COMPARISON OF ORAL MINOXIDIL WITH ORAL DUTASTERIDE IN THE TREATMENT OF MALE ANDROGENETIC ALOPECIA – A RANDOMIZED CONTROL TRIAL

BY

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DISSERTATION SUBMITTED TO

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DOCTOR OF MEDICINE(M.D.)

IN

DERMATOLOGY, VENEREOLOGY AND LEPROSY

UNDER THE GUIDANCE OF

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ABSTRACT

INTRODUCTION:

Androgentic alopecia is a gentically predetrmined disorder due to increased response to androgens affecting 50% males. MPHL is common hair loss disorder. The MPHL is a non-scarring progressive thinning of hair which causes psychological distress affecting quality of life of the patient. Though various modalities are available, assessment,efficacy & treatment must be considered.

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COMPARISON OF ORAL MINOXIDIL WITH ORAL DUTASTERIDE IN THE TREATMENT OF MALE ANDROGENETIC ALOPECIA- A RANDOMIZED CONTROL TRIAL. ABSTRACT INTRODUCTION: Androgentic alopecia is a gentically predetrmined disorder due to increased response to androgens affecting 50% males. MPHL is common hair loss disorder. The MPHL is a non-scarring progressive thinning of hair which causes psychological distress affecting quality of life of the patient. Though various modalities are available, assessment,efficacy & treatment must be considered. AIMS AND OBJECTIVES: 1. To compare the efficacy of low dose oral minoxidil with that of low dose oral dutasteride. 2. To document the adverse effects associated with that of oral minoxidil and oral After obtaining informed consent,64 male patients of age 18-30 years diagnosed to have androgenetic alopecia from October 2022- March 2024 will be taken for the

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DATE: Signature of the candidate

PLACE: KOLAR **DR.G.ANJANA**

LIST OF ABBREVIATIONS

ABBREVIATIONS	FULL FORMS
AGA	Androgenetic Alopecia
MPHL	Male Pattern Hair Loss
MAA	Male Androgenetic Alopecia
GPA	Global Photographic Assessment
FPHL	Female Pattern Hair Loss
QOL	Quality Of Life
FDA	Food and Drug Administration
US	United States
ARs	Androgen Receptors
DHT	Dihydro Testosterone
DP	Dermal Papilla
EDA2R	Ectodysplasia A2 Receptor
DHEA	Dehydroepiandrosterone
HSD	Hydroxysteroid Dehydrogenase
SCF	Stem Cell Factor
FT	Fronto Temporal

FU	Follicular Unit
DUPA	Diffuse Unpatterned Alopecia
ORS	Outer Root Sheath
VEGF	Vascular Endothelial Growth Factor
PDGF	Platelet Derived Growth Factor
ARI	Alpha Reductase Inhibitors
LLLT	Low Level Laser Therapy
nm	nanometer
mW	milliwatt
BPH	Benign Prostatic Hyperplasia
AMP	Adenosine Mono Phosphate
PRP	Platelet Rich Plasma
PDGF	Platelet Derived Growth Factor
GFC	Growth Factor Concentrate
LDOM	Low Dose Oral Minoxidil

ABSTRACT

INTRODUCTION:

Androgenetic alopecia is a genetically predetermined disorder due to excessive response to androgens which affects up to 50% of males. MPHL is the most common hair loss disorder in men. The MPHL is a non-scarring progressive thinning of hair which causes psychological distress affecting quality of life of the patient. Although MAA is often regarded as a relatively minor dermatological condition, hair loss impacts self-image and is a great cause of anxiety and depression in some men. Though various modalities are available for the treatment of androgenetic alopecia, assessment of efficacy of treatment must be considered.

AIMS AND OBJECTIVES:

1. To compare the efficacy of low dose oral minoxidil with that of low dose oral dutasteride.
2. To document the adverse effects associated with that of oral minoxidil and oral dutasteride.

MATERIALS AND METHODS:

After obtaining informed consent,64 male patients of age 18-30 years diagnosed to have androgenetic alopecia attending the Department of Dermatology, R L Jalappa Hospital and Research centre from March 2024 – August 2024 will be taken for the hospital based Comparative Interventional Study. Among 64 male patients with androgenetic alopecia, 32 patients are treated with oral minoxidil 5mg once daily and 32 patients are treated with oral dutasteride 0.5 mg thrice a week for a period of 12 months.

