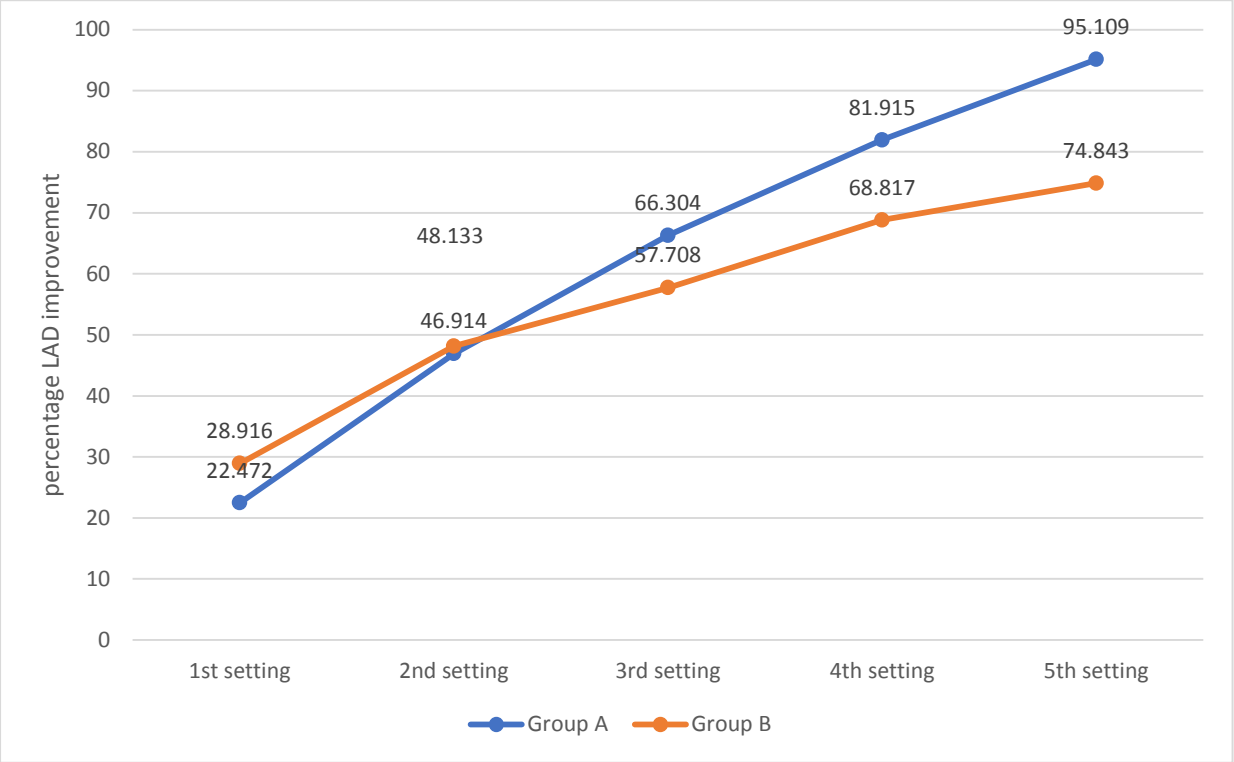


Table-9: Efficacy of the treatment modality based on GPA scale

GPA		Group A (n=37)	Group B (n=37)	Total (n=74)
1ST setting	<25%	9(24.3%)	0	9(12.2%)
	25-50%	28(75.7%)	37(100.0)	65(87.8%
2nd setting	25-50%	33(89.2%)	20(54.1)	53(71.6%)
	51-75%	4(10.8%)	17(45.9%)	21(28.4%)
3rd setting	51-75%	30(81.1%)	37(100.0)	67(90.5%)
	>75%	7(18.9%)	0	7(9.5%)
4th setting	25-50%	0	1(2.7%)	1(1.4%)
	51-75%	1(2.7%)	35(94.6%)	36(48.6%)
	>75%	36(97.3%)	1(2.7%)	37(50.0%)
5th setting	51-75%	0	21(56.8%)	21(28.4%)
	>75%	37(100.0)	16(43.2%)	53(71.6%)

n(%) is reported

The efficacy the treatment modality based on GPA scale is markers as 25%, 25-50%, 51-75% and >75%. At first setting in group A, 9(24.3%) and in group B no participants had GPA improvement less than 28%. In 2nd setting higher proportion showed an improvement of 51 to 75% in group B (45.9%) compared to group B (10.8%). Whereas at 3rd setting 18.9% in group A and in group B none had improvement above 75%. At 4th setting only 1(2.7%) had improvement in group B and 97.3% had improvement in group A. At 5th setting 100% had >75% improvement in group A and only 43.2% in group B. Overall, Group A shows higher GPA response in the 3rd, 4th and 5th setting and group B trends to have study participants at the range of 25 to 75%.

Table-10: Comparison of GPA score between two groups

GPA	group	N	Mean	SD	Percentiles			Test statistic	p value
					25th	50th	75th		
1st setting	Group A	37	0.757	0.435	1	1	1	518	0.002*
	Group B	37	1	0	1	1	1		
2nd setting	Group A	37	1.108	0.315	1	1	1	444	0.0001*
	Group B	37	1.459	0.505	1	1	2		
3rd setting	Group A	37	2.189	0.397	2	2	2	555	0.006*
	Group B	37	2	0	2	2	2		
4th setting	Group A	37	2.973	0.164	3	3	3	36.5	0.0001*
	Group B	37	2	0.236	2	2	2		
5th setting	Group A	37	3	0	3	3	3	296	0.0001*
	Group B	37	2.432	0.502	2	2	3		

Statistical test used: Mann Whitney U test
*p value <0.05 is considered statistically significant

The study shows that group A shows higher GPA response in the 3rd, 4th and 5th setting compared to group B and the difference is found to be statistically significant (p value < 0.05)

GRAPH-9: Line diagram depicting GPA

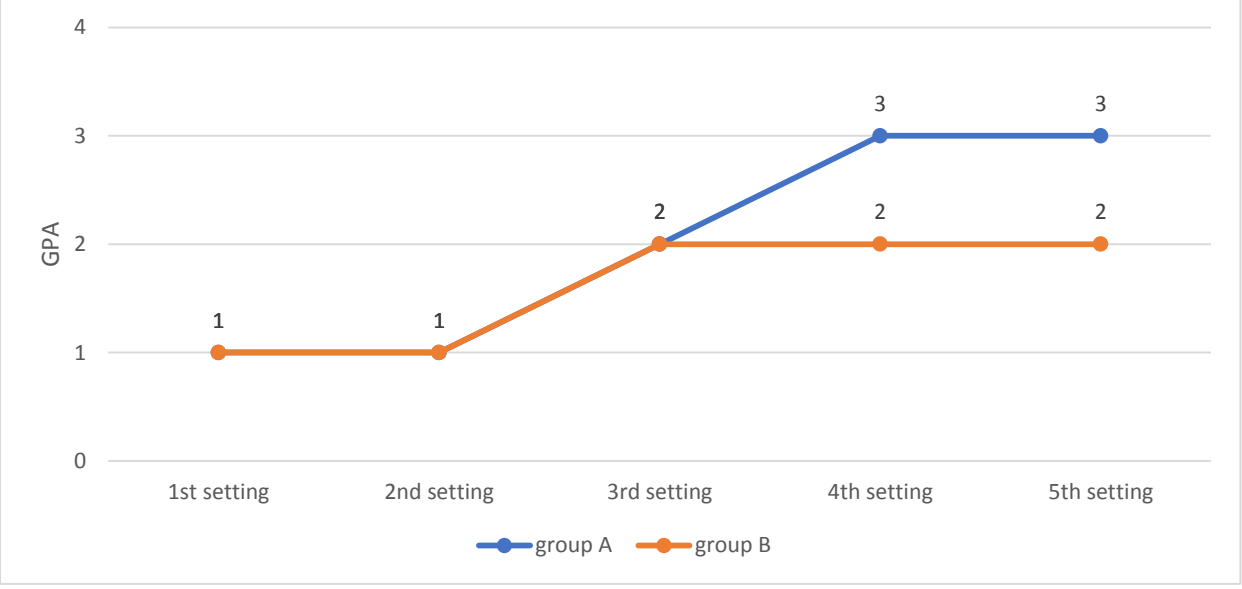


Table-11: Comparison of VDS and VAS score between two groups

	Group	N	Mean	SD	Percentiles			Test statistic	p value
					25th	50th	75th		
VDS	Group A	37	4.27	1.018	3	4	5	585.5	0.266
	Group B	37	4.027	0.957	3	4	5		
VAS	Group A	37	7.703	0.968	7	8	9	653.5	0.727
	Group B	37	7.622	0.924	7	7	8		

Statistical test used: Mann Whitney U test
*p value <0.05 is considered statistically significant

The study shows that there is no significant difference in VDS and VAS score between the group A and group B.

GRAPH-10: Bar diagram depicting VDS and VAS score between two groups

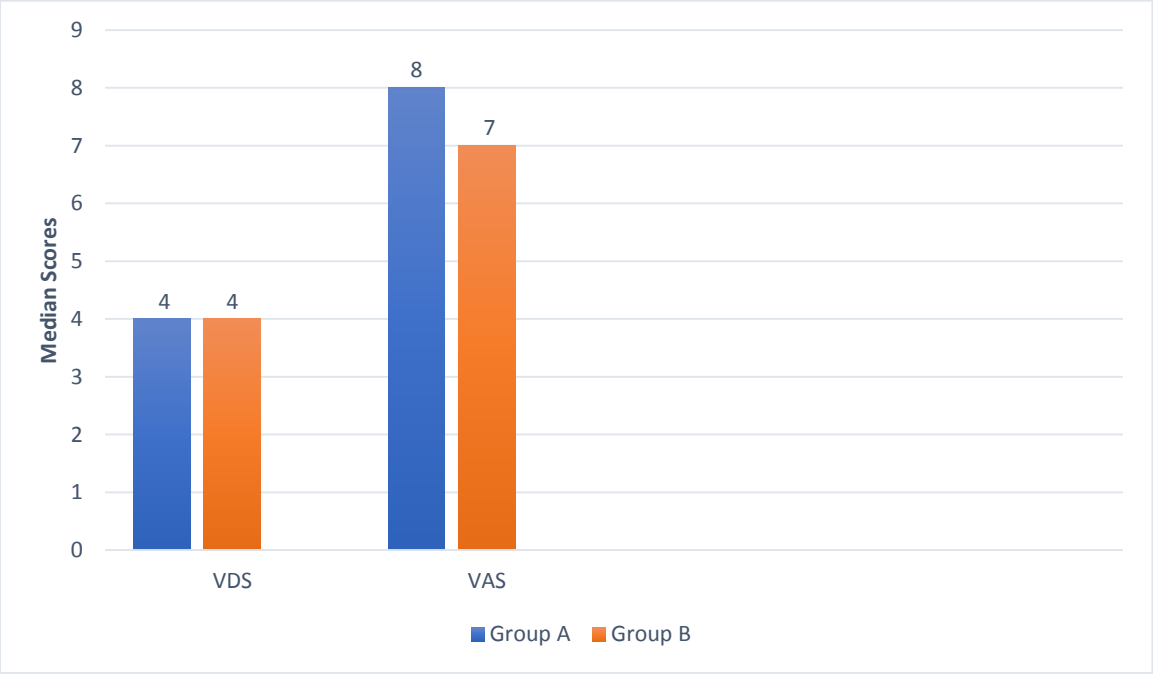


Table-12: Association of adverse effect of Erythema between two groups

ER	Group A	Group B	Total	Test statistic	p value
Present	13(35.1)	9 (24.3)	22(29.7)	1.035	0.309
Absent	24(64.9)	28(75.7)	52(70.3)		
Total	37(100.0)	37(100.0)	74(100.0)		

Statistical test used: Chi Square test; n(%) is reported
*p value <0.05 is considered statistically significant

The study shows that ER was observed among 13(35.1%) in group A and 9(24.3%) in group B and the association is not found to be statistically significant (p value =0.309)

Table-13: Association adverse effect of Edema between two groups

ED	Group A	Group B	Total	Test statistic	p value
Present	5(13.5)	9(24.3)	14(18.9)	1.410	0.235
Absent	32(86.5)	28(75.7)	60(81.1)		
Total	37(100.0)	37(100.0)	74(100.0)		

Statistical test used: Chi Square test; n (%) is reported
*p value <0.05 is considered statistically significant

The study shows that ED was observed among 5(13.5%) in group A and 9(24.3%) in group B and the association is not found to be statistically significant (p value =0.235)

Table-14: Association of adverse effect of Atrophy between two groups

AP	Group A	Group B	Total	Test statistic	p value
Present	0	6(16.2)	6(8.1)	---	----
Absent	37 (100.0)	31(83.8)	68(91.9)		
Total	37(100.0)	37(100.0)	74(100.0)		

Statistical test used: Chi Square test; n (%) is reported
*p value <0.05 is considered statistically significant
The study shows that none in group A had AP and 6(16.2%) in group B had AP

Table-15: Association of adverse effect of Post Inflammatory Hyperpigmentation between two groups.

PIH	Group A	Group B	Total	Test statistic	p value
Present	3(8.1)	6(16.2)	9(12.2)	1.138	0.286
Absent	34(91.9)	31(83.8)	65(87.8)		
Total	37(100.0)	37(100.0)	74(100.0)		

Statistical test used: Chi Square test; n(%) is reported
*p value <0.05 is considered statistically significant

The study shows that PIH was observed among 3(8.1%) in group A and 6(16.2%) in group B and the association is not found to be statistically significant (p value =0.286)

GRAPH-11: Bar diagram depicting Adverse effect between the two groups

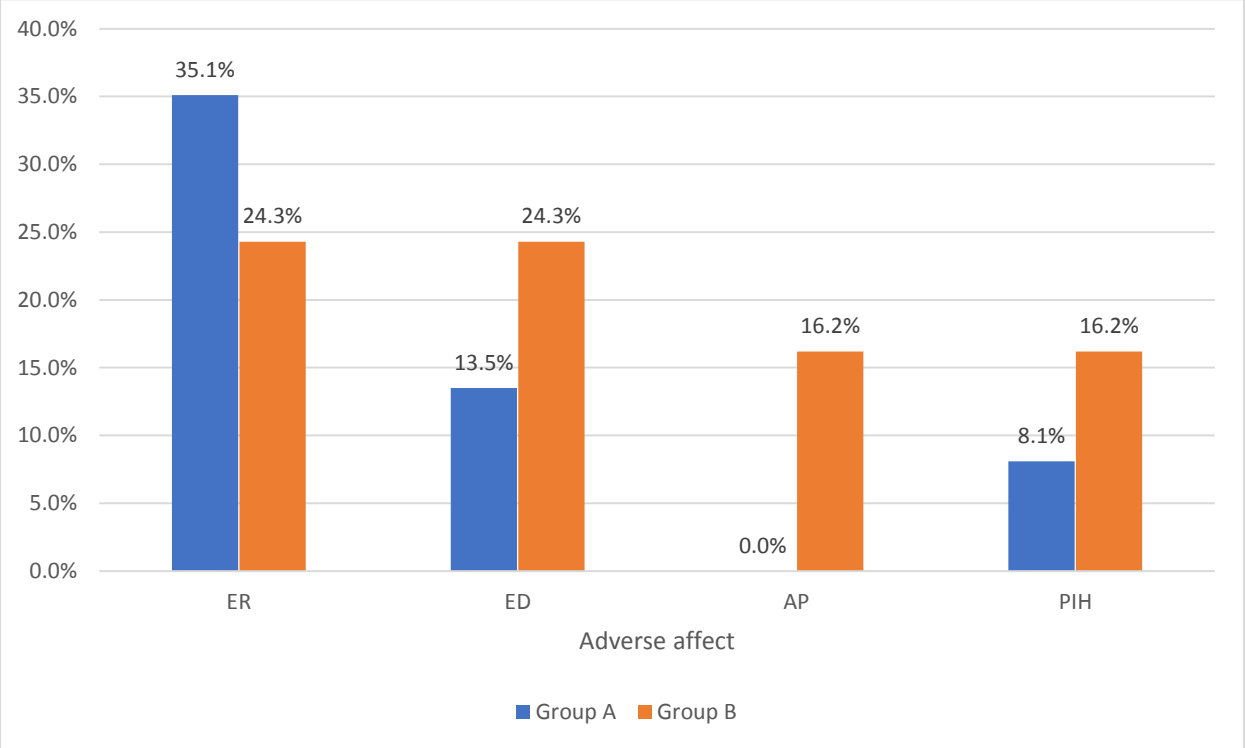
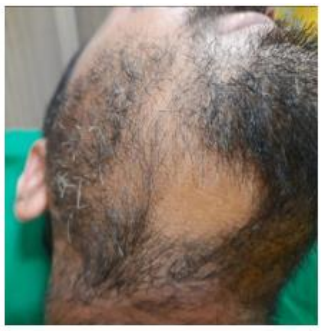
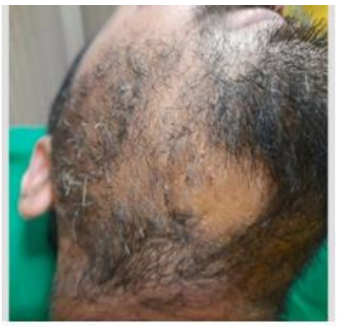


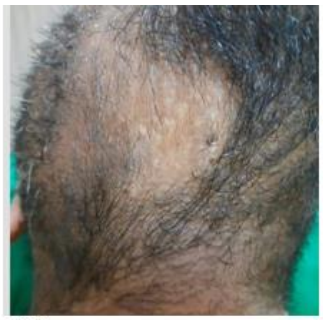
FIGURE-7: GROUP A- FRACTIONAL CO2 LASER WITH TOPICAL BIMATOPROST



BASELINE
GPA-0
LAD-9.2

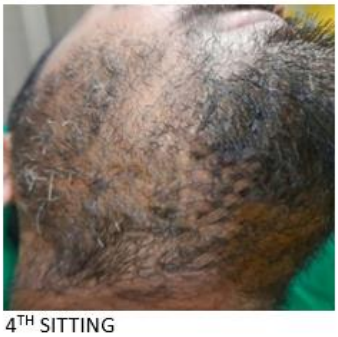


1ST SITTING
GPA-0
LAD-7.1

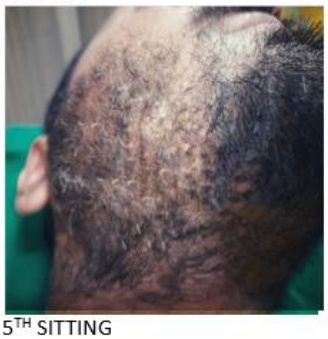


2ND SITTING
GPA-1
LAD-4.86

3RD SITTING
GPA-2
LAD-3.2

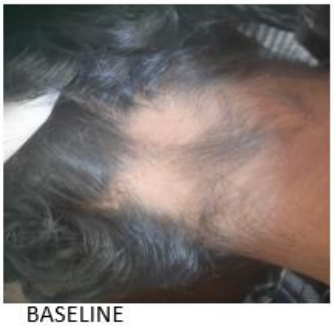


4TH SITTING
GPA-3
LAD-2.07

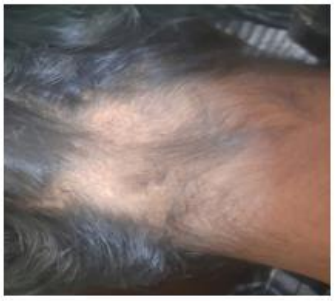


5TH SITTING
GPA-3
LAD-0.45

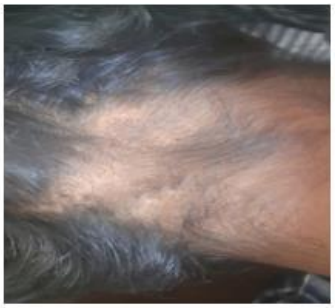
FIGURE-8: GROUP A- FRACTIONAL CO2 LASER WITH TOPICAL BIMATOPROST



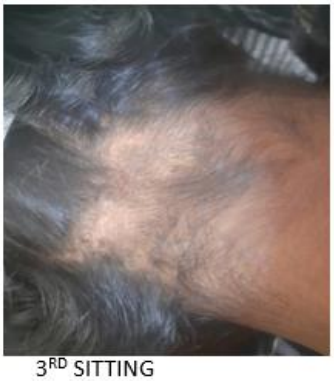
BASELINE
GPA-0
LAD-9.3



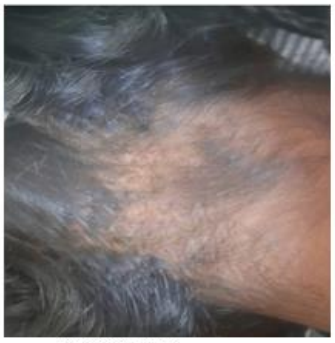
1ST SITTING
GPA-1
LAD-7.1



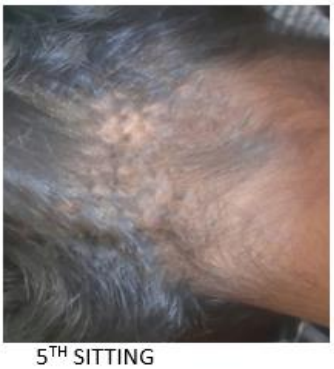
2ND SITTING
GPA-1
LAD-4.7



3RD SITTING
GPA-2
LAD-3.1



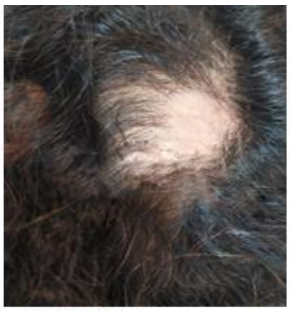
4TH SITTING
GPA-3
LAD-1.8



5TH SITTING
GPA-3
LAD-0.6

FIGURE-9: GROUP B- FRACTIONAL CO2 LASER WITH TOPICAL TRIAMCINOLONE ACETONIDE

BASELINE
GPA-0
LAD-9.3

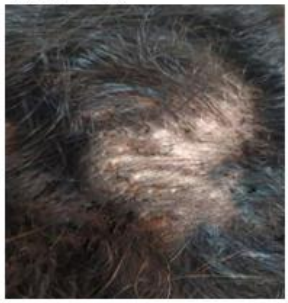


1ST SITTING
GPA – 0
LAD – 6.3

2ND SITTING
GPA – 1
LAD - 4.9

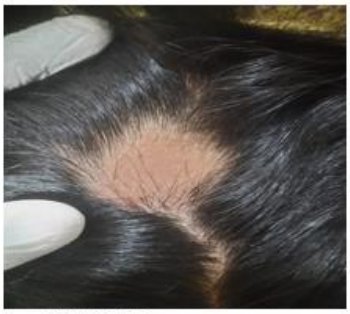
3RD SITTING
GPA – 2
LAD – 4.0

4TH SITTING
GPA – 2
LAD – 2.9

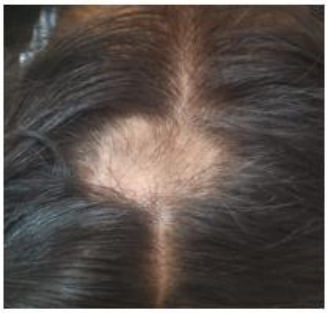


5TH SITTING
GPA – 2
LAD - 2.3

FIGURE-10: GROUP B- FRACTIONAL CO2 LASER WITH TOPICAL TRIAMCINOLONE ACETONIDE

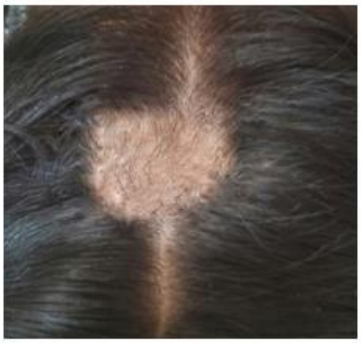


BASELINE
GPA-0
LAD-16.2

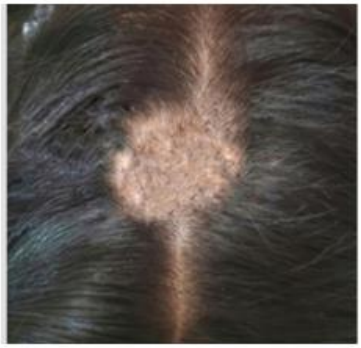


1ST SITTING
GPA-1
LAD-11.4

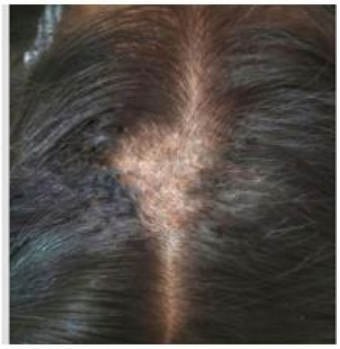
2ND SITTING
GPA-2
LAD-8.2



3RD SITTING
GPA-2
LAD-7.2



4TH SITTING
GPA-2
LAD-5.2



5TH SITTING
GPA-3
LAD-3.6

DISCUSSION

DISCUSSION

Alopecia areata (AA) is a complex genetic, immune mediated disease that targets anagen hair follicles. It is a long-term, immune-mediated condition marked by sudden, non-scarring hair loss that can range from small, localized patches to whole body and scalp hair loss.[2,7]

Bimatoprost eye drops usage in Glaucoma was associated with hypertrichosis of eyelashes as an adverse effect. This led to a need to research Bimatoprost as a potential therapeutic modality in treatment of alopecia areata. As a result, we evaluated and documented the safety and effectiveness of a novel treatment modality of Fractional CO2 LASER with Topical Bimatoprost in comparison to a conventional therapeutic approach of Fractional CO2 LASER with Topical Triamcinolone Acetonide.[81,82]

In addition to their direct therapeutic effect, Fractional LASER also act through Transepidermal drug delivery (TED) into the hair follicle. This property of fractional lasers is popularly known as "laser-assisted drug delivery," which has found use in many dermatological disorders. The generation of Micro-thermal zone by fractional lasers provides the channels for a uniform and controlled delivery of drugs. As the channels are distributed throughout the target area in a uniform manner, the drug deposition also becomes uniform.[93,94]

In this study, the age range of 19 to 30 years comprised the majority of AA patients (54.05%), followed by 31 to 40 years of age (22.97%). This finding is in association with the observations in the study done by Hamidpour E et al.[95] where the average age of patients was 27.2 ± 13.4 years old. Therefore, it is observed that Alopecia Areata have maximum incidence in the age range of 19 to 40 years. This observation may be because the young adults are more cosmetically concerned and thus report to Dermatology clinics for seeking treatments

and also the higher stress levels in this age group might be one of the triggering factors for onset of Alopecia Areata in this age group.

In our study, 62.16% were men (46 cases) and 37.83% were women (28 cases). This finding is consistent with the observations in the study by Nasimi M et al.[96] on effects of age and sex on the comorbidities of alopecia areata where males had a higher incidence of alopecia areata. This may be because of the higher expressivity of HLA genes and more susceptibility of anagen hair follicles to damage by breakdown of immune privilege associated with Alopecia Areata in males more than females.

In this study, 31.08% (23 cases) of the AA patients had a history of constant stress regarding day-to-day activities. Nakamura M et al.[97] observed the association of stress in 23% of the AA cases. This result is consistent with findings of our study which suggests that stress has a role in the etiopathogenesis of alopecia areata as a triggering factor.

Regarding the site of Patchy AA in our study, 89.1% (66 cases) had Scalp involvement, 8.1% (6 cases) had Beard involvement and 2.7% (2 cases) had Moustache involvement. This result is congruent with the study conducted by Sundberg JP et al.[30], where the majority of the cases recorded involved the scalp. Study by Uchida et al.[98] indicated that the number of γδ T cells was significantly higher in the scalp of AA patients compared to other sites. γδ T cells promote inflammation, resulting in high IFN-γ expressions and subsequent damage to anagen hair follicles. This might be the reason for higher incidence of AA patch at the scalp than any other site.

91.89% (68 cases) of the patients in our study were asymptomatic. 8.10% (6 cases) of the patients in our study were associated with itching. The study of Avilla L et al.[99] on Alopecia areata: Disease characteristics, clinical evaluation, and new perspectives on pathogenesis

supports this conclusion where most patients where asymptomatic on initial presentation of the disease.

77.02 % (57 cases) of our study population resorted to treatment for cosmetic non appealing nature of AA. 22.97% (17 cases) resorted to treatment for therapeutic benefit. This result is in line with those of Liu et al[100], who found that patients sought treatment primarily due to cosmetic concerns.

In our study, the efficacy of both treatment modalities was assessed using GPA – Scale and LAD Score Improvement %. At the end of 5th Setting, the GPA – Scale showed a score of 3 (> 75% improvement) to all cases treated with Fractional CO2 LASER followed by Topical Bimatoprost (100%) and 43.2% of cases treated with Fractional CO2 LASER with topical Triamcinolone Acetonide. Mean LAD Score Improvement % after 5th setting in cases treated with Fractional CO2 LASERS followed by Topical Bimatoprost was 95.34% and 75.64% in cases treated with Fractional CO2 LASER with topical Triamcinolone Acetonide.

In the study of the effect of latanoprost 0.005% solution in the management of scalp alopecia areata, a randomized double-blind placebo-controlled trial by Rafati M et al.[101], Latanoprost significantly increased hair density and regrowth (79.09%) based on the Severity of Alopecia Tool (SALT) system compared to the control group after 12 weeks of treatment. This finding is consistent with our study where mean LAD Improvement % at the end of 12 weeks was 82.03% in patients who was treated with Fractional CO2 LASER followed by Topical Bimatoprost.

In our study's first two settings where LAD Score Improvement % for patients treated with Fractional CO2 LASER with Topical Triamcinolone was better than with Fractional CO2 LASER with Topical Bimatoprost. Although in subsequent settings, drastic statistically

significant LAD Score Improvement % for patients who receive Fractional CO2 LASER with Topical Bimatoprost was present in our study. This result is congruent with the study conducted by Zaher et al.[84], where Topical Bimatoprost was shown to be more effective than topical mometasone furoate cream in the treatment of localized alopecia areata.

In our study, there is no significant difference in VDS in subjects treated with Fractional CO2 LASER with Topical Bimatoprost and cases treated with Fractional CO2 LASER with Topical Triamcinolone since mean VDS was 4.27 and 4.02 respectively.

In our study, patients who were treated with Fractional CO2 LASER with Topical Bimatoprost had mean VAS of 7.70 and patients treated with Fractional CO2 LASER with Topical Triamcinolone had mean VAS of 7.62. This result is in line with the observations of Majid et al.[89] in their research studies.

In our study, Patients who were treated with Fractional CO2 LASER with Topical Bimatoprost showed more transient adverse effect of Erythema in 35.1% as compared to the patients treated with Fractional CO2 LASER with Topical Triamcinolone which showed Erythema in 24.3%. This may be because the Bimatoprost is a Prostaglandin Analogue which also acts as a vasodilator.

However other complications such as edema, atrophy and post inflammatory hyperpigmentation were seen more with patients treated with Fractional CO2 LASER with Topical Triamcinolone. This finding is consistent with the research of Bhat S et al.[102] of A randomized comparative study of the efficacy of topical latanoprost versus topical betamethasone dipropionate lotion in the treatment of localized alopecia areata where out of 25 patients treated with topical betamethasone 3(12%) showed Atrophy of skin.

CONCLUSION

CONCLUSION:

The majority of AA patients are young adults (18–40 years old). Cosmetic concern is the primary reason for the patient's seeking treatment for Alopecia Areata. A patchy AA that deforms their overall appearance has a major psychological effect on the patient's mental state. Although there are various treatment options for AA but there still exists lacunae for a treatment modality which can provide faster and safer therapeutic results to the patients.

Bimatoprost with Fractional CO2 LASER achieved faster and cosmetically better outcome as compared to topical Triamcinolone with Fractional CO2 LASER.

Skin atrophy which was the major irreversible adverse effect with conventional topical and intralesional steroids was not seen with topical Bimatoprost. Except for transient self-resolving erythema which may be due to inherent vasodilatory effect of Bimatoprost, all other adverse effects like edema, post inflammatory hyperpigmentation were seen more with Topical Triamcinolone Acetonide group.

Although our study showed initial faster improvement in percentage LAD score with first two sittings of patients treated with Fractional CO2 LASER with Topical Triamcinolone but subsequent 3^{rd}, 4^{th} and 5^{th} sitting showed overall better improvement in percentage LAD score in patients treated with Fractional CO2 LASER with Topical Bimatoprost demonstrating the overall higher efficacy of Topical Bimatoprost over Topical Triamcinolone.

Therefore, in terms of efficacy, safety, and side effects, we conclude that Fractional CO2 laser combined with Topical Bimatoprost is a better therapeutic option for alopecia areata than conventional Fractional CO2 LASER with topical triamcinolone acetonide.

SUMMARY

SUMMARY:

The study involved 74 patients with Alopecia Areata (AA) and was conducted as a Randomized Controlled Trial (RCT). The patients were divided evenly between two groups, A and B, with 37 patients in each. Patients with AA were asked to provide informed permission regarding study participation, the nature of their disease, and the need for follow-up. The study included patients with Patchy Alopecia Areata over any part of the body. Patients who were immunocompromised, pregnant, or systemically ill were excluded from the trial.

Group A received fractional CO2 laser combined with topical Bimatoprost. Patients in Group B received Fractional CO2 combined with topical Triamcinolone Acetonide.

Treatment was given to both groups in five sittings, separated by three weeks between each sitting. Using the Global Photograph Assessment (GPA) Scale, the Visual Discomfort Scale (VDS), the Visual Analogue Scale (VAS), the Lesional Area Density Score Percentage of Improvement (LAD Score Improvement %), and the documentation of adverse effects in each sitting, the efficacy and safety of the treatment methods were evaluated in both groups.

The mean age of study participants in group A was 29.973 ± 8.513 years and group B was 28.216 ± 9.396 years with minimum age being 13 years and maximum age being 51 years. The age group of 19 to 30 (38.3%) is followed by those between the ages of 18 and 30 (41.6%), who made up the majority of research participants.

Males were 62.2% of the cases in our study, while females were 37.8%. 39.3% of the AA cases in our study had a strong family history of the disease. 19.3% had previously experienced Patchy AA and had received therapy using a variety of methods.

In the initial presentation, 43.6% of participants had one patchy AA, while 56.4% had multiple patchy AAs. 2.7% involved the moustache, 8.1% involved the beard, and 89.1% involved the scalp.

In 91.89% of cases, there were no symptoms, and 8.1% had itching. 22.97% of patients sought therapy for therapeutic benefit, while 77.02% of instances sought treatment due to the cosmetic concerns due to AA.

The mean GPA-Scale score at the end of Group A's first, second, third, fourth, and fifth sets was 0.75, 1.10, 2.18, 2.9, and 3, respectively. At the end of the first, second, third, fourth, and fifth sets in Group B, the mean GPA-Scale score was, correspondingly, 1, 1.45, 2, 2, and 2.43.