RESULTS:

Efficacy of treatment modality-assessed using means score of GPA - Scale suggests significant improvement in both the groups initially but drastic improvement and patient satisfaction was observed in Group B with subsequent sittings compared to Group A. Patients in Group B had lesser side effects than Group A.

CONCLUSION:

This study showed that oral Dutasteride 0.5 mg thrice weekly is a better treatment modality than oral Minoxidil 5mg daily for the treatment of Male Androgenetic alopecia with respect to efficacy, safety and adverse effects.

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INTRODUCTION

The most prevalent type of alopecia in the world, androgenetic alopecia (AGA), referred to as male pattern hair loss (MPHL) or female pattern hair loss (FPHL), is influenced by genetics and sensitivity to androgens.[1]

AGA manifests in a distinctive distribution that is specific to both genders. This disorder is characterized by a progressive loss of scalp terminal hair that usually happens after puberty and has a unique pattern in both males and females.[2]

While females tend to maintain their frontal hairline while having diffuse apical hair thinning, which gives the anterior area of the hair a larger appearance, males often exhibit hair loss most prominently at the vertex and frontotemporal regions.[4] Even with its widespread occurrence, AGA can be difficult to treat.

The quality of life may be impacted by AGA's effects on appearance and psychosocial experiences (QOL). According to numerous studies, men who lose their hair too soon frequently display emotional discomfort and express major concern to their peers and family.[8,9,10]

The chronic nature of the illness is caused by a combination of environmental and hereditary factors.[6,7] Topical minoxidil and oral finasteride are the only two medications for the illness that have received FDA approval in the US.[5] Nonetheless, a number of studies have demonstrated the efficacy of non-FDA-approved therapies in the management of AGA.

Low-dose oral minoxidil and low-dose oral dutasteride have drawn increased attention recently for the treatment of AGA, despite not being FDA

authorized.[3] Hence, this study was proposed to assess and compare the efficacy and safety of low dose oral Minoxidil and low dose oral Dutasteride in the treatment of Male Androgenetic Alopecia.

AIMS & OBJECTIVES

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REVIEW OF LITERATURE

REVIEW OF LITERATURE

SYNONYMS:

- Pattern alopecia
- Male or Female pattern baldness.

HISTORY

Alopecia is the medical term for mange fur loss in foxes, and it is derived from the Greek word alopex, which means "fox." One of the earliest depictions of hair loss, dating back to 30,000 years, can be seen on the walls of prehistoric caves. These engravings even resemble the patterns of hair loss that occur today.[14]

Balding has been documented throughout history, and Aristotle noted that ladies and eunuchs did not have it. According to Darwin, being bald is a secondary sexual trait. He reasoned that although most adult individuals have hair, men tend to lose their hair more frequently than women.[12]

Osborn found that there is a dominating genetic pattern associated with baldness. Hamilton and Orentreich associated baldness with age, androgens, and genetic propensity.[13]

Male and female AGA progress according to distinct classification scales. There are differences in the patterns of hair loss and more evidence points to androgens in males, even if the etiology may be same in both genders.[11]

The biggest advancements in hair loss medication came with the introduction of topical minoxidil in the 1980s and oral finasteride in the 1990s.

EPIDEMIOLOGY

By the time they turned 50, 30% to 50% of males, according to Hamilton, got MAA. The incidence and pattern of hair loss in MAA vary depending on race and age, according to numerous research conducted in the West.[15]

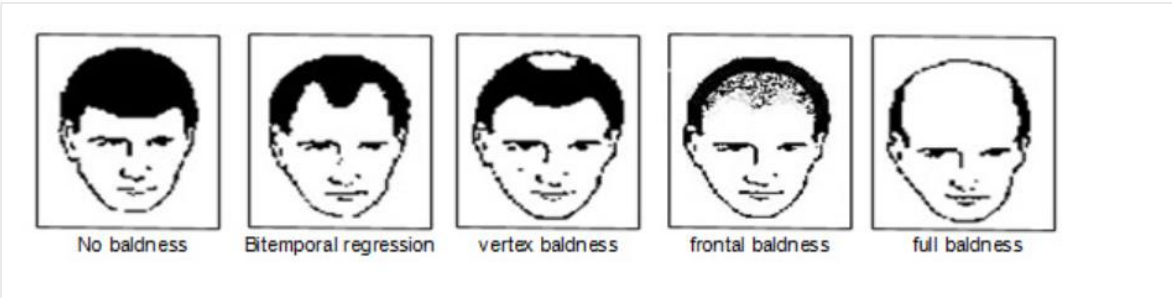
Caucasian males are reported to have a higher incidence and severity of MAA than men of other ethnicities. It has been shown that Caucasian populations experience advanced degrees of alopecia more frequently and at an earlier age than Mongolian populations.[16]