The mean LAD Score Improvement percent was 21.94 percent, 46.32 percent, 66.67 percent, 82.03 percent, and 95.34 percent, respectively, at the end of the first, second, third, fourth, and fifth sets in Group A. The mean percentage of LAD Score Improvement at the conclusion of the first, second, third, fourth, and fifth sets in Group B were 28.50, 48.39, 57.39, 68.29 and 75.64 percent, in that order.

As a result, the statistics on the effectiveness of both treatment modalities as determined by the GPA Scale and LAD Score Improvement % at the conclusion of five settings were statistically significant.

The mean VDS values for Groups A and B were 4.27 and 4.02, respectively. The VAS values for 75^{th} percentile for Groups A and B were 9 and 8 respectively.

After the Fractional CO2 LASER with Topical Bimatoprost, erythema was present in 35.1% of cases in Group A whereas Group B showed erythema in 24.3% cases. In Group B, 16.2%

of cases showed signs of skin atrophy, whereas there were no instances in Group A that showed signs of skin atrophy.

Therefore, in terms of efficacy, safety, and side effects, we conclude that Fractional CO2 laser combined with Topical Bimatoprost is a better therapeutic option for alopecia areata than conventional Fractional CO2 LASER with topical triamcinolone acetonide

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ANNEXURES

ANNEXURE – I

PROFORMA

- **Name:**
- **Age:**
- **Sex:**
- **Occupation:**
- **UHID number:**
- **Phone number:**
- **Address:**
- **Date :**

History of Presenting Illness:

1. Age of Onset:
2. Site of onset:
3. Duration:
4. Any Associated Symptoms: itching/ burning/ pain
5. Mode of spread: static/ growing/ receding
6. Use of any drugs before onset of illness
7. Aggravating factors: Occupational/ hobbies/ trauma/ drug/ work/ sunlight/ emotional factors/ menstruation/ pregnancy/ food/ cosmetics/ chemicals/ any other:
8. Recovery: Some/ good/ poor/ no response

Past history:

- Associated systemic diseases: DM/ HTN/ Thyroid disease.
- Associated cutaneous diseases:

Family history:

A. Similar complaints:
B. Other skin problems:
C. **Personal history:**
D. Diet: veg/ nonveg/ mixed
E. Bowel/ Bladder habits: regular/ altered.
F. Sleep- adequate/ disturbed
G. Appetite-
H. Habits: smoking/ tobacco chewing/ alcoholism

GENERAL PHYSICAL EXAMINATION :

- Built and nourishment
- Pallor/Icterus/Cyanosis/Clubbing/Generalised Lymphadenopathy/Edema

VITALS :

- Pulse :
- BP :
- Temperature :
- Respiratory Rate :

SYSTEMIC EXAMINATION :

1. CVS
2. RS
3. PER ABDOMEN
4. CNS

LOCAL EXAMINATION :

INSPECTION :

- SITE
- SIZE
- NUMBER
- SYMMETRY
- BORDER

PALPATION :

- LOCAL RISE OF TEMPERATURE
- TENDERNESS
- NUMBER
- SIZE
- SURFACE
- BORDER

Nail examination :

- **INVESTIGATIONS :**
- **FINAL DIAGNOSIS :**
- **TREATMENT :**
- **REMARKS OF THE GUIDE :**

ANNEXURE – II

INFORMED CONSENT FORM

I Mr./Mrs. _____ have been explained in my own understandable language, that I will be included in a study which is **A COMPARATIVE STUDY OF FRACTIONAL CO2 LASER WITH TOPICAL BIMATOPROST 0.03% VERSUS FRACTIONAL CO2 LASER WITH TOPICAL TRIAMCINOLONE ACETONIDE IN THE TREATMENT OF ALOPECIA AREATA.**

Hence as per the computer generated randomization of the study – I am allotted to

Group - ____ for whom _____ will be given as a treatment modality for my illness.

I have been explained about the randomization of the treatment modality I receive and that my clinical findings, investigations, intra operative findings will be assessed and documented for study purpose.

I have been explained my participation in this study is entirely voluntary, and I can withdraw from the study any time and this will not affect my relation with my doctor or the treatment for my ailment.

I have been explained about the necessity of the intervention, possible benefits and adverse effects due to interventions, in my own understandable language.

I have understood that all my details found during the study are kept confidential and while publishing or sharing of the findings, my details will be masked.

The principal investigator will bear the cost of the study.

I have principal investigator mobile number for enquiries.

I in my sound mind give full consent to be added in the part of this study.

No monetary benefits will be given to me during the study.

Signature of the patient	Signature of the witness
Name:	Name:
Date: Place:	Relation to patient:

ಮಾಹಿತಿ ನೀಡಿದ ಒಪ್ಪಿಗೆ ನಮೂನೆ

ನಾನು ಶ್ರೀ/ಶ್ರೀಮತಿ. _____ ಅನ್ನು ನನ್ನ ಸ್ವಂತ ಅರ್ಥವಾಗುವ ಭಾಷೆಯಲ್ಲಿ ವಿವರಿಸಲಾಗಿದೆ, ಇದು ಒಂದು ಅಧ್ಯಯನದಲ್ಲಿ ನನ್ನನ್ನು ಸೇರಿಸಿಕೊಳ್ಳಲಾಗುವುದು ಇದು ವಿಷಯಾಧಾರಿತ ಬೈಮಾಟೊಪ್ರೊಸ್ಟ್ **0.03%** ವಿತ್ ಫ್ರ್ಯಾಕ್ಶನಲ್ **CO2** ಲೇಸರ್‌ನ ತುಲನಾತ್ಮಕ ಅಧ್ಯಯನವಾಗಿದೆ.

ಆದ್ದರಿಂದ ಕಂಪ್ಯೂಟರ್ ರಚಿತವಾದ ಅಧ್ಯಯನದ ಯಾದೃಚ್ಛಿಕತೆಯ ಪ್ರಕಾರ - ನನಗೆ ನಿಯೋಜಿಸಲಾಗಿದೆ

ಗುಂಪು - ____ ಯಾರಿಗೆ _____ ನನ್ನ ಅನಾರೋಗ್ಯಕ್ಕೆ ಚಿಕಿತ್ಸಾ ವಿಧಾನವಾಗಿ ನೀಡಲಾಗುವುದು.

ನಾನು ಸ್ವೀಕರಿಸುವ ಚಿಕಿತ್ಸಾ ವಿಧಾನದ ಯಾದೃಚ್ಛಿಕತೆಯ ಬಗ್ಗೆ ನನಗೆ ವಿವರಿಸಲಾಗಿದೆ ಮತ್ತು ನನ್ನ ಕ್ಲಿನಿಕಲ್ ಸಂಶೋಧನೆಗಳು, ತನಿಖೆಗಳು, ಇಂಟ್ರಾ ಆಪರೇಟಿವ್ ಸಂಶೋಧನೆಗಳನ್ನು ಮೌಲ್ಯಮಾಪನ ಮಾಡಲಾಗುತ್ತದೆ ಮತ್ತು ಅಧ್ಯಯನ ಉದ್ದೇಶಕ್ಕಾಗಿ ದಾಖಲಿಸಲಾಗುತ್ತದೆ.
ಈ ಅಧ್ಯಯನದಲ್ಲಿ ನನ್ನ ಭಾಗವಹಿಸುವಿಕೆಯು ಸಂಪೂರ್ಣವಾಗಿ ಸ್ವಯಂಪ್ರೇರಿತವಾಗಿದೆ ಎಂದು ನನಗೆ ವಿವರಿಸಲಾಗಿದೆ ಮತ್ತು ನಾನು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಅಧ್ಯಯನದಿಂದ ಹಿಂದೆ ಸರಿಯಬಹುದು ಮತ್ತು ಇದು ನನ್ನ ವೈದ್ಯರೊಂದಿಗಿನ ನನ್ನ ಸಂಬಂಧ ಅಥವಾ ನನ್ನ ಕಾಯಿಲೆಯ ಚಿಕಿತ್ಸೆಯ ಮೇಲೆ ಪರಿಣಾಮ ಬೀರುವುದಿಲ್ಲ.
ನನ್ನ ಸ್ವಂತ ಅರ್ಥವಾಗುವ ಭಾಷೆಯಲ್ಲಿ ಹಸ್ತಕ್ಷೇಪದ ಅಗತ್ಯತೆ, ಸಂಭವನೀಯ ಪ್ರಯೋಜನಗಳು ಮತ್ತು ಮಧ್ಯಸ್ಥಿಕೆಗಳಿಂದಾಗುವ ಪ್ರತಿಕೂಲ ಪರಿಣಾಮಗಳ ಬಗ್ಗೆ ನನಗೆ ವಿವರಿಸಲಾಗಿದೆ.

ಅಧ್ಯಯನದ ಸಮಯದಲ್ಲಿ ಪತ್ತೆಯಾದ ನನ್ನ ಎಲ್ಲಾ ವಿವರಗಳನ್ನು ಗೌಪ್ಯವಾಗಿ ಇರಿಸಲಾಗಿದೆ ಮತ್ತು ಸಂಶೋಧನೆಗಳನ್ನು ಪ್ರಕಟಿಸುವಾಗ ಅಥವಾ ಹಂಚಿಕೊಳ್ಳುವಾಗ, ನನ್ನ ವಿವರಗಳನ್ನು ಮರೆಮಾಚಲಾಗುತ್ತದೆ ಎಂದು ನಾನು ಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ.

ಮುಖ್ಯ ತನಿಖಾಧಿಕಾರಿಗಳು ಅಧ್ಯಯನದ ವೆಚ್ಚವನ್ನು ಭರಿಸುತ್ತಾರೆ.

ವಿಚಾರಣೆಗಾಗಿ ನಾನು ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿಯ ಮೊಬೈಲ್ ಸಂಖ್ಯೆಯನ್ನು ಹೊಂದಿದ್ದೇನೆ.

ಈ ಅಧ್ಯಯನದ ಭಾಗದಲ್ಲಿ ಸೇರಿಸಲು ನನ್ನ ಉತ್ತಮ ಮನಸ್ಸಿನಲ್ಲಿ ನಾನು ಸಂಪೂರ್ಣ ಒಪ್ಪಿಗೆಯನ್ನು ನೀಡುತ್ತೇನೆ.

ಅಧ್ಯಯನದ ಸಮಯದಲ್ಲಿ ನನಗೆ ಯಾವುದೇ ಹಣಕಾಸಿನ ಪ್ರಯೋಜನಗಳನ್ನು ನೀಡಲಾಗುವುದಿಲ್ಲ.

· ರೋಗಿಯ ಸಹಿ ಸಾಕ್ಷಿಯ ಸಹಿ

· ಹೆಸರು: ಹೆಸರು:

· ದಿನಾಂಕ: ರೋಗಿಗೆ ಸಂಬಂಧ:

· ಸ್ಥಳ

ANNEXURE – III

PATIENT INFORMATION SHEET

STUDY TITLE:

A COMPARATIVE STUDY OF FRACTIONAL CO2 LASER WITH TOPICAL BIMATOPROST 0.03% VERSUS FRACTIONAL CO2 LASER WITH TOPICAL TRIAMCINOLONE ACETONIDE IN THE TREATMENT OF ALOPECIA AREATA

PLACE OF STUDY: R.L Jalappa Hospital and Research Centre, Tamaka, Kolar.

OBJECTIVES :

1) To assess and compare the efficacy of Fractional CO2 Laser with Topical Bimatoprost 0.03% versus Fractional CO2 Laser with Topical Triamcinolone Acetonide in the treatment of Alopecia Areata.

2) To document the post procedural adverse effects of Fractional CO2 with Topical Bimatoprost 0.03% and Fractional Co2 Laser with Topical Triamcinolone Acetonide in the treatment of Alopecia areata.

- Alopecia Areata is the patchy loss of hair on any part of the body affecting both gender, which gives psychological distress affecting quality of life of the patient, and it is also associated with other diseases. It is not contagious and not transmitted from one person to another by touching, eating together, sharing clothes.
- Alopecia Areata can be diagnosed by clinical examination.In this study two treatment modalities for alopecia areata are documented.

In this study, Group A Participants - A topical anaesthetic, containing a mixture of lidocaine-2.5% w/w + prilocaine-2.5% w/w in a cream base will be applied for 1 hour on the treatment area. After satisfactory anaesthesia is achieved, the treatment area will be cleaned with a mild cleanser. Eyes will be protected with eye shields. Fractional CO2 LASER will be then delivered to the Alopecia Areata site at the fluence of 50-60 mJ/cm2(trans epidermal pores are created in the skin) .Ice pack will be applied immediately. Then within 2 minutes of

LASER procedure, Topical Bimatoprost 0.03% will be applied and asked to continue twice daily application till next sitting.

GROUP B – A topical anaesthetic, containing a mixture of lidocaine-2.5% w/w + prilocaine-2.5% w/w in a cream base will be applied for 1 hour on the treatment area. After satisfactory anaesthesia is achieved, the treatment area will be cleaned with a mild cleanser. Eyes will be protected with eye shields. Fractional CO2 LASER will be then delivered to the Alopecia Areata site at the fluence of 50-60 mJ/cm2 (trans epidermal pores are created in the skin). Ice pack will be applied immediately. Then within 2 minutes of LASER procedure, Topical Triamcinolone Acetonide Aqueous solution (2.5 mg/ml to 10 mg/ml) will be applied.

- Please read the following information and discuss with your family members. You can ask any question regarding the study. If you agree to participate in this study we will collect information (as per proforma) from you. Relevant blood investigations will be carried out if required. This information collected will be used for dissertation and publication only. NO MONETARY BENEFITS WILL BE MADE AVAILABLE FOR PARTICIPANTS OF THE STUDY. Even If you are not willing to participate in this study the care, treatment & relationship with doctor will not affect All information collected from you will be kept confidential and will not be disclosed to any outsider. Your identity will not be revealed. The expenses required for the above study will be taken care by the principal investigator. This study has been reviewed by the Institutional Ethics Committee and you are free to contact the member of the Institutional Ethics Committee. There is no compulsion to agree to this study.The care you will get will not change if you don't wish to participate. You are required to sign/ provide thumb impression only if you voluntarily agree to participate in this study.

- Left Thumb Impression/Signature of the Patient Left Thumb Impression/Signature of the Witness
- **For any further clarification you can contact the study investigator:**
- Dr. Hussain Kolsawala
- Mobile no: 8105907922
- E-mail id: hussainkolsa786@gmail.com

ರೋಗಿಯ ಮಾಹಿತಿ ಹಾಳೆ

ಅಧ್ಯಯನದ ಶೀರ್ಷಿಕೆ:

ಅಲೋಪೆಸಿಯಾ ಚಿಕಿತ್ಸೆಯಲ್ಲಿ ಸಾಮಯಿಕ ಟ್ರಯಾಮಿಸಿನೋಲೋನ್ ಅಸಿಟೋನೈಡ್‌ನೊಂದಿಗಿನ 0.03% ವರ್ಸಸ್ ಫ್ರ್ಯಾಕ್ಷನಲ್ CO2 ಲೇಸರ್‌ನೊಂದಿಗೆ ಫ್ರ್ಯಾಕ್ಷನಲ್ CO2 ಲೇಸರ್‌ನ ತುಲನಾತ್ಮಕ ಅಧ್ಯಯನ

ಸ್ಥಿ ಸೈಟ್: ಆರ್.ಎಲ್ ಜಾಲಪ್ಪ ಆಸ್ಪತ್ರೆ ಮತ್ತು ಸಂಶೋಧನಾ ಕೇಂದ್ರ, ಟಮಕ, ಕೋಲಾರ.

ಉದ್ದೇಶಗಳು:

1) ಅಲೋಪೆಸಿಯಾ ಏರಿಯಾಟಾ ಚಿಕಿತ್ಸೆಯಲ್ಲಿ ಫ್ರ್ಯಾಕ್ಷನಲ್ CO2 ಲೇಸರ್‌ನ ಪರಿಣಾಮಕಾರಿತ್ವವನ್ನು 0.03% ವರ್ಸಸ್ ಫ್ರ್ಯಾಕ್ಷನಲ್ CO2 ಲೇಸರ್ ಜೊತೆಗೆ ಟಾಪಿಕಲ್ ಟ್ರಯಾಮ್ಸಿನೋಲೋನ್ ಅಸಿಟೋನೈಡ್‌ನೊಂದಿಗೆ ಹೋಲಿಸಲು.

2) ಅಲೋಪೆಸಿಯಾ ಅರೆಟಾ ಚಿಕಿತ್ಸೆಯಲ್ಲಿ ಫ್ರ್ಯಾಕ್ಷನಲ್ CO2 ನ ನಂತರದ ಪ್ರತಿಕೂಲ ಪರಿಣಾಮಗಳನ್ನು ದಾಖಲಿಸಲು.

· ಅಲೋಪೆಸಿಯಾ ಏರಿಯಾಟಾ ಎನ್ನುವುದು ದೇಹದ ಯಾವುದೇ ಭಾಗದಲ್ಲಿ ಕೂದಲು ಉದುರುವಿಕೆಯಾಗಿದ್ದು, ಎರಡೂ ಲಿಂಗಗಳ ಮೇಲೆ ಪರಿಣಾಮ ಬೀರುತ್ತದೆ, ಇದು ರೋಗಿಯ ಜೀವನದ ಗುಣಮಟ್ಟದ ಮೇಲೆ ಪರಿಣಾಮ ಬೀರುವ ಮಾನಸಿಕ ಯಾತನೆಯನ್ನು ನೀಡುತ್ತದೆ ಮತ್ತು ಇದು ಇತರ ಕಾಯಿಲೆಗಳೊಂದಿಗೆ ಸಹ ಸಂಬಂಧಿಸಿದೆ. ಇದು ಸಾಂಕ್ರಾಮಿಕವಲ್ಲ ಮತ್ತು ಒಬ್ಬರಿಂದ ಇನ್ನೊಬ್ಬರಿಗೆ ಮುಟ್ಟುವ, ಒಟ್ಟಿಗೆ ತಿನ್ನುವ, ಬಟ್ಟೆ ಹಂಚಿಕೊಳ್ಳುವ ಮೂಲಕ ಹರಡುವುದಿಲ್ಲ.

· ಅಲೋಪೆಸಿಯಾ ಏರಿಯಾಟಾವನ್ನು ಕ್ಲಿನಿಕಲ್ ಪರೀಕ್ಷೆಯ ಮೂಲಕ ನಿರ್ಣಯಿಸಬಹುದು. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಅಲೋಪೆಸಿಯಾ ಏರಿಯಾಟಾದ ಎರಡು ಚಿಕಿತ್ಸಾ ವಿಧಾನಗಳನ್ನು ದಾಖಲಿಸಲಾಗಿದೆ.

ಈ ಅಧ್ಯಯನದಲ್ಲಿ, ಗ್ರೂಪ್ ಎ ಭಾಗವಹಿಸುವವರು - ಲಿಡೋಕೇಯ್ನ್-2.5% w/w + prilocaine-2.5% w/w ಮಿಶ್ರಣವನ್ನು ಒಳಗೊಂಡಿರುವ ಒಂದು ಸಾಮಯಿಕ ಅರಿವಳಿಕೆ, ಕ್ರೀಮ್ ಬೇಸ್‌ನಲ್ಲಿ 1 ಗಂಟೆಯವರೆಗೆ ಚಿಕಿತ್ಸಾ ಪ್ರದೇಶದ ಮೇಲೆ ಅನ್ವಯಿಸಲಾಗುತ್ತದೆ. ತೃಪ್ತಿಕರ ಅರಿವಳಿಕೆ ಸಾಧಿಸಿದ ನಂತರ, ಚಿಕಿತ್ಸೆಯ ಪ್ರದೇಶವನ್ನು ಸೌಮ್ಯವಾದ ಕ್ಲೆನ್ಸರ್ನೊಂದಿಗೆ ಸ್ವಚ್ಛಗೊಳಿಸಲಾಗುತ್ತದೆ. ಕಣ್ಣುಗಳನ್ನು ಕಣ್ಣಿನ ಗುರಾಣಿಗಳಿಂದ ರಕ್ಷಿಸಲಾಗುವುದು. ಫ್ರ್ಯಾಕ್ಷನಲ್ CO2 ಲೇಸರ್ ಅನ್ನು ನಂತರ 50-60 mJ/cm2 ನ ಫ್ಲೂಯೆನ್ಸ್‌ನಲ್ಲಿ ಅಲೋಪೆಸಿಯಾ ಏರಿಯಾಟಾ ಸೈಟ್‌ಗೆ ತಲುಪಿಸಲಾಗುತ್ತದೆ. ನಂತರ ಲೇಸರ್ ಕಾರ್ಯವಿಧಾನದ 2 ನಿಮಿಷಗಳಲ್ಲಿ, ಟಾಪಿಕಲ್ ಬಿಮಾಟೊಪ್ರೊಸ್ಟ್ 0.03% ಅನ್ನು ಅನ್ವಯಿಸಲಾಗುತ್ತದೆ ಮತ್ತು ಮುಂದಿನ ಕುಳಿತುಕೊಳ್ಳುವವರೆಗೆ ದಿನಕ್ಕೆ ಎರಡು ಬಾರಿ ಅನ್ವಯಿಸುವುದನ್ನು ಮುಂದುವರಿಸಲು ಕೇಳಲಾಗುತ್ತದೆ.

ಗ್ರೂಪ್ ಬಿ - ಒಂದು ಸಾಮಯಿಕ ಅರಿವಳಿಕೆ, ಕ್ರೀಮ್ ಬೇಸ್ನಲ್ಲಿ ಲಿಡೋಕೇಯ್ನ್-2.5% w/w + prilocaine-2.5% w/w ಮಿಶ್ರಣವನ್ನು ಹೊಂದಿರುವ 1 ಗಂಟೆ ಚಿಕಿತ್ಸೆಯ ಪ್ರದೇಶದಲ್ಲಿ ಅನ್ವಯಿಸಲಾಗುತ್ತದೆ. ತೃಪ್ತಿಕರ ಅರಿವಳಿಕೆ ಸಾಧಿಸಿದ ನಂತರ,

ಚಿಕಿತ್ಸೆಯ ಪ್ರದೇಶವನ್ನು ಸೌಮ್ಯವಾದ ಕ್ಲೆನ್ಸರ್ನೊಂದಿಗೆ ಸ್ವಚ್ಛಗೊಳಿಸಲಾಗುತ್ತದೆ. ಕಣ್ಣುಗಳನ್ನು ಕಣ್ಣಿನ ಗುರಾಣಿಗಳಿಂದ ರಕ್ಷಿಸಲಾಗುವುದು. ಫ್ರಾಕ್ಷನಲ್ CO2 ಲೇಸರ್ ಅನ್ನು 50-60 mJ/cm2 ನ ಫ್ಲೂಯೆನ್ಸ್ನಲ್ಲಿ ಅಲೋಪೆಸಿಯಾ ಏರಿಯಾಟಾ ಸೈಟ್ಗೆ ತಲುಪಿಸಲಾಗುತ್ತದೆ (ಟ್ರಾನ್ಸ್ ಎಪಿಡರ್ಮಲ್ ರಂಧ್ರಗಳನ್ನು ಚರ್ಮದಲ್ಲಿ ರಚಿಸಲಾಗುತ್ತದೆ). ಐಸ್ ಪ್ಯಾಕ್ ಅನ್ನು ತಕ್ಷಣವೇ ಅನ್ವಯಿಸಲಾಗುತ್ತದೆ. ನಂತರ ಲೇಸರ್ ಕಾರ್ಯವಿಧಾನದ 2 ನಿಮಿಷಗಳಲ್ಲಿ, ಟಾಪಿಕಲ್ ಟ್ರಯಾಮ್ಸಿನೋಲೋನ್ ಅಸಿಟೋನೈಡ್ ಜಲೀಯ ದ್ರಾವಣವನ್ನು (2.5 mg/ml ನಿಂದ 10 mg/ml) ಅನ್ವಯಿಸಲಾಗುತ್ತದೆ.

· ದಯವಿಟ್ಟು ಕೆಳಗಿನ ಮಾಹಿತಿಯನ್ನು ಓದಿ ಮತ್ತು ನಿಮ್ಮ ಕುಟುಂಬದ ಸದಸ್ಯರೊಂದಿಗೆ ಚರ್ಚಿಸಿ. ಅಧ್ಯಯನಕ್ಕೆ ಸಂಬಂಧಿಸಿದಂತೆ ನೀವು ಯಾವುದೇ ಪ್ರಶ್ನೆಯನ್ನು ಕೇಳಬಹುದು. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ನೀವು ಒಪ್ಪಿದರೆ ನಾವು ನಿಮ್ಮಿಂದ ಮಾಹಿತಿಯನ್ನು (ಪ್ರೊಫಾರ್ಮಾ ಪ್ರಕಾರ) ಸಂಗ್ರಹಿಸುತ್ತೇವೆ. ಅಗತ್ಯವಿದ್ದರೆ ಸಂಬಂಧಿತ ರಕ್ತ ಪರೀಕ್ಷೆಗಳನ್ನು ಕೈಗೊಳ್ಳಲಾಗುತ್ತದೆ. ಸಂಗ್ರಹಿಸಿದ ಈ ಮಾಹಿತಿಯನ್ನು ಪ್ರಬಂಧ ಮತ್ತು ಪ್ರಕಟಣೆಗೆ ಮಾತ್ರ ಬಳಸಲಾಗುತ್ತದೆ. ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸುವವರಿಗೆ ಯಾವುದೇ ವಿತ್ತೀಯ ಪ್ರಯೋಜನಗಳು ಲಭ್ಯವಾಗುವುದಿಲ್ಲ. ನೀವು ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ಸಿದ್ಧರಿಲ್ಲದಿದ್ದರೂ ಸಹ ವೈದ್ಯರೊಂದಿಗಿನ ಆರೈಕೆ, ಚಿಕಿತ್ಸೆ ಮತ್ತು ಸಂಬಂಧವು ಪರಿಣಾಮ ಬೀರುವುದಿಲ್ಲ ನಿಮ್ಮಿಂದ ಸಂಗ್ರಹಿಸಿದ ಎಲ್ಲಾ ಮಾಹಿತಿಯನ್ನು ಗೌಪ್ಯವಾಗಿ ಇರಿಸಲಾಗುತ್ತದೆ ಮತ್ತು ಯಾವುದೇ ಹೊರಗಿನವರಿಗೆ ಬಹಿರಂಗಪಡಿಸಲಾಗುವುದಿಲ್ಲ. ನಿಮ್ಮ ಗುರುತನ್ನು ಬಹಿರಂಗಪಡಿಸಲಾಗುವುದಿಲ್ಲ. ಮೇಲಿನ ಅಧ್ಯಯನಕ್ಕೆ ಅಗತ್ಯವಿರುವ ವೆಚ್ಚಗಳನ್ನು ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿಗಳು ನೋಡಿಕೊಳ್ಳುತ್ತಾರೆ. ಈ ಅಧ್ಯಯನವನ್ನು ಸಾಂಸ್ಥಿಕ ನೀತಿಶಾಸ್ತ್ರ ಸಮಿತಿಯು ಪರಿಶೀಲಿಸಿದೆ ಮತ್ತು ನೀವು ಸಾಂಸ್ಥಿಕ ನೀತಿಶಾಸ್ತ್ರ ಸಮಿತಿಯ ಸದಸ್ಯರನ್ನು ಸಂಪರ್ಕಿಸಲು ಮುಕ್ತರಾಗಿದ್ದೀರಿ. ಈ ಅಧ್ಯಯನವನ್ನು ಒಪ್ಪಿಕೊಳ್ಳಲು ಯಾವುದೇ ಒತ್ತಾಯವಿಲ್ಲ. ನೀವು ಭಾಗವಹಿಸಲು ಬಯಸದಿದ್ದರೆ ನೀವು ಪಡೆಯುವ ಕಾಳಜಿಯು ಬದಲಾಗುವುದಿಲ್ಲ. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ನೀವು ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ ಸಮ್ಮತಿಸಿದರೆ ಮಾತ್ರ ನೀವು ಸಹಿ/ಹೆಬ್ಬೆರಳಿನ ಗುರುತನ್ನು ಒದಗಿಸಬೇಕಾಗುತ್ತದೆ.

· ಎಡ ಹೆಬ್ಬೆರಳಿನ ಅನಿಸಿಕೆ/ರೋಗಿಯ ಸಹಿ ಎಡ ಹೆಬ್ಬೆರಳಿನ ಅನಿಸಿಕೆ/ಸಾಕ್ಷಿಯ ಸಹಿ

· ಯಾವುದೇ ಹೆಚ್ಚಿನ ಸ್ಪಷ್ಟೀಕರಣಕ್ಕಾಗಿ ನೀವು ಅಧ್ಯಯನ ತನಿಖಾಧಿಕಾರಿಯನ್ನು ಸಂಪರ್ಕಿಸಬಹುದು:

· ಡಾ.ಹುಸೇನ್ ಕೊಲಸಾವಾಲ

· ಮೊಬೈಲ್ ಸಂಖ್ಯೆ: 8105907922

· ಇ-ಮೇಲ್ ಐಡಿ: hussainkolsa786@gmail.com

ANNEXURE – IV

ASSENT CONSENT FORM (12-17 years)

Title of the study: A COMPARATIVE STUDY OF FRACTIONAL CO2 LASER WITH TOPICAL BIMATOPROST 0.03% VERSUS FRACTIONAL CO2 LASER WITH TOPICAL TRIAMCINOLONE ACETONIDE IN THE TREATMENT OF ALOPECIA AREATA

Name of the Participant:

Name of the Principal investigator: Dr.HUSSAIN KOLSAWALA

Name of the Institution: R.L Jalappa Hospital and Research Centre, Tamaka, Kolar.

Documentation of the informed consent

I Mr./Ms._____ have been explained in my own understandable language, that I will be included in the study which is **A COMPARATIVE STUDY OF FRACTIONAL CO2 LASER WITH TOPICAL BIMATOPROST 0.03% VERSUS FRACTIONAL CO2 LASER WITH TOPICAL TRIAMCINOLONE ACETONIDE IN THE TREATMENT OF ALOPECIA AREATA**

I have been explained that my clinical findings, investigations will be assessed and documented for study purpose.

I have been explained that my participation in this study is entirely voluntary, and I can withdraw from the study any time and this will not affect my relation with my doctor or the treatment for my ailment.

I have understood that all my details found during the study are kept confidential and while publishing or sharing of the findings, my details will be masked.

The principal investigator will bear the cost of study.

I have principal investigator's mobile number for enquires.

I, in my sound mind give full consent to be added in the part of this study.

Participant's initials:_____

Name and signature/thumb impression of the patient

_____	_____	_____
Name	Signature	Date

Name and signature of witness

_____	_____	_____
Name	Signature	Date

Name and Signature of the investigator or his representative obtaining consent:

_____	_____	_____
Name	Signature	Date

ಸಮ್ಮತಿ ನಮೂನೆ (12-17 ವರ್ಷಗಳು)

ಅಧ್ಯಯನದ ಶೀರ್ಷಿಕೆ: ಸಾಮಯಿಕ ಬೈಮಾಟೊಪ್ರೊಸ್ಟ್ 0.03% ವಿರುದ್ಧ ಫ್ರಾಕ್ಶನಲ್ CO2 ಲೇಸರ್ ಜೊತೆಗೆ ಫ್ರ್ಯಾಕ್ಶನಲ್ CO2 ಲೇಸರ್‌ನ ತುಲನಾತ್ಮಕ ಅಧ್ಯಯನವು ಟಾಪಿಕಲ್ ಟ್ರೈಯಾಮಿನೋಲೋನ್ ಅಸಿಟೋನೈಡ್ ಜೊತೆಗೆ ಚಿಕಿತ್ಸೆಯಲ್ಲಿ

ಭಾಗವಹಿಸುವವರ ಹೆಸರು:

ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿಯ ಹೆಸರು: ಡಾ.ಹುಸೇನ್ ಕೋಲ್ಸವಾಲಾ

ಸಂಸ್ಥೆಯ ಹೆಸರು: R.L ಜಾಲಪ್ಪ ಆಸ್ಪತ್ರೆ ಮತ್ತು ಸಂಶೋಧನಾ ಕೇಂದ್ರ, ಟಮಕಾ,

ಕೋಲಾರ.

ತಿಳುವಳಿಕೆಯುಳ್ಳ ಒಪ್ಪಿಗೆಯ ದಾಖಲೆ

ನನ್ನ ಸ್ವಂತ ಅರ್ಥವಾಗುವ ಭಾಷೆಯಲ್ಲಿ ನಾನು ಶ್ರೀ. ಅಲೋಪೆಸಿಯಾ ಏರಿಯಾಟಾ

ನನ್ನ ಕ್ಲಿನಿಕಲ್ ಸಂಶೋಧನೆಗಳು, ತನಿಖೆಗಳನ್ನು ಮೌಲ್ಯಮಾಪನ ಮಾಡಲಾಗುತ್ತದೆ ಮತ್ತು ಅಧ್ಯಯನ ಉದ್ದೇಶಕ್ಕಾಗಿ ದಾಖಲಿಸಲಾಗುತ್ತದೆ ಎಂದು ನನಗೆ ವಿವರಿಸಲಾಗಿದೆ.

ಈ ಅಧ್ಯಯನದಲ್ಲಿ ನನ್ನ ಭಾಗವಹಿಸುವಿಕೆಯು ಸಂಪೂರ್ಣವಾಗಿ ಸ್ವಯಂಪ್ರೇರಿತವಾಗಿದೆ ಮತ್ತು ನಾನು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಅಧ್ಯಯನದಿಂದ ಹಿಂದೆ ಸರಿಯಬಹುದು ಮತ್ತು ಇದು ನನ್ನ ವೈದ್ಯರೊಂದಿಗಿನ ನನ್ನ ಸಂಬಂಧ ಅಥವಾ ನನ್ನ ಕಾಯಿಲೆಯ ಚಿಕಿತ್ಸೆಯ ಮೇಲೆ ಪರಿಣಾಮ ಬೀರುವುದಿಲ್ಲ ಎಂದು ನನಗೆ ವಿವರಿಸಲಾಗಿದೆ.

ಅಧ್ಯಯನದ ಸಮಯದಲ್ಲಿ ಪತ್ತೆಯಾದ ನನ್ನ ಎಲ್ಲಾ ವಿವರಗಳನ್ನು ಗೌಪ್ಯವಾಗಿ ಇರಿಸಲಾಗಿದೆ ಮತ್ತು ಸಂಶೋಧನೆಗಳನ್ನು ಪ್ರಕಟಿಸುವಾಗ ಅಥವಾ ಹಂಚಿಕೊಳ್ಳುವಾಗ, ನನ್ನ ವಿವರಗಳನ್ನು ಮರೆಮಾಚಲಾಗುತ್ತದೆ ಎಂದು ನಾನು ಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ.

ಮುಖ್ಯ ತನಿಖಾಧಿಕಾರಿಗಳು ಅಧ್ಯಯನದ ವೆಚ್ಚವನ್ನು ಭರಿಸುತ್ತಾರೆ.