Compared to Caucasians, Japanese people experience the onset of MAA ten years later. Compared to Caucasians, Black, Oriental, Native American, and African-American men are more likely to have less extensive and late-onset baldness as well as preservation of their frontal hair lines. The findings of a population survey conducted in Singapore corroborate the notion that Chinese men experience a reduced incidence of MAA.[17,18]

MAA's age prevalence has been shown in a number of research populations. To ascertain the frequency and risk factors for MAA, 1390 men in Australia between the ages of 40 and 69 were studied.[19,22] The Norwood Hamilton scale indicates that the prevalence of vertex or complete baldness rises with age, from 31% (age 40–55) to 53% (age 65–69). A receding frontal hairline was observed in 31% of men 65-69 years old and 25% of men 40-55 years old.

According to a US poll, 53% of people in the 40–49 age range had moderate or severe MAA. In the Korean population, there has also been evidence of an increased incidence of MAA with aging; type III-vertex involvement is most frequently observed in the third to seventh decades.[20,23] According to reports,

the prevalence of MAA in male Singaporeans was 63%; it increased with age, from 32% in those aged 17 to 26 to 100% in those aged 80 and beyond.[21]



Patterns of androgenetic alopecia in men.

PSYCHOSOCIAL IMPACT OF MAA

A person's hair has a crucial role in their self-perception, mostly because it facilitates socializing. Consequently, the effects of MAA are primarily psychological. Numerous research indicate that balding individuals' negative self-perceptions seem to be shared by Asian and Western cultures.[24] Those who are not impacted by MAA frequently minimize or disregard its detrimental effects. Nonetheless, there is proof that balding males may experience psychological issues that are exacerbated by how other people see them.[25]

According to a Korean study, the negative perceptions that women and non-balding males had of men with MAA were consistent with the psychosocial impacts that the patients themselves had mentioned.[26] Notably, almost 90% of participants in the survey believed that males with beards were less attractive.

Compared to non-balding men, women held this opinion more frequently. Balding men's ability to operate socially may be further hampered by these unfavourable opinions. According to a recent study, individuals with

androgenetic alopecia frequently experience anxiety and sadness and resort to avoidant coping mechanisms.[27] Nonetheless, it's crucial to remember that the majority of impacted men manage androgenetic alopecia well, with little to no negative effects on their psychosocial functioning.

As a result, individuals who do seek assistance are probably experiencing more emotional discomfort and are not happy with the care they have gotten. males who have more significant hair loss, extremely early onset, and a belief that their balding is progressive are the most distressed balding males.[28] The patient's emotional reactions to alopecia, such as their feelings of anger, anxiety, and sadness, as well as their perceptions of the condition's consequences, must be addressed by the doctor.

ETIOPATHOGENESIS:

AGA is a genetically mediated illness that is androgen-dependent (also known as androgen-heredity; genetic-heredity). The dermal papilla (DP) capillaries allow the circulating androgens, primarily dihydrotestosterone (DHT), to enter the hair follicle.[29] DHT upregulates the genes that cause terminal hair follicles to gradually shrink into shrunken hair follicles by binding to androgen receptors (ARs) in hair follicles and DP cells.

GENETICS:

It is commonly known that there is racial heterogeneity in the prevalence of baldness as well as a familial susceptibility to MAA. According to twin studies, heredity is responsible for about 80% of the baldness tendency. The extent to which hair follicles react to circulating androgens is altered by genetic variables. A modest propensity may delay baldness until the 60s or 70s, while a strong predisposition causes baldness to appear in adolescence.[32]

By the time they become 70, less than 15% of men have little to no baldness. In 1916, Osborne proposed that the baldness gene acted as an autosomal recessive gene in women and an autosomal dominant gene in men.[31]

The X-chromosome's AR and ectodysplasin A2 receptor (EDA2R) loci are the two main susceptibility loci for MPHL.[35] In their study of Indian patients, Sehgal et al. discovered that 87% of patients had a positive family history of AGA, with 67% of patients showing paternal inheritance, 5% showing maternal inheritance, and 13% showing both.[30]

The metabolism of testosterone to dihydrotestosterone (DHT) and the action of 5 alpha reductase inhibitors in the treatment of hair loss demonstrate the involvement of the 5 alpha reductase enzyme in MAA. Androgenetic alopecia has also been linked to the cytochrome p450 alpha aromatase enzyme.[33,34]By promoting the conversion of testosterone to estrogen, aromatase reduces intra-follicular testosterone levels. The expression of aromatase varies between balding and non-balding scalps. Yip et al. speculate that women may be predisposed to hair loss due to the aromatase gene (CYP19A1).[34]

HORMONES AND ANDROGENETIC ALOPECIA:

It is commonly known that androgen plays a part in male pattern hair loss. Male castration prevented the development of MAA unless testosterone was added, according to American anatomist James Hamilton.[36]

Testosterone, dehydroepiandrosterone sulphate (DHEA), free testosterone, and serum androgen levels have not consistently shown a difference between case and control subjects.[37,40]

Cortisol and androstenedione levels were higher in MAA patients than in age-matched controls in a study that examined the hormonal differences

between the two groups. This research also implies that a wide variety of hormones could impact androgenetic alopecia. Despite the fact that hirsutism and scalp hair loss are crucial indicators of hyperandrogenism in women, multiple studies were unable to show elevated testosterone levels in females.[38,39] Thus, it's possible that among genetically predisposed people, normal androgen levels are enough to induce hair loss.Balding scalps have been shown to have higher DHT levels than non-balding scalps.[49]

Local variables such as a higher number of androgen receptors, functional polymorphisms of the androgen receptor, increased local DHT synthesis, and decreased local DHT degradation can also contribute to intrafollicular androgen over-activity.

The skin and all of its appendages—hair follicles, sebaceous glands, and eccrine/apocrine glands—are provided with all the enzymes needed for androgen synthesis and metabolism, just like the classical steroidogenic organs like gonads and adrenal glands. Through the intrafollicular conversion of testosterone to the more active metabolite DHT, the enzyme 5 alpha reductase plays a crucial role. Five times as avidly as testosterone, DHT binds to the androgen receptor and has a greater potency to activate downstream processes.[48] Two isoenzymes of alpha reductase, identified by their distinct tissue expression patterns and pH optima, have been characterized.

Immunohistochemical analysis reveals the presence of type 1 5 alpha reductase in hair follicles, eccrine sweat glands, apocrine sweat glands, and sebaceous glands. Sebaceous glands in the face and scalp have far higher activity of type 1 5 alpha reductase than sebaceous glands in parts of the skin that are not prone to acne.[44] According to northern blot studies, type 1 mRNA is abundant in newborn foreskin keratinocytes, which are

subsequently followed by adult face sebocytes.[43,46] The expression of type 1 mRNA is stronger in dermal papilla (DP) from occipital hair cells than it is in beard cells. Additionally, the kidneys, liver, and adrenal glands contain it.

Although type 1 enzyme exhibits a broad expression pattern, its physiological role remains unclear. The dermal papilla, the inner layer of the outer root sheath, the sebaceous ducts, and the proximal inner root sheath of scalp hair follicles have all been identified by immunohistochemistry as having type 2 enzymes.[41,42,47] Additionally, the liver, testes, and prostate contain it. About 80% of the DHT in circulation is produced by type 2 5 alpha reductase.