ವಿಚಾರಣೆಗಾಗಿ ನನ್ನ ಬಳಿ ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿಯ ಮೊಬೈಲ್ ಸಂಖ್ಯೆ ಇದೆ.

ನಾನು, ನನ್ನ ಉತ್ತಮ ಮನಸ್ಸಿನಲ್ಲಿ ಈ ಅಧ್ಯಯನದ ಭಾಗದಲ್ಲಿ ಸೇರಿಸಲು ಸಂಪೂರ್ಣ ಒಪ್ಪಿಗೆಯನ್ನು ನೀಡುತ್ತೇನೆ.

ಭಾಗವಹಿಸುವವರ ಮೊದಲಕ್ಷರಗಳು:_____

ರೋಗಿಯ ಹೆಸರು ಮತ್ತು ಸಹಿ/ಹೆಬ್ಬೆರಳಿನ ಗುರುತು

_____

ಹೆಸರು ಸಹಿ ದಿನಾಂಕ

ಸಾಕ್ಷಿಯ ಹೆಸರು ಮತ್ತು ಸಹಿ

_____

ಹೆಸರು ಸಹಿ ದಿನಾಂಕ

ತನಿಖಾಧಿಕಾರಿಯ ಹೆಸರು ಮತ್ತು ಸಹಿ ಅಥವಾ ಅವರ ಪ್ರತಿನಿಧಿ ಒಪ್ಪಿಗೆಯನ್ನು ಪಡೆಯುವುದು:

_____

ಹೆಸರು ಸಹಿ ದಿನಾಂಕ

ANNEXURE – V

ASSENT INFORMATION SHEET (12-17 years)

Study Title : A COMPARATIVE STUDY OF FRACTIONAL CO2 LASER WITH TOPICAL BIMATOPROST 0.03% VERSUS FRACTIONAL CO2 LASER WITH TOPICAL TRIAMCINOLONE ACETONIDE IN THE TREATMENT OF ALOPECIA AREATA

Place of study: R.L Jalappa Hospital and research centre, Tamaka, Kolar.

Aim: 1) To assess and compare the efficacy of Fractional CO2 Laser with Topical Bimatoprost 0.03% versus Fractional CO2 Laser with Topical Triamcinolone Acetonide in the treatment of Alopecia Areata.

2) To document the post procedural adverse effects of Fractional CO2 with Topical Bimatoprost 0.03% and Fractional Co2 Laser with Topical Triamcinolone Acetonide in the treatment of Alopecia areata.

All patients satisfying the inclusion criteria will be divided into two groups as follows:
GROUP A- participants will be treated with Fractional CO2 Laser in combination with Bimatoprost 0.03% within 2 minutes immediately after laser for 5 sittings with an interval gap of 3 weeks with topical Bimatoprost 0.03% once daily application between each sitting

GROUP B- participants will be treated with Fractional CO2 Laser in combination with Topical Triamcinolone acetonide Aqueous solution (2.5 mg/ml to 10 mg/ml) within 2 minutes immediately after laser for 5 sittings with an interval gap of 3 weeks between each sitting.

Group A Participants - A topical anaesthetic, containing a mixture of lidocaine-2.5% w/w + prilocaine-2.5% w/w in a cream base will be applied for 1 hour on the treatment area. After satisfactory anaesthesia is achieved, the treatment area will be cleaned with a mild cleanser. Eyes will be protected with eye shields. Fractional CO2 LASER will be then delivered to the Alopecia Areata site at the fluence of 50-60 mJ/cm2(trans epidermal pores are created in the skin) .Ice pack will be applied immediately. Then within 2 minutes of LASER procedure,

Topical Bimatoprost 0.03% will be applied and asked to continue twice daily application till next sitting.

GROUP B – A topical anaesthetic, containing a mixture of lidocaine-2.5% w/w + prilocaine-2.5% w/w in a cream base will be applied for 1 hour on the treatment area. After satisfactory anaesthesia is achieved, the treatment area will be cleaned with a mild cleanser. Eyes will be protected with eye shields. Fractional CO2 LASER will be then delivered to the Alopecia Areata site at the fluence of 50-60 mJ/cm2 (trans epidermal pores are created in the skin). Ice pack will be applied immediately. Then within 2 minutes of LASER procedure, Topical Triamcinolone Acetonide Aqueous solution (2.5 mg/ml to10 mg/ml) will be applied.

Participants are required to undergo serial photography at baseline and at subsequent sittings.

Please read the following information and discuss with your family members. You can ask any question regarding the study. If you agree to participate in this study we will collect information (as per proforma) from you. Relevant blood investigations will be carried out if required. This information collected will be used for dissertation and publication only.

All information collected from you will be kept confidential and will not be disclosed to any outsider. Your identity will not be revealed. The expenses required for the study will be funded by the study investigator. This study has been reviewed by the Institutional Ethics Committee and you are free to contact the member of the Institutional Ethics Committee. There is no compulsion to agree to this study. The care that you get will not change even if you don't wish to participate. You are required to sign/ provide thumb impression only if you voluntarily agree to participate in this study.

For any further clarification you can contact the study investigator:

Dr. HUSSAIN KOLSAWALA

Mobile no: 8105907922

E-mail id: hussainkolsa786@gmail.com

ಒಪ್ಪಿಗೆಯ ಮಾಹಿತಿ ಹಾಳೆ (12-17 ವರ್ಷಗಳು)

ಅಧ್ಯಯನದ ಶೀರ್ಷಿಕೆ : ಸಾಮಯಿಕ ಬೈಮಾಟೊಪ್ರೊಸ್ಟ್ 0.03% ಮತ್ತು ಫ್ರಾಕ್ಯನಲ್ CO2 ಲೇಸರ್ ಜೊತೆಗೆ ಫ್ರ್ಯಾಕ್ಯನಲ್ CO2 ಲೇಸರ್‌ನ ತುಲನಾತ್ಮಕ ಅಧ್ಯಯನವು ಚಿಕಿತ್ಸೆಯಲ್ಲಿ ಟಾಪಿಕಲ್ ಟ್ರಯಾಮ್ಸಿನೋಲೋನ್ ಅಸಿಟೋನೈಡ್ ಜೊತೆಗೆ

ಅಧ್ಯಯನ ಸ್ಥಳ: ಆರ್.ಎಲ್ ಜಾಲಪ್ಪ ಆಸ್ಪತ್ರೆ ಮತ್ತು ಸಂಶೋಧನಾ ಕೇಂದ್ರ, ಟಮಕ, ಕೋಲಾರ.

ಗುರಿ: 1) ಅಲೋಪೆಸಿಯಾ ಏರಿಯಾಟಾ ಚಿಕಿತ್ಸೆಯಲ್ಲಿ ಫ್ರ್ಯಾಕ್ಷನಲ್ CO2 ಲೇಸರ್‌ನ ಪರಿಣಾಮಕಾರಿತ್ವವನ್ನು 0.03% ಮತ್ತು ಫ್ರ್ಯಾಕ್ಷನಲ್ CO2 ಲೇಸರ್ ಜೊತೆಗೆ ಟಾಪಿಕಲ್ ಟ್ರಯಾಮ್ಸಿನೋಲೋನ್ ಅಸಿಟೋನೈಡ್‌ನೊಂದಿಗೆ ಹೋಲಿಸುವುದು.

2) ಅಲೋಪೆಸಿಯಾ ಅರೆಟಾ ಚಿಕಿತ್ಸೆಯಲ್ಲಿ ಫ್ರ್ಯಾಕ್ಷನಲ್ CO2 ನ ನಂತರದ ಪ್ರತಿಕೂಲ ಪರಿಣಾಮಗಳನ್ನು ದಾಖಲಿಸಲು.

ಸೇರ್ಪಡೆ ಮಾನದಂಡಗಳನ್ನು ಪೂರೈಸುವ ಎಲ್ಲಾ ರೋಗಿಗಳನ್ನು ಈ ಕೆಳಗಿನಂತೆ ಎರಡು ಗುಂಪುಗಳಾಗಿ ವಿಂಗಡಿಸಲಾಗಿದೆ:

ಗ್ರೂಪ್ ಎ- ಭಾಗವಹಿಸುವವರಿಗೆ 5 ಸಿಟ್ಟಿಂಗ್‌ಗಳಿಗೆ ಲೇಸರ್ ನಂತರ ತಕ್ಷಣವೇ 2 ನಿಮಿಷಗಳಲ್ಲಿ ಬಿಮಾಟೊಪ್ರೊಸ್ಟ್ 0.03% ನೊಂದಿಗೆ ಫ್ರ್ಯಾಕ್ಷನಲ್ CO2 ಲೇಸರ್‌ನೊಂದಿಗೆ ಚಿಕಿತ್ಸೆ ನೀಡಲಾಗುತ್ತದೆ, ಜೊತೆಗೆ 3 ವಾರಗಳ ಮಧ್ಯಂತರ ಅಂತರದೊಂದಿಗೆ ಸಾಮಯಿಕ ಬಿಮಾಟೊಪ್ರೊಸ್ಟ್ 0.03% ಪ್ರತಿ ಕುಳಿತುಕೊಳ್ಳುವ ನಡುವೆ ದಿನಕ್ಕೆ ಒಮ್ಮೆ ಅನ್ವಯಿಸಲಾಗುತ್ತದೆ.

ಗ್ರೂಪ್ ಬಿ- ಭಾಗವಹಿಸುವವರಿಗೆ ಫ್ರ್ಯಾಕ್ಷನಲ್ CO2 ಲೇಸರ್‌ನೊಂದಿಗೆ ಟಾಪಿಕಲ್ ಟ್ರಯಾಮ್ಸಿನೋಲೋನ್ ಅಸಿಟೋನೈಡ್ ಜಲೀಯ ದ್ರಾವಣದೊಂದಿಗೆ (2.5 mg/ml ನಿಂದ 10 mg/ml) ಲೇಸರ್ ನಂತರ ತಕ್ಷಣವೇ 2 ನಿಮಿಷಗಳಲ್ಲಿ 5 ಸಿಟ್ಟಿಂಗ್‌ಗಳಿಗೆ 3 ವಾರಗಳ ಮಧ್ಯಂತರದೊಂದಿಗೆ ಚಿಕಿತ್ಸೆ ನೀಡಲಾಗುತ್ತದೆ.

ಗ್ರೂಪ್ ಎ ಭಾಗವಹಿಸುವವರು - ಕ್ರೀಮ್ ಬೇಸ್‌ನಲ್ಲಿ ಲಿಡೋಕೇಯ್ನ್-2.5% w/w + ಪ್ರಿಲೋಕೈನ್-2.5% w/w ಮಿಶ್ರಣವನ್ನು ಒಳಗೊಂಡಿರುವ ಸಾಮಯಿಕ ಅರಿವಳಿಕೆಯನ್ನು ಚಿಕಿತ್ಸೆಯ ಪ್ರದೇಶದ ಮೇಲೆ 1 ಗಂಟೆ ಅನ್ವಯಿಸಲಾಗುತ್ತದೆ. ತೃಪ್ತಿಕರ ಅರಿವಳಿಕೆ ಸಾಧಿಸಿದ ನಂತರ, ಚಿಕಿತ್ಸೆಯ ಪ್ರದೇಶವನ್ನು ಸೌಮ್ಯವಾದ ಕ್ಲೆನ್ಸರ್ನೊಂದಿಗೆ ಸ್ವಚ್ಛಗೊಳಿಸಲಾಗುತ್ತದೆ. ಕಣ್ಣುಗಳನ್ನು ಕಣ್ಣಿನ ಗುರಾಣಿಗಳಿಂದ ರಕ್ಷಿಸಲಾಗುವುದು. ಫ್ರಾಕ್ಷನಲ್ CO2 ಲೇಸರ್ ಅನ್ನು ನಂತರ 50-60 mJ/cm2 (ಚರ್ಮದಲ್ಲಿ ಟ್ರಾನ್ಸ್ ಎಪಿಡರ್ಮಲ್ ರಂಧ್ರಗಳನ್ನು ರಚಿಸಲಾಗುತ್ತದೆ) ದ ಅಲೋಪೆಸಿಯಾ ಏರಿಯಾಟಾ ಸೈಟ್‌ಗೆ ತಲುಪಿಸಲಾಗುತ್ತದೆ. ಐಸ್ ಪ್ಯಾಕ್ ಅನ್ನು ತಕ್ಷಣವೇ ಅನ್ವಯಿಸಲಾಗುತ್ತದೆ. ನಂತರ ಲೇಸರ್ ಕಾರ್ಯವಿಧಾನದ 2 ನಿಮಿಷಗಳಲ್ಲಿ, ಟಾಪಿಕಲ್ ಬಿಮಾಟೊಪ್ರೊಸ್ಟ್ 0.03% ಅನ್ನು ಅನ್ವಯಿಸಲಾಗುತ್ತದೆ ಮತ್ತು ಮುಂದಿನ ಕುಳಿತುಕೊಳ್ಳುವವರೆಗೆ ದಿನಕ್ಕೆ ಎರಡು ಬಾರಿ ಅನ್ವಯಿಸುವುದನ್ನು ಮುಂದುವರಿಸಲು ಕೇಳಲಾಗುತ್ತದೆ.

ಗ್ರೂಪ್ ಬಿ - ಒಂದು ಸಾಮಯಿಕ ಅರಿವಳಿಕೆ, ಕ್ರೀಮ್ ಬೇಸ್ನಲ್ಲಿ ಲಿಡೋಕೇಯ್ನ್-2.5% w/w + prilocaine-2.5% w/w ಮಿಶ್ರಣವನ್ನು ಹೊಂದಿರುವ 1 ಗಂಟೆ ಚಿಕಿತ್ಸೆಯ ಪ್ರದೇಶದಲ್ಲಿ ಅನ್ವಯಿಸಲಾಗುತ್ತದೆ. ತೃಪ್ತಿಕರ ಅರಿವಳಿಕೆ ಸಾಧಿಸಿದ ನಂತರ, ಚಿಕಿತ್ಸೆಯ ಪ್ರದೇಶವನ್ನು ಸೌಮ್ಯವಾದ ಕ್ಲೆನ್ಸರ್ನೊಂದಿಗೆ ಸ್ವಚ್ಛಗೊಳಿಸಲಾಗುತ್ತದೆ. ಕಣ್ಣುಗಳನ್ನು ಕಣ್ಣಿನ ಗುರಾಣಿಗಳಿಂದ

ರಕ್ಷಿಸಲಾಗುವುದು. ಫ್ರಾಕ್ಷನಲ್ CO2 ಲೇಸರ್ ಅನ್ನು 50-60 mJ/cm2 ನ ಫ್ಲೂಯೆನ್ಸ್‌ನಲ್ಲಿ ಅಲೋಪೆಸಿಯಾ ಏರಿಯಾಟಾ ಸೈಟ್‌ಗೆ ತಲುಪಿಸಲಾಗುತ್ತದೆ (ಟ್ರಾನ್ಸ್ ಎಪಿಡರ್ಮಲ್ ರಂಧ್ರಗಳನ್ನು ಚರ್ಮದಲ್ಲಿ ರಚಿಸಲಾಗುತ್ತದೆ). ಐಸ್ ಪ್ಯಾಕ್ ಅನ್ನು ತಕ್ಷಣವೇ ಅನ್ವಯಿಸಲಾಗುತ್ತದೆ. ನಂತರ ಲೇಸರ್ ಕಾರ್ಯವಿಧಾನದ 2 ನಿಮಿಷಗಳಲ್ಲಿ, ಟಾಪಿಕಲ್ ಟ್ರಯಾಮ್ಸಿನೋಲೋನ್ ಅಸಿಟೋನೈಡ್ ಜಲೀಯ ದ್ರಾವಣವನ್ನು (2.5 mg/ml to10 mg/ml) ಅನ್ವಯಿಸಲಾಗುತ್ತದೆ.

ಭಾಗವಹಿಸುವವರು ಬೇಸ್‌ಲೈನ್‌ನಲ್ಲಿ ಮತ್ತು ನಂತರದ ಸಿಟ್ಟಿಂಗ್‌ಗಳಲ್ಲಿ ಸರಣಿ ಛಾಯಾಗ್ರಹಣಕ್ಕೆ ಒಳಗಾಗಬೇಕಾಗುತ್ತದೆ.

ದಯವಿಟ್ಟು ಕೆಳಗಿನ ಮಾಹಿತಿಯನ್ನು ಓದಿ ಮತ್ತು ನಿಮ್ಮ ಕುಟುಂಬದ ಸದಸ್ಯರೊಂದಿಗೆ ಚರ್ಚಿಸಿ. ಅಧ್ಯಯನಕ್ಕೆ ಸಂಬಂಧಿಸಿದಂತೆ ನೀವು ಯಾವುದೇ ಪ್ರಶ್ನೆಯನ್ನು ಕೇಳಬಹುದು. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ನೀವು ಒಪ್ಪಿದರೆ ನಾವು ನಿಮ್ಮಿಂದ ಮಾಹಿತಿಯನ್ನು (ಪ್ರೊಫಾರ್ಮಾ ಪ್ರಕಾರ) ಸಂಗ್ರಹಿಸುತ್ತೇವೆ. ಅಗತ್ಯವಿದ್ದರೆ ಸಂಬಂಧಿತ ರಕ್ತ ಪರೀಕ್ಷೆಗಳನ್ನು ಕೈಗೊಳ್ಳಲಾಗುತ್ತದೆ. ಸಂಗ್ರಹಿಸಿದ ಈ ಮಾಹಿತಿಯನ್ನು ಪ್ರಬಂಧ ಮತ್ತು ಪ್ರಕಟಣೆಗೆ ಮಾತ್ರ ಬಳಸಲಾಗುತ್ತದೆ.