Steroid sulfatase, 3-beta-HSD1, 17-beta-HSD3, and type 1 5 alpha reductase are the main steroidogenic enzymes responsible for the formation of potent androgens. Recent studies by Hoffmann et al. show that a number of other enzymes involved in the pathogenesis of androgenetic hair loss. 17-beta- and 3-beta-hydroxysteroid dehydrogenases (HSD), along with type 2 5 alpha reductase within the dermal papilla, play a central role in the intrafollicular conversion of testosterone to DHT.[45]

It appears that human hair follicles, which are dispersed throughout the body, are genetically predisposed to androgen-dependent development that begins during puberty. Androgens paradoxically affect human hair follicles differently depending on the bodily regions. In genetically predisposed individuals, androgens inhibit the growth of frontal scalp hair and promote the growth of hair in certain locations, such as the pubic, axillary, and beard regions.[50]

Scalp hair loss is a result of variables inherent to individual hair follicles and progresses in an ordered and repeatable fashion. Hair follicles can self-

regulate their response to androgens by controlling the expression of 5 alpha reductase and androgen receptors, according to in vitro tests.[52]

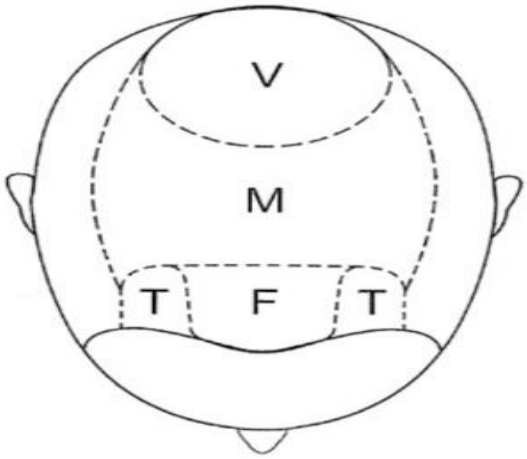
The measurable variation in androgen receptor counts and 5 alpha reductase activity between balding and non-balding scalp regions is thought to result from this self-regulation.[51,53] The easiest way to illustrate this intrinsic regulation is through hair transplantation experiments: scalp hairs from the vertex transplanted to the forearm miniaturize at the same rate as hairs around the donor location, whereas occipital hairs transplanted to the vertex retain their resistance to MAA.

PATHOPHYSIOLOGY:

In androgenetic alopecia, large terminal follicles shed and are replaced by little vellus hairs. The vertex scalp, mid frontal scalp, and temples are the three sections of the scalp that are most commonly afflicted. The procedure is rigorously patterned in these areas. Bitemporal hair loss spreads posteriorly across the head, beginning at the anterior hairline. Around the vertex scalp, hair loss starts in the center and spreads circumferentially. Hair follicle shrinkage over the mid frontal scalp causes a pattern of hair loss that resembles a Christmas tree. Due to unequal effects on these three areas, there are clinical differences in the pattern of hair loss in males; some bald more in the front, others more over the crown**.**

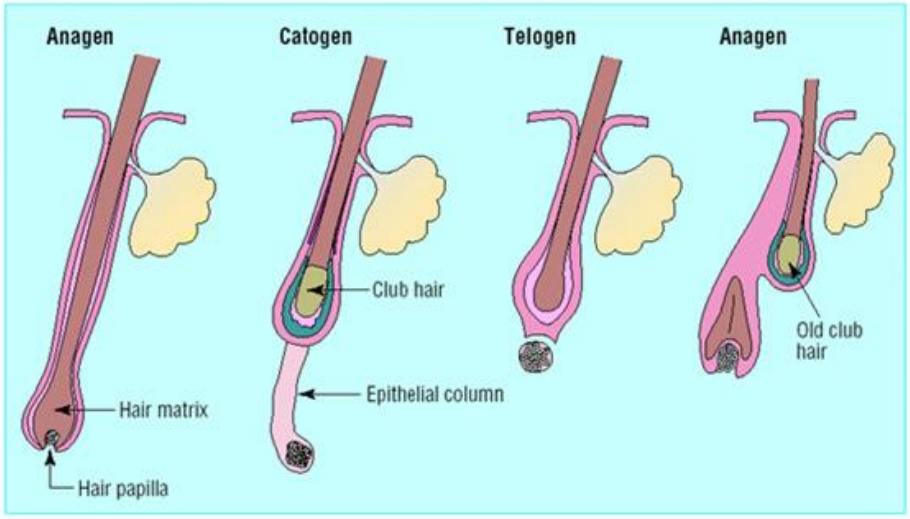
Areas of the scalp. F-Frontal / M-Mid frontal / T-Temple / V-Vertex.

Follicular shrinkage, inflammation, and altered hair cycle dynamics are the three main characteristics of MAA.