ನಿಮ್ಮಿಂದ ಸಂಗ್ರಹಿಸಲಾದ ಎಲ್ಲಾ ಮಾಹಿತಿಯನ್ನು ಗೌಪ್ಯವಾಗಿ ಇರಿಸಲಾಗುತ್ತದೆ ಮತ್ತು ಯಾವುದೇ ಹೊರಗಿನವರಿಗೆ ಬಹಿರಂಗಪಡಿಸಲಾಗುವುದಿಲ್ಲ. ನಿಮ್ಮ ಗುರುತನ್ನು ಬಹಿರಂಗಪಡಿಸಲಾಗುವುದಿಲ್ಲ. ತನಿಖೆಗಳಿಗೆ ಅಗತ್ಯವಿರುವ ವೆಚ್ಚಗಳನ್ನು ಅಧ್ಯಯನ ತನಿಖಾಧಿಕಾರಿಗಳು ಧನಸಹಾಯ ಮಾಡುತ್ತಾರೆ. ಈ ಅಧ್ಯಯನವನ್ನು ಸಾಂಸ್ಥಿಕ ನೀತಿಶಾಸ್ತ್ರ ಸಮಿತಿಯು ಪರಿಶೀಲಿಸಿದೆ ಮತ್ತು ನೀವು ಸಾಂಸ್ಥಿಕ ನೀತಿಶಾಸ್ತ್ರ ಸಮಿತಿಯ ಸದಸ್ಯರನ್ನು ಸಂಪರ್ಕಿಸಲು ಮುಕ್ತರಾಗಿದ್ದೀರಿ. ಈ ಅಧ್ಯಯನವನ್ನು ಒಪ್ಪಿಕೊಳ್ಳಲು ಯಾವುದೇ ಒತ್ತಾಯವಿಲ್ಲ. ನೀವು ಭಾಗವಹಿಸಲು ಬಯಸದಿದ್ದರೂ ಸಹ ನೀವು ಪಡೆಯುವ ಕಾಳಜಿಯು ಬದಲಾಗುವುದಿಲ್ಲ. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ನೀವು ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ ಸಮ್ಮತಿಸಿದರೆ ಮಾತ್ರ ನೀವು ಸಹಿ/ಹೆಬ್ಬೆರಳಿನ ಗುರುತನ್ನು ಒದಗಿಸಬೇಕಾಗುತ್ತದೆ.

ಯಾವುದೇ ಹೆಚ್ಚಿನ ಸ್ಪಷ್ಟೀಕರಣಕ್ಕಾಗಿ ನೀವು ಅಧ್ಯಯನ ತನಿಖಾಧಿಕಾರಿಯನ್ನು ಸಂಪರ್ಕಿಸಬಹುದು:

ಡಾ. ಹುಸೇನ್ ಕೋಲ್ಸವಾಲ

ಮೊಬೈಲ್ ಸಂಖ್ಯೆ: 8105907922

ಇ-ಮೇಲ್ ಐಡಿ: hussainkolsa786@gmail.com

ANNEXURE – VI

PARENT/ GUARDIAN CONSENT FORM (10-17 years)

Title of the study: A COMPARATIVE STUDY OF FRACTIONAL CO2 LASER WITH TOPICAL BIMATOPROST 0.03% VERSUS FRACTIONAL CO2 LASER WITH TOPICAL TRIAMCINOLONE ACETONIDE IN THE TREATMENT OF ALOPECIA AREATA

Name of the Participant:

Name of the Principal investigator: Dr.HUSSAIN KOLSAWALA

Name of the Institution: R.L Jalappa Hospital and Research Centre, Tamaka, Kolar.

Documentation of the informed consent

I Mr./Mrs. _____F/o or M/o or G/o Mr/Ms._____ have been explained in my own understandable language, that my child/ward will be included in the study which is **A COMPARATIVE STUDY OF FRACTIONAL CO2 LASER WITH TOPICAL BIMATOPROST 0.03% VERSUS FRACTIONAL CO2 LASER WITH TOPICAL TRIAMCINOLONE ACETONIDE IN THE TREATMENT OF ALOPECIA AREATA**

1. I have read and understood this consent form and the information provided to me. I have had the consent document explained to me.
2. I have been explained that my child's clinical findings, investigations, will be assessed and documented for study purpose
3. I have informed the investigator of all the treatments my child has been taking or have taken in the past.
4. I agree for my child to cooperate with the investigator and will inform her immediately if my child suffers unusual symptoms.
5. My child has not participated in any research study at any time .
6. I am aware of the fact that my child can opt out of the study at any time without having to give any reason and this will not affect my relation with my doctor or the treatment for my childs's ailment in this hospital.
7. I have understood that all the details found during the study are kept confidential and while publishing or sharing of the findings, the details will be masked. I hereby give

permission to the investigators to release the information obtained from me about my as a result of participation in this study

8. I have decided for my child to be included in this research study
9. I am aware that if I have any question during this study, I should contact my principal investigator mobile number for enquiries.
10. The principal investigator will bear the cost of study.

I was free to ask any questions which have been answered and clarified. I do hereby give my consent for my child to be included as a participant in the study.

By signing this consent form I attest that the information given in this document has been clearly explained to me and apparently understood by me. I will be given a copy of this consent document.

Participant's initials:_____

Name and signature/thumb impression of the guardian

_____	_____	_____
Name	Signature	Date

Name and signature of witness

_____	_____	_____
Name	Signature	Date

Name and Signature of the investigator or his representative obtaining consent:

_____	_____	_____
Name	Signature	Date

ಪೋಷಕ/ಪಾಲಕರ ಸಮ್ಮತಿ ನಮೂನೆ (10-17 ವರ್ಷಗಳು)

ಅಧ್ಯಯನದ ಶೀರ್ಷಿಕೆ: ಸಾಮಯಿಕ ಬೈಮಾಟೊಪ್ರೊಸ್ಟ್ 0.03% ವಿರುದ್ಧ ಫ್ರಾಕ್ಶನಲ್ CO2 ಲೇಸರ್ ಜೊತೆಗೆ ಫ್ರ್ಯಾಕ್ಶನಲ್ CO2 ಲೇಸರ್‌ನ ತುಲನಾತ್ಮಕ ಅಧ್ಯಯನವು ಟಾಪಿಕಲ್ ಟ್ರೈಯಾಮಿನೋಲೋನ್ ಅಸಿಟೋನೈಡ್ ಜೊತೆಗೆ ಚಿಕಿತ್ಸೆಯಲ್ಲಿ

ಭಾಗವಹಿಸುವವರ ಹೆಸರು:

ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿಯ ಹೆಸರು: ಡಾ.ಹುಸೇನ್ ಕೋಲ್ಸವಾಲಾ

ಸಂಸ್ಥೆಯ ಹೆಸರು: R.L ಜಾಲಪ್ಪ ಆಸ್ಪತ್ರೆ ಮತ್ತು ಸಂಶೋಧನಾ ಕೇಂದ್ರ, ಟಮಕಾ,

ಕೋಲಾರ.

ತಿಳುವಳಿಕೆಯುಳ್ಳ ಒಪ್ಪಿಗೆಯ ದಾಖಲೆ

ನಾನು ಶ್ರೀ/ಶ್ರೀಮತಿ. _____ F/O ಅಥವಾ M/O ಅಥವಾ G/O MR/MS ._____ ಅನ್ನು ನನ್ನ ಸ್ವಂತ ಅರ್ಥವಾಗುವ ಭಾಷೆಯಲ್ಲಿ ವಿವರಿಸಲಾಗಿದೆ, ನನ್ನ ಮಗು/ವಾರ್ಡ್ ಅನ್ನು ಅಧ್ಯಯನದಲ್ಲಿ ಸೇರಿಸಲಾಗುವುದು, ಇದು ಸಾಮಯಿಕ ಬೈಮಾಟೊಪ್ರೊಸ್ಟ್ 0.03% ನೊಂದಿಗೆ ಭಾಗಶಃ CO2 ಲೇಸರ್ನ ತುಲನಾತ್ಮಕ ಅಧ್ಯಯನವಾಗಿದೆ ಅಲೋಪೆಸಿಯಾ ಏರಿಯಾಟಾದ ಚಿಕಿತ್ಸೆಯಲ್ಲಿ ಸಾಮಯಿಕ ಟ್ರಯಾಮಿಸಿನೋಲೋನ್ ಅಸಿಟೋನೈಡ್‌ನೊಂದಿಗೆ ಫ್ರಾಕ್ಶನಲ್ CO2 ಲೇಸರ್

1. ನಾನು ಈ ಒಪ್ಪಿಗೆ ನಮೂನೆ ಮತ್ತು ನನಗೆ ಒದಗಿಸಿದ ಮಾಹಿತಿಯನ್ನು ಓದಿದ್ದೇನೆ ಮತ್ತು ಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ.

2. ನಾನು ಒಪ್ಪಿಗೆಯ ದಾಖಲೆಯನ್ನು ನನಗೆ ವಿವರಿಸಿದ್ದೇನೆ.

3. ನನ್ನ ಮಗುವಿನ ಕ್ಲಿನಿಕಲ್ ಸಂಶೋಧನೆಗಳು, ತನಿಖೆಗಳನ್ನು ಮೌಲ್ಯಮಾಪನ ಮಾಡಲಾಗುತ್ತದೆ ಮತ್ತು ಅಧ್ಯಯನ ಉದ್ದೇಶಕ್ಕಾಗಿ ದಾಖಲಿಸಲಾಗುತ್ತದೆ ಎಂದು ನನಗೆ ವಿವರಿಸಲಾಗಿದೆ

4. ನನ್ನ ಮಗು ಈ ಹಿಂದೆ ತೆಗೆದುಕೊಳ್ಳುತ್ತಿರುವ ಅಥವಾ ತೆಗೆದುಕೊಂಡಿರುವ ಎಲ್ಲಾ ಚಿಕಿತ್ಸೆಗಳ ಬಗ್ಗೆ ನಾನು ತನಿಖಾಧಿಕಾರಿಗೆ ತಿಳಿಸಿದ್ದೇನೆ.

5. ನನ್ನ ಮಗುವಿಗೆ ತನಿಖಾಧಿಕಾರಿಯೊಂದಿಗೆ ಸಹಕರಿಸಲು ನಾನು ಒಪ್ಪುತ್ತೇನೆ ಮತ್ತು ನನ್ನ ಮಗು ಅಸಾಮಾನ್ಯ ರೋಗಲಕ್ಷಣಗಳನ್ನು ಅನುಭವಿಸಿದರೆ ತಕ್ಷಣವೇ ಅವಳಿಗೆ ತಿಳಿಸುತ್ತೇನೆ.

6. ನನ್ನ ಮಗು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಯಾವುದೇ ಸಂಶೋಧನಾ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಿಲ್ಲ.

7. ನನ್ನ ಮಗು ಯಾವುದೇ ಕಾರಣವನ್ನು ನೀಡದೆ ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಅಧ್ಯಯನದಿಂದ ಹೊರಗುಳಿಯಬಹುದು ಮತ್ತು ಇದು ನನ್ನ ವೈದ್ಯರೊಂದಿಗಿನ ನನ್ನ ಸಂಬಂಧ ಅಥವಾ ಈ ಆಸ್ಪತ್ರೆಯಲ್ಲಿನ ನನ್ನ ಮಗುವಿನ ಕಾಯಿಲೆಯ ಚಿಕಿತ್ಸೆಯ ಮೇಲೆ ಪರಿಣಾಮ ಬೀರುವುದಿಲ್ಲ ಎಂಬ ಅಂಶದ ಬಗ್ಗೆ ನನಗೆ ತಿಳಿದಿದೆ.

8. ಅಧ್ಯಯನದ ಸಮಯದಲ್ಲಿ ಕಂಡುಬರುವ ಎಲ್ಲಾ ವಿವರಗಳನ್ನು ಗೌಪ್ಯವಾಗಿ ಇರಿಸಲಾಗುತ್ತದೆ ಮತ್ತು ಸಂಶೋಧನೆಗಳನ್ನು ಪ್ರಕಟಿಸುವಾಗ ಅಥವಾ ಹಂಚಿಕೊಳ್ಳುವಾಗ, ವಿವರಗಳನ್ನು ಮರೆಮಾಚಲಾಗುತ್ತದೆ ಎಂದು ನಾನು ಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಿದ ಪರಿಣಾಮವಾಗಿ ನನ್ನ ಬಗ್ಗೆ ನನ್ನಿಂದ ಪಡೆದ ಮಾಹಿತಿಯನ್ನು ಬಿಡುಗಡೆ ಮಾಡಲು ತನಿಖಾಧಿಕಾರಿಗಳಿಗೆ ನಾನು ಈ ಮೂಲಕ ಅನುಮತಿ ನೀಡುತ್ತೇನೆ

9. ನನ್ನ ಮಗುವನ್ನು ಈ ಸಂಶೋಧನಾ ಅಧ್ಯಯನದಲ್ಲಿ ಸೇರಿಸಲು ನಾನು ನಿರ್ಧರಿಸಿದ್ದೇನೆ.

10. ಈ ಅಧ್ಯಯನದ ಸಮಯದಲ್ಲಿ ನಾನು ಯಾವುದೇ ಪ್ರಶ್ನೆಗಳನ್ನು ಹೊಂದಿದ್ದರೆ, ವಿಚಾರಣೆಗಾಗಿ ನಾನು ನನ್ನ ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿಯ ಮೊಬೈಲ್ ಸಂಖ್ಯೆಯನ್ನು ಸಂಪರ್ಕಿಸಬೇಕು ಎಂದು ನನಗೆ ತಿಳಿದಿದೆ.

11. ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿಯು ಅಧ್ಯಯನದ ವೆಚ್ಚವನ್ನು ಭರಿಸುತ್ತಾರೆ.

ಉತ್ತರಿಸಿರುವ ಮತ್ತು ಸ್ಪಷ್ಟಪಡಿಸಿದ ಯಾವುದೇ ಪ್ರಶ್ನೆಗಳನ್ನು ಕೇಳಲು ನಾನು ಸ್ವತಂತ್ರನಾಗಿದ್ದೆ. ನನ್ನ ಮಗುವನ್ನು ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳಲು ನಾನು ಈ ಮೂಲಕ ನನ್ನ ಒಪ್ಪಿಗೆಯನ್ನು ನೀಡುತ್ತೇನೆ.

ಈ ಸಮ್ಮತಿಯ ನಮೂನೆಗೆ ಸಹಿ ಹಾಕುವ ಮೂಲಕ ಈ ಡಾಕ್ಯುಮೆಂಟ್‌ನಲ್ಲಿ ನೀಡಲಾದ ಮಾಹಿತಿಯನ್ನು ನನಗೆ ಸ್ಪಷ್ಟವಾಗಿ ವಿವರಿಸಲಾಗಿದೆ ಮತ್ತು ನಾನು ಸ್ಪಷ್ಟವಾಗಿ ಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ ಎಂದು ನಾನು ದೃಢೀಕರಿಸುತ್ತೇನೆ. ಈ ಒಪ್ಪಿಗೆಯ ದಾಖಲೆಯ ಪ್ರತಿಯನ್ನು ನನಗೆ ನೀಡಲಾಗುವುದು.

ಭಾಗವಹಿಸುವವರ ಮೊದಲಕ್ಷರಗಳು:_____

ಪೋಷಕರ ಹೆಸರು ಮತ್ತು ಸಹಿ/ಹೆಬ್ಬೆರಳಿನ ಗುರುತು

_____

ಹೆಸರು ಸಹಿ ದಿನಾಂಕ

ಸಾಕ್ಷಿಯ ಹೆಸರು ಮತ್ತು ಸಹಿ

_____

ಹೆಸರು ಸಹಿ ದಿನಾಂಕ

ತನಿಖಾಧಿಕಾರಿಯ ಹೆಸರು ಮತ್ತು ಸಹಿ ಅಥವಾ ಅವರ ಪ್ರತಿನಿಧಿ ಒಪ್ಪಿಗೆಯನ್ನು ಪಡೆಯುವುದು:

_____

ಹೆಸರು ಸಹಿ ದಿನಾಂಕ

ANNEXURE – VII

PARENT/ GUARDIAN INFORMATION SHEET (10-17 years)

Study title : A COMPARATIVE STUDY OF FRACTIONAL CO2 LASER WITH TOPICAL BIMATOPROST 0.03% VERSUS FRACTIONAL CO2 LASER WITH TOPICAL TRIAMCINOLONE ACETONIDE IN THE TREATMENT OF ALOPECIA AREATA

Study site: R.L Jalappa Hospital and research centre, Tamaka, Kolar.

Aim: 1) To assess and compare the efficacy of Fractional CO2 Laser with Topical Bimatoprost 0.03% versus Fractional CO2 Laser with Topical Triamcinolone Acetonide in the treatment of Alopecia Areata.

2) To document the post procedural adverse effects of Fractional CO2 with Topical Bimatoprost 0.03% and Fractional Co2 Laser with Topical Triamcinolone Acetonide in the treatment of Alopecia areata.

All patients satisfying the inclusion criteria will be divided into two groups as follows:
GROUP A- participants will be treated with Fractional CO2 Laser in combination with Bimatoprost 0.03% within 2 minutes immediately after laser for 5 sittings with an interval gap of 3 weeks with topical Bimatoprost 0.03% once daily application between each sitting

GROUP B- participants will be treated with Fractional CO2 Laser in combination with Topical Triamcinolone acetonide Aqueous solution (2.5 mg to 10 mg/ml) within 2 minutes immediately after laser for 5 sittings with an interval gap of 3 weeks between each sitting.

Group A Participants - A topical anaesthetic, containing a mixture of lidocaine-2.5% w/w + prilocaine-2.5% w/w in a cream base will be applied for 1 hour on the treatment area. After satisfactory anaesthesia is achieved, the treatment area will be cleaned with a mild cleanser. Eyes will be protected with eye shields. Fractional CO2 LASER will be then delivered to the Alopecia Areata site at the fluence of 50-60 mJ/cm2(trans epidermal pores are created in the skin) .Ice pack will be applied immediately. Then within 2 minutes of LASER procedure, Topical Bimatoprost 0.03% will be applied and asked to continue twice daily application till next sitting.

GROUP B – A topical anaesthetic, containing a mixture of lidocaine-2.5% w/w + prilocaine-2.5% w/w in a cream base will be applied for 1 hour on the treatment area. After satisfactory anaesthesia is achieved, the treatment area will be cleaned with a mild cleanser. Eyes will be protected with eye shields. Fractional CO2 LASER will be then delivered to the Alopecia Areata site at the fluence of 50-60 mJ/cm2 (trans epidermal pores are created in the skin). Ice pack will be applied immediately. Then within 2 minutes of LASER procedure, Topical Triamcinolone Acetonide Aqueous solution (2.5mg/ml to10 mg/ml) will be applied.

Participants are required to undergo serial photography at baseline and at subsequent sittings.

Please read the following information and discuss with your family members. You can ask any question regarding the study. If you agree for your child/ward to participate in this study we will collect information (as per proforma) from you and your child. Relevant blood investigations will be carried out if required. This information collected will be used for dissertation and publication only.

All information collected from you regarding your child/ward will be kept confidential and will not be disclosed to any outsider. Your child's/ward's identity will not be revealed. The expenses required for the study will be funded by the study investigator. This study has been reviewed by the Institutional Ethics Committee and you are free to contact the member of the Institutional Ethics Committee. There is no compulsion to agree to this study. The care your child will get will not change if you don't wish to participate. You are required to sign/ provide thumb impression only if you voluntarily agree for your child to participate in this study.

For any further clarification you can contact the study investigator:

Dr. HUSSAIN KOLSAWALA
Mobile no: 8105907922
E-mail id: hussainkolsa786@gmail.com

ಪೋಷಕ/ರಕ್ಷಕ ಮಾಹಿತಿ ಹಾಳೆ (10-17 ವರ್ಷಗಳು)

ಅಧ್ಯಯನದ ಶೀರ್ಷಿಕೆ : ಸಾಮಯಿಕ ಬೈಮಾಟೊಪ್ರೊಸ್ಟ್ 0.03% ವಿತ್ ಫ್ರ್ಯಾಕ್ಶನಲ್ CO2 ಲೇಸರ್‌ನ ತುಲನಾತ್ಮಕ ಅಧ್ಯಯನ ಮತ್ತು ಫ್ರ್ಯಾಕ್ಶನಲ್ CO2 ಲೇಸರ್ ಜೊತೆಗೆ ಟಾಪಿಕಲ್ ಟ್ರೈಯಾಮಿನೋಲೋನ್ ಅಸಿಟೋನೈಡ್ ಚಿಕಿತ್ಸೆಯಲ್ಲಿ

ಅಧ್ಯಯನ ಸ್ಥಳ: ಆರ್.ಎಲ್ ಜಾಲಪ್ಪ ಆಸ್ಪತ್ರೆ ಮತ್ತು ಸಂಶೋಧನಾ ಕೇಂದ್ರ, ಟಮಕ, ಕೋಲಾರ.

ಗುರಿ: 1) ಅಲೋಪೆಸಿಯಾ ಏರಿಯಾಟಾ ಚಿಕಿತ್ಸೆಯಲ್ಲಿ ಫ್ರ್ಯಾಕ್ಷನಲ್ CO2 ಲೇಸರ್‌ನ ಪರಿಣಾಮಕಾರಿತ್ವವನ್ನು 0.03% ಮತ್ತು ಫ್ರ್ಯಾಕ್ಷನಲ್ CO2 ಲೇಸರ್ ಜೊತೆಗೆ ಟಾಪಿಕಲ್ ಟ್ರಯಾಮ್ಸಿನೋಲೋನ್ ಅಸಿಟೋನೈಡ್‌ನೊಂದಿಗೆ ಹೋಲಿಸುವುದು.

2) ಅಲೋಪೆಸಿಯಾ ಅರೆಟಾ ಚಿಕಿತ್ಸೆಯಲ್ಲಿ ಫ್ರ್ಯಾಕ್ಷನಲ್ CO2 ನ ನಂತರದ ಪ್ರತಿಕೂಲ ಪರಿಣಾಮಗಳನ್ನು ದಾಖಲಿಸಲು.

ಸೇರ್ಪಡೆ ಮಾನದಂಡಗಳನ್ನು ಪೂರೈಸುವ ಎಲ್ಲಾ ರೋಗಿಗಳನ್ನು ಈ ಕೆಳಗಿನಂತೆ ಎರಡು ಗುಂಪುಗಳಾಗಿ ವಿಂಗಡಿಸಲಾಗಿದೆ:

ಗ್ರೂಪ್ ಎ- ಭಾಗವಹಿಸುವವರಿಗೆ 5 ಸಿಟ್ಟಿಂಗ್‌ಗಳಿಗೆ ಲೇಸರ್ ನಂತರ ತಕ್ಷಣವೇ 2 ನಿಮಿಷಗಳಲ್ಲಿ ಬಿಮಾಟೊಪ್ರೊಸ್ಟ್ 0.03% ನೊಂದಿಗೆ ಫ್ರ್ಯಾಕ್ಷನಲ್ CO2 ಲೇಸರ್‌ನೊಂದಿಗೆ ಚಿಕಿತ್ಸೆ ನೀಡಲಾಗುತ್ತದೆ, ಜೊತೆಗೆ 3 ವಾರಗಳ ಮಧ್ಯಂತರ ಅಂತರದೊಂದಿಗೆ ಸಾಮಯಿಕ ಬಿಮಾಟೊಪ್ರೊಸ್ಟ್ 0.03% ಪ್ರತಿ ಕುಳಿತುಕೊಳ್ಳುವ ನಡುವೆ ದಿನಕ್ಕೆ ಒಮ್ಮೆ ಅನ್ವಯಿಸಲಾಗುತ್ತದೆ.

ಗ್ರೂಪ್ ಬಿ- ಭಾಗವಹಿಸುವವರಿಗೆ ಫ್ರ್ಯಾಕ್ಷನಲ್ CO2 ಲೇಸರ್‌ನೊಂದಿಗೆ ಟಾಪಿಕಲ್ ಟ್ರಯಾಮ್ಸಿನೋಲೋನ್ ಅಸಿಟೋನೈಡ್ ಜಲೀಯ ದ್ರಾವಣದೊಂದಿಗೆ (2.5 mg ನಿಂದ 10 mg/ml) ಲೇಸರ್ ನಂತರ ತಕ್ಷಣವೇ 2 ನಿಮಿಷಗಳಲ್ಲಿ 5 ಸಿಟ್ಟಿಂಗ್‌ಗಳಿಗೆ 3 ವಾರಗಳ ಮಧ್ಯಂತರದೊಂದಿಗೆ ಚಿಕಿತ್ಸೆ ನೀಡಲಾಗುತ್ತದೆ.

ಗ್ರೂಪ್ ಎ ಭಾಗವಹಿಸುವವರು - ಕ್ರೀಮ್ ಬೇಸ್‌ನಲ್ಲಿ ಲಿಡೋಕೇಯ್ನ್-2.5% w/w + ಪ್ರಿಲೋಕೈನ್-2.5% w/w ಮಿಶ್ರಣವನ್ನು ಒಳಗೊಂಡಿರುವ ಸಾಮಯಿಕ ಅರಿವಳಿಕೆಯನ್ನು ಚಿಕಿತ್ಸೆಯ ಪ್ರದೇಶದ ಮೇಲೆ 1 ಗಂಟೆ ಅನ್ವಯಿಸಲಾಗುತ್ತದೆ. ತೃಪ್ತಿಕರ ಅರಿವಳಿಕೆ ಸಾಧಿಸಿದ ನಂತರ, ಚಿಕಿತ್ಸೆಯ ಪ್ರದೇಶವನ್ನು ಸೌಮ್ಯವಾದ ಕ್ಲೆನ್ಸರ್ನೊಂದಿಗೆ ಸ್ವಚ್ಛಗೊಳಿಸಲಾಗುತ್ತದೆ. ಕಣ್ಣುಗಳನ್ನು ಕಣ್ಣಿನ ಗುರಾಣಿಗಳಿಂದ ರಕ್ಷಿಸಲಾಗುವುದು. ಫ್ರಾಕ್ಷನಲ್ CO2 ಲೇಸರ್ ಅನ್ನು ನಂತರ 50-60 mJ/cm2 (ಚರ್ಮದಲ್ಲಿ ಟ್ರಾನ್ಸ್ ಎಪಿಡರ್ಮಲ್ ರಂಧ್ರಗಳನ್ನು ರಚಿಸಲಾಗುತ್ತದೆ) ದ ಅಲೋಪೆಸಿಯಾ ಏರಿಯಾಟಾ ಸೈಟ್‌ಗೆ ತಲುಪಿಸಲಾಗುತ್ತದೆ. ಐಸ್ ಪ್ಯಾಕ್ ಅನ್ನು ತಕ್ಷಣವೇ ಅನ್ವಯಿಸಲಾಗುತ್ತದೆ. ನಂತರ ಲೇಸರ್ ಕಾರ್ಯವಿಧಾನದ 2 ನಿಮಿಷಗಳಲ್ಲಿ, ಟಾಪಿಕಲ್ ಬಿಮಾಟೊಪ್ರೊಸ್ಟ್ 0.03% ಅನ್ನು ಅನ್ವಯಿಸಲಾಗುತ್ತದೆ ಮತ್ತು ಮುಂದಿನ ಕುಳಿತುಕೊಳ್ಳುವವರೆಗೆ ದಿನಕ್ಕೆ ಎರಡು ಬಾರಿ ಅನ್ವಯಿಸುವುದನ್ನು ಮುಂದುವರಿಸಲು ಕೇಳಲಾಗುತ್ತದೆ.

ಗ್ರೂಪ್ ಬಿ - ಒಂದು ಸಾಮಯಿಕ ಅರಿವಳಿಕೆ, ಕ್ರೀಮ್ ಬೇಸ್ನಲ್ಲಿ ಲಿಡೋಕೇಯ್ನ್-2.5% w/w + prilocaine-2.5% w/w ಮಿಶ್ರಣವನ್ನು ಹೊಂದಿರುವ 1 ಗಂಟೆ ಚಿಕಿತ್ಸೆಯ ಪ್ರದೇಶದಲ್ಲಿ ಅನ್ವಯಿಸಲಾಗುತ್ತದೆ. ತೃಪ್ತಿಕರ ಅರಿವಳಿಕೆ ಸಾಧಿಸಿದ ನಂತರ,

ಚಿಕಿತ್ಸೆಯ ಪ್ರದೇಶವನ್ನು ಸೌಮ್ಯವಾದ ಕ್ಲೆನ್ಸರ್ನೊಂದಿಗೆ ಸ್ವಚ್ಛಗೊಳಿಸಲಾಗುತ್ತದೆ. ಕಣ್ಣುಗಳನ್ನು ಕಣ್ಣಿನ ಗುರಾಣಿಗಳಿಂದ ರಕ್ಷಿಸಲಾಗುವುದು. ಫ್ರಾಕ್ಷನಲ್ CO2 ಲೇಸರ್ ಅನ್ನು 50-60 mJ/cm2 ನ ಫ್ಲೂಯೆನ್ಸ್ನಲ್ಲಿ ಅಲೋಪೆಸಿಯಾ ಅರಿಯಾಟಾ ಸೈಟ್ಗೆ ತಲುಪಿಸಲಾಗುತ್ತದೆ (ಟ್ರಾನ್ಸ್ ಎಪಿಡರ್ಮಲ್ ರಂಧ್ರಗಳನ್ನು ಚರ್ಮದಲ್ಲಿ ರಚಿಸಲಾಗುತ್ತದೆ). ಐಸ್ ಪ್ಯಾಕ್ ಅನ್ನು ತಕ್ಷಣವೇ ಅನ್ವಯಿಸಲಾಗುತ್ತದೆ. ನಂತರ ಲೇಸರ್ ಕಾರ್ಯವಿಧಾನದ 2 ನಿಮಿಷಗಳಲ್ಲಿ, ಟಾಪಿಕಲ್ ಟ್ರಯಾಮ್ಸಿನೋಲೋನ್ ಅಸಿಟೋನೈಡ್ ಜಲೀಯ ದ್ರಾವಣವನ್ನು (2.5mg/ml to10 mg/ml) ಅನ್ವಯಿಸಲಾಗುತ್ತದೆ.

ಭಾಗವಹಿಸುವವರು ಬೇಸ್ಲೈನ್ನಲ್ಲಿ ಮತ್ತು ನಂತರದ ಸಿಟ್ಟಿಂಗ್ಗಳಲ್ಲಿ ಸರಣಿ ಛಾಯಾಗ್ರಹಣಕ್ಕೆ ಒಳಗಾಗಬೇಕಾಗುತ್ತದೆ.

ದಯವಿಟ್ಟು ಕೆಳಗಿನ ಮಾಹಿತಿಯನ್ನು ಓದಿ ಮತ್ತು ನಿಮ್ಮ ಕುಟುಂಬದ ಸದಸ್ಯರೊಂದಿಗೆ ಚರ್ಚಿಸಿ. ಅಧ್ಯಯನಕ್ಕೆ ಸಂಬಂಧಿಸಿದಂತೆ ನೀವು ಯಾವುದೇ ಪ್ರಶ್ನೆಯನ್ನು ಕೇಳಬಹುದು. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ನಿಮ್ಮ ಮಗು/ವಾರ್ಡ್ಗೆ ನೀವು ಸಮ್ಮತಿಸಿದರೆ ನಾವು ನಿಮ್ಮಿಂದ ಮತ್ತು ನಿಮ್ಮ ಮಗುವಿನಿಂದ ಮಾಹಿತಿಯನ್ನು (ಪ್ರೊಫಾರ್ಮಾ ಪ್ರಕಾರ) ಸಂಗ್ರಹಿಸುತ್ತೇವೆ. ಅಗತ್ಯವಿದ್ದರೆ ಸಂಬಂಧಿತ ರಕ್ತ ಪರೀಕ್ಷೆಗಳನ್ನು ಕೈಗೊಳ್ಳಲಾಗುತ್ತದೆ. ಸಂಗ್ರಹಿಸಿದ ಈ ಮಾಹಿತಿಯನ್ನು ಪ್ರಬಂಧ ಮತ್ತು ಪ್ರಕಟಣೆಗೆ ಮಾತ್ರ ಬಳಸಲಾಗುತ್ತದೆ.

ನಿಮ್ಮ ಮಗು/ವಾರ್ಡ್ಗೆ ಸಂಬಂಧಿಸಿದಂತೆ ನಿಮ್ಮಿಂದ ಸಂಗ್ರಹಿಸಿದ ಎಲ್ಲಾ ಮಾಹಿತಿಯನ್ನು ಗೌಪ್ಯವಾಗಿ ಇರಿಸಲಾಗುತ್ತದೆ ಮತ್ತು ಯಾವುದೇ ಹೊರಗಿನವರಿಗೆ ಬಹಿರಂಗಪಡಿಸಲಾಗುವುದಿಲ್ಲ. ನಿಮ್ಮ ಮಗುವಿನ/ವಾರ್ಡ್ನ ಗುರುತನ್ನು ಬಹಿರಂಗಪಡಿಸಲಾಗುವುದಿಲ್ಲ. ತನಿಖೆಗಳಿಗೆ ಅಗತ್ಯವಿರುವ ವೆಚ್ಚಗಳನ್ನು ಅಧ್ಯಯನ ತನಿಖಾಧಿಕಾರಿಗಳು ಧನಸಹಾಯ ಮಾಡುತ್ತಾರೆ. ಈ ಅಧ್ಯಯನವನ್ನು ಸಾಂಸ್ಥಿಕ ನೀತಿಶಾಸ್ತ್ರ ಸಮಿತಿಯು ಪರಿಶೀಲಿಸಿದೆ ಮತ್ತು ನೀವು ಸಾಂಸ್ಥಿಕ ನೀತಿಶಾಸ್ತ್ರ ಸಮಿತಿಯ ಸದಸ್ಯರನ್ನು ಸಂಪರ್ಕಿಸಲು ಮುಕ್ತರಾಗಿದ್ದೀರಿ. ಈ ಅಧ್ಯಯನವನ್ನು ಒಪ್ಪಿಕೊಳ್ಳಲು ಯಾವುದೇ ಒತ್ತಾಯವಿಲ್ಲ. ನೀವು ಭಾಗವಹಿಸಲು ಬಯಸದಿದ್ದರೆ ನಿಮ್ಮ ಮಗು ಪಡೆಯುವ ಕಾಳಜಿಯು ಬದಲಾಗುವುದಿಲ್ಲ. ನಿಮ್ಮ ಮಗು ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ನೀವು ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ ಒಪ್ಪಿಕೊಂಡರೆ ಮಾತ್ರ ನೀವು ಹೆಬ್ಬೆರಳಿನ ಗುರುತನ್ನು ಸಹಿ ಮಾಡಬೇಕಾಗುತ್ತದೆ.

ಯಾವುದೇ ಹೆಚ್ಚಿನ ಸ್ಪಷ್ಟೀಕರಣಕ್ಕಾಗಿ ನೀವು ಅಧ್ಯಯನ ತನಿಖಾಧಿಕಾರಿಯನ್ನು ಸಂಪರ್ಕಿಸಬಹುದು:

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ANNEXURE – VIII

KEY TO MASTER CHART

1. GRP – Group
2. LAD SCORE - Lesional Area and Density Score
3. LAD % - Lesional Area and Density Score Improvement Percentage from Baseline Presentation
4. GPA - Global Photograph

   Assessment Scale

   Score – 0: < 25 %

   Improvement

   Score– 1: 25-50 %

   Improvement

   Score – 2: 50-75 %

   Improvement

   Score – 3: > 75 %

   Improvement

5. VDS - Visual Discomfort Scale of the patients following the procedure Scale ranges from 0 – 10
6. VAS - Visual Analogue Scale of the patient's satisfaction of the treatment modality at the end of 5 settings. Scale ranges from 0 – 10
7. ER – Erythema
8. ED – Edema
9. AP – Atrophy of skin
10. PIH – Post Inflammatory Hyperpigmentation

11. BL – Baseline presentation
12. 1 – At the end of 1st Setting
13. 2 – At the end of 2nd Setting
14. 3 – At the end of 3rd Setting
15. 4 – At the end of 4th Setting
16. 5 – At the end of 5th Setting

MASTER CHART

					LAD SCORE							LAD %					GPA					VDS	VAS	ADVERSE EFFECTS			
SLNO.	AGE	SEX	UHID	GRP	BL	1	2	3	4	5	PERCENTAGE	1	2	3	4	5	1	2	3	4	5			ER	ED	AP	PIH
1	20	F	291459	A	9.2	7.1	4.86	3.2	2.07	0.45	95.1%	22.8%	47.2%	65.2%	77.5%	95.1%	1	1	2	3	3	5	9	1	0	0	0
2	26	F	264670	B	15.6	11.5	8.1	7.2	5.2	3.6	76.9%	26.3%	48.1%	53.8%	66.7%	76.9%	1	1	2	2	3	4	7	1	1	0	0
3	31	M	95833	B	24.8	19.75	14.25	11.7	8.5	6	75.8%	20.4%	42.5%	52.8%	65.7%	75.8%	1	1	2	2	3	4	8	0	0	0	0
4	26	M	246327	A	8.6	7.3	5.6	3.3	1.7	0.5	94.2%	15.1%	34.9%	61.6%	80.2%	94.2%	0	1	2	3	3	3	7	0	0	0	0
5	35	F	127785	B	4.2	2.8	2.04	1.68	1.28	0.96	77.1%	33.3%	51.4%	60.0%	69.5%	77.1%	1	2	2	3	3	6	9	1	0	1	0
6	14	F	79989	B	25.4	18.2	12.5	10	7.7	6.5	74.4%	28.3%	50.8%	60.6%	69.7%	74.4%	1	2	2	2	2	4	9	0	0	0	0
7	31	F	293177	A	16.3	12.6	8.8	5.6	2.7	0.9	94.5%	22.7%	46.0%	65.6%	83.4%	94.5%	1	1	2	2	3	3	7	0	1	0	0
8	24	M	293412	A	8.7	7.2	5.04	2.9	1.3	0.3	96.6%	17.2%	42.1%	66.7%	85.1%	96.6%	0	1	2	3	3	3	7	1	0	0	0
9	33	M	207448	B	15.9	11.2	8.6	7.2	5.6	4	74.8%	29.6%	45.9%	54.7%	64.8%	74.8%	1	1	2	2	2	3	7	0	1	0	0
10	23	M	292206	B	36.4	26.2	19.8	16.5	11.5	7.9	78.3%	28.0%	45.6%	54.7%	68.4%	78.3%	1	1	2	2	3	5	7	1	0	1	0
11	40	M	293867	B	4.2	2.9	2.04	1.72	1.3	1.04	75.2%	31.0%	51.4%	59.0%	69.0%	75.2%	1	2	2	2	3	5	7	0	1	0	1
12	25	M	295083	A	9.3	7.1	4.7	3.1	1.8	0.6	93.5%	23.7%	49.5%	66.7%	80.6%	93.5%	1	1	2	3	3	3	8	0	1	0	0
13	22	M	295619	B	16.1	11.3	8.4	6.8	4.8	3.8	76.4%	29.8%	47.8%	57.8%	70.2%	76.4%	1	1	2	2	3	4	9	0	0	0	0
14	38	M	182275	A	4.1	3.2	2.04	1.2	0.68	0.2	95.1%	22.0%	50.2%	70.7%	83.4%	95.1%	1	2	3	3	3	3	9	1	0	0	0
15	35	F	271664	A	15.9	12.3	8.3	5.4	3.04	0.4	97.5%	22.6%	47.8%	66.0%	80.9%	97.5%	1	1	2	3	3	4	9	0	0	0	0
16	13	F	266279	B	24.7	17.7	13.2	10.7	7.7	6.5	73.7%	28.3%	46.6%	56.7%	68.8%	73.7%	1	1	2	2	2	4	7	0	0	0	0
17	22	M	229781	A	9.2	6.8	5.1	2.8	1.5	0.27	97.1%	26.1%	44.6%	69.6%	83.7%	97.1%	1	1	2	3	3	6	8	1	0	0	0
18	14	M	197129	B	16.1	11.04	8.4	7.2	5.6	3.36	79.1%	31.4%	47.8%	55.3%	65.2%	79.1%	1	1	2	2	3	5	8	0	0	1	0
19	35	F	260214	A	24.2	19.7	13.5	9	5.7	1.7	93.0%	18.6%	44.2%	62.8%	76.4%	93.0%	0	1	2	3	3	4	7	1	0	0	0
20	23	M	59220	A	8.9	7.29	4.6	3.3	1.5	0.5	94.4%	18.1%	48.3%	62.9%	83.1%	94.4%	0	1	2	3	3	5	8	0	0	0	0
21	24	M	290110	A	9.4	7.02	4.86	3.2	1.7	0.2	97.9%	25.3%	48.3%	66.0%	81.9%	97.9%	1	1	2	3	3	4	8	0	0	0	0
22	31	M	298119	B	15.9	11.5	8.1	6.5	4.9	4	74.8%	27.7%	49.1%	59.1%	69.2%	74.8%	1	2	2	2	2	5	9	1	1	0	0
23	23	M	288784	B	4.2	2.9	2	1.6	1.2	0.9	78.6%	31.0%	52.4%	61.9%	71.4%	78.6%	1	2	2	2	3	4	7	0	0	0	0
24	42	F	298735	A	15.9	13.2	8.6	5.4	2.4	0.8	95.0%	17.0%	45.9%	66.0%	84.9%	95.0%	0	1	2	3	3	3	9	1	0	0	0
25	20	F	298821	A	9.3	6.9	4.9	2.9	1.7	0.3	96.8%	25.8%	47.3%	68.8%	81.7%	96.8%	1	1	2	3	3	5	6	0	0	0	0
26	28	M	233635	A	25.1	19	13.2	8.7	3.7	1.7	93.2%	24.3%	47.4%	65.3%	85.3%	93.2%	1	1	2	3	3	5	8	0	0	0	0
27	16	M	298997	B	9.3	6.3	4.9	4.05	2.9	2.34	74.8%	32.3%	47.3%	56.5%	68.8%	74.8%	1	1	2	2	2	4	7	0	1	1	0
28	42	F	299631	A	36	28.4	19.8	11.1	5.7	2.5	93.1%	21.1%	45.0%	69.2%	84.2%	93.1%	1	1	2	3	3	4	9	1	1	0	0
29	25	M	299532	B	16.2	11.5	8	6.8	5.7	4.1	74.7%	29.0%	50.6%	58.0%	64.8%	74.7%	1	2	2	2	2	3	7	0	1	0	1
30	30	M	298325	A	9.1	7.1	4.8	3.2	2.07	0.2	97.8%	22.0%	47.3%	64.8%	77.3%	97.8%	1	1	2	3	3	5	7	1	0	0	0
31	23	F	300318	A	24.7	20.5	15.7	9.2	4.7	1.5	93.9%	17.0%	36.4%	62.8%	81.0%	93.9%	0	1	2	3	3	6	9	0	0	0	0
32	25	F	300961	B	16.2	11.5	8.1	7.2	5.2	3.6	77.8%	29.0%	50.0%	55.6%	67.9%	77.8%	1	2	2	2	3	4	9	0	0	0	0
33	41	M	301194	B	25.2	19.75	14.25	11.7	8.5	6	76.2%	21.6%	43.5%	53.6%	66.3%	76.2%	1	1	2	2	3	3	7	0	0	1	0
34	45	M	301958	A	8.7	7.1	4.86	3.2	2.07	0.45	94.8%	18.4%	44.1%	63.2%	76.2%	94.8%	0	1	2	3	3	4	8	1	0	0	0
35	26	F	302112	B	4.2	2.8	2.04	1.68	1.28	0.96	77.1%	33.3%	51.4%	60.0%	69.5%	77.1%	1	2	2	2	3	5	8	0	1	0	0
36	24	F	277831	B	24.9	18.2	12.5	10	7.7	6.5	73.9%	26.9%	49.8%	59.8%	69.1%	73.9%	1	2	2	2	2	3	7	0	0	0	0
37	36	M	302396	A	9.2	7.3	5.6	3.3	1.7	0.5	94.6%	20.7%	39.1%	64.1%	81.5%	94.6%	1	1	2	3	3	4	7	1	0	0	0
38	30	M	64091	B	25.2	18.2	12.5	10	7.7	6.5	74.2%	27.8%	50.4%	60.3%	69.4%	74.2%	1	2	2	2	2	5	8	1	0	0	0
39	32	M	248212	A	9.2	7.1	4.7	3.1	1.8	0.6	93.5%	22.8%	48.9%	66.3%	80.4%	93.5%	1	1	2	3	3	4	7	0	1	0	0
40	22	F	276265	B	35.8	26.2	19.8	16.5	11.5	7.9	77.9%	26.8%	44.7%	53.9%	67.9%	77.9%	1	1	2	1	3	6	6	0	0	0	0
41	42	F	290744	A	4.2	3.2	2.04	1.2	0.68	0.2	95.2%	23.8%	51.4%	71.4%	83.8%	95.2%	1	2	3	3	3	5	7	0	0	0	0
42	30	M	303873	B	25.2	17.7	13.2	10.7	7.7	6.5	74.2%	29.8%	47.6%	57.5%	69.4%	74.2%	1	1	2	2	2	4	6	1	0	0	0
43	40	M	304338	A	9.3	6.8	5.1	2.8	1.5	0.27	97.1%	26.9%	45.2%	69.9%	83.9%	97.1%	1	1	2	3	3	5	6	0	0	0	0
44	14	M	286249	A	8.9	7.02	4.86	3.2	1.7	0.2	97.8%	21.1%	45.4%	64.0%	80.9%	97.8%	1	1	2	3	3	4	7	0	1	0	0

					LAD SCORE							LAD %					GPA					VDS	VAS	ADVERSE EFFECTS			
SLNO.	AGE	SEX	UHID	GRP	BL	1	2	3	4	5	PERCENTAGE	1	2	3	4	5	1	2	3	4	5			ER	ED	AP	PIH
45	29	F	249638	A	16.2	13.2	8.6	5.4	2.4	0.8	95.1%	18.5%	46.9%	66.7%	85.2%	95.1%	0	1	2	3	3	5	8	0	0	0	0
46	25	F	305408	A	9.2	6.9	4.9	2.9	1.7	0.3	96.7%	25.0%	46.7%	68.5%	81.5%	96.7%	1	1	2	3	3	6	7	1	0	0	0
47	24	M	306816	B	15.9	11.5	8	6.8	5.7	4.1	74.2%	27.7%	49.7%	57.2%	64.2%	74.2%	1	2	2	2	2	4	9	1	0	1	1
48	44	F	308523	B	16.3	11.5	8.1	7.2	5.2	3.6	77.9%	29.4%	50.3%	55.8%	68.1%	77.9%	1	2	2	2	3	5	8	0	0	0	0
49	28	M	90145	A	36.2	28.4	19.8	11.1	5.7	2.5	93.1%	21.5%	45.3%	69.3%	84.3%	93.1%	1	1	3	3	3	5	9	0	0	0	0
50	29	M	277167	B	24.1	18.2	12.5	10	7.7	6.5	73.0%	24.5%	48.1%	58.5%	68.0%	73.0%	1	1	2	2	2	3	8	0	1	0	0
51	23	M	309785	A	4	3.2	2.04	1.2	0.68	0.2	95.0%	20.0%	49.0%	70.0%	83.0%	95.0%	1	1	3	3	3	2	7	1	0	0	1
52	23	F	311390	B	16.3	11.5	8.1	6.5	4.9	4	75.5%	29.4%	50.3%	60.1%	69.9%	75.5%	1	2	2	2	2	5	7	1	0	0	0
53	51	F	297429	B	24.9	17.7	13.2	10.7	7.7	6.5	73.9%	28.9%	47.0%	57.0%	69.1%	73.9%	1	1	2	2	2	2	9	0	0	0	0
54	49	F	312527	A	8.9	6.9	4.9	2.9	1.7	0.3	96.6%	22.5%	44.9%	67.4%	80.9%	96.6%	1	1	3	3	3	3	9	0	0	0	0
55	23	M	312685	A	4.2	3.2	2.04	1.2	0.68	0.2	95.2%	23.8%	51.4%	71.4%	83.8%	95.2%	1	2	3	3	3	3	7	0	0	0	0
56	24	F	313332	B	16.1	11.5	8.1	6.5	4.9	4	75.2%	28.6%	49.7%	59.6%	69.6%	75.2%	1	1	2	2	2	3	7	0	0	0	0
57	29	M	314282	B	9.2	6.3	4.9	4.05	2.9	2.34	74.6%	31.5%	46.7%	56.0%	68.5%	74.6%	1	1	2	2	2	4	8	0	0	0	0
58	21	M	274552	B	25.3	18.2	12.5	10	7.7	6.5	74.3%	28.1%	50.6%	60.5%	69.6%	74.3%	1	2	2	2	2	4	7	0	1	0	0
59	40	F	311452	B	36.1	26.2	19.8	16.5	11.5	7.9	78.1%	27.4%	45.2%	54.3%	68.1%	78.1%	1	1	2	2	3	3	8	0	0	0	0
60	23	M	315792	A	9.2	7.02	4.86	3.2	1.7	0.2	97.8%	23.7%	47.2%	65.2%	81.5%	97.8%	1	1	2	3	3	5	9	0	0	0	0
61	38	M	315774	B	25.3	17.7	13.2	10.7	7.7	6.5	74.3%	30.0%	47.8%	57.7%	69.6%	74.3%	1	1	2	2	2	3	7	0	0	0	0
62	24	M	319147	A	9.1	7.1	4.86	3.2	2.07	0.45	95.1%	22.0%	46.6%	64.8%	77.3%	95.1%	1	1	2	3	3	5	6	0	0	0	0
63	23	M	152714	A	9.4	6.8	5.2	2.8	1.5	0.27	97.1%	27.7%	44.7%	70.2%	84.0%	97.1%	1	1	2	3	3	5	7	0	0	0	0
64	29	M	156822	A	16.5	13.2	8.6	5.4	2.4	0.8	95.2%	20.0%	47.9%	67.3%	85.5%	95.2%	0	1	2	3	3	5	7	0	0	0	1
65	17	M	68192	B	16.2	11.4	8.2	7.2	5.2	3.6	77.8%	29.6%	49.4%	55.6%	67.9%	77.8%	1	2	2	2	3	5	9	0	0	0	1
66	45	F	148491	A	8.9	7	4.8	3.2	1.7	0.2	97.8%	21.3%	46.1%	64.0%	80.9%	97.8%	1	1	2	3	3	4	8	0	0	0	0
67	23	F	124115	B	25.3	17.9	13.2	10.7	7.7	6.5	74.3%	29.2%	47.8%	57.7%	69.6%	74.3%	1	1	2	2	2	4	8	0	0	0	0
68	38	M	45830	A	4.4	3.4	2.04	1.3	0.68	0.2	95.5%	22.7%	53.6%	70.5%	84.5%	95.5%	1	2	3	3	3	5	7	0	0	0	0
69	17	M	94222	B	24.7	18	12.3	9.9	7.7	6.5	73.7%	27.1%	50.2%	59.9%	68.8%	73.7%	1	2	2	2	2	3	8	0	0	0	1
70	30	F	131492	A	9.3	7.1	4.7	3.1	1.8	0.6	93.5%	23.7%	49.5%	66.7%	80.6%	93.5%	1	1	2	3	3	3	8	1	0	0	0
71	23	M	152714	A	25.6	19.3	13.4	8.9	3.9	1.9	92.6%	24.6%	47.7%	65.2%	84.8%	92.6%	1	1	2	3	3	5	9	0	0	0	1
72	42	M	138954	B	16.6	11.7	8.3	6.7	5.2	4.3	74.1%	29.5%	50.0%	59.6%	68.7%	74.1%	1	2	2	2	2	5	6	0	0	0	0
73	39	M	151288	B	24.4	18.4	12.7	10.2	7.9	6.5	73.4%	24.6%	48.0%	58.2%	67.6%	73.4%	1	1	2	2	2	3	7	0	0	0	1
74	39	M	177812	B	36.5	26.5	20.1	16.7	11.7	7.9	78.4%	27.4%	44.9%	54.2%	67.9%	78.4%	1	1	2	2	3	3	7	1	0	0	0