HAIR CYCLE DYNAMICS AND ANDROGENETIC ALOPECIA:

Follicles go through corresponding cyclic phases of growth, involution, quiescence, and regeneration. The growth phase, known as anagen, lasts for three to five years. Since hair elongation is relatively constant at one centimeter per month, the length of the growth phase determines the final hair length. The involutional phase, or catagen, ends after anagen and lasts for a few weeks. The subsequent period of hair follicle quiescence, or telogen, lasts for about three months. Hair follicle regeneration happens during the first week of anagen and continues until the hair reaches its final (possibly predetermined) length.



Normal hair cycle

Normal hair cycle - Each telogen hair is replaced by a new anagen hair. In androgenetic alopecia, the duration of anagen diminishes with each cycle, while the length of telogen remains constant or is prolonged; this leads to the reduction of the anagen to telogen ratio. Patients who are balding frequently talk about episodes of excessive hair loss, which is especially apparent when brushing or washing. This is brought on by the telogen's proportionate increase in follicle count. As the hair growth rate remains generally constant, the period of anagen growth influences hair length.

As a result, each hair shaft gets shorter with each succeeding hair cycle that is foreshortened. The anagen phase ultimately ends in such a way that the growing hair is unable to grow long enough to reach the skin's surface, resulting in an empty follicular pore. In MAA, there appears to be a longer duration of the lag phase, also known as the prolonged replacement of telogen hair, which results in a higher proportion of empty hair follicles that contribute to balding. Moreover, MAA prolongs the kenogen (latent phase), which lowers hair count and speeds up the balding process.

Androgens appear to lower alopecia hair color by decreasing dermal papilla stem cell factor (SCF) production, which is critical in embryonic melanocyte migration and bulbar melanocyte pigmentation. In MAA, tiny, pale hairs eventually replace large, pigmented ones.

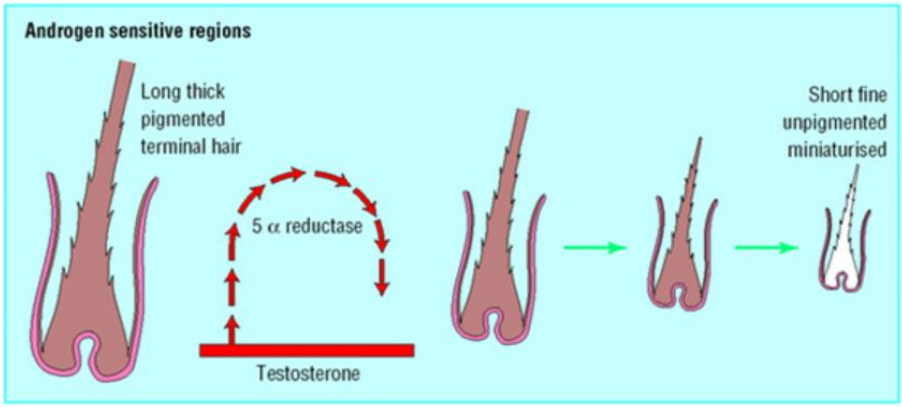
HAIR FOLLICLE MINIATURISATION:

The histopathological characteristic of androgenetic alopecia is shrinking of hair follicles. The mesenchymal and ectodermal components make up hair follicles. Invagination of the epidermis into the dermis and subcutaneous fat make up the ectodermal component. The hair shaft is produced by the hair matrix, which is found in the hair bulb. The dermal

papilla, a little group of specialized fibroblasts completely encircled by the hair bulb, represents the mesenchymal component.[54]

All of the follicular machinery is gradually and stepwise miniaturized in MAA, which is associated with changes in the dynamics of the hair cycle. The dermal papilla formed from mesenchyme is situated at the base of the hair bulb and controls various features of the epithelial follicle, including the type of hair that is produced.

The androgen-mediated processes that cause miniaturization and modifications to the hair cycle are likely to target the dermal papilla since it plays a crucial role in the regulation of hair growth. According to the consistent geometric link between the size of the dermal papilla and the hair matrix, the size of the dermal papilla dictates the size of the hair bulb and, eventually, the length of the hair shaft that is formed.[55]



Stepwise miniaturisation of hair follicles and shortening of anagen growth phase mediated by Dihydrotestosterone.

The decrease in hair follicular size is most likely due to a reduction of more than ten times in total cell counts. The exact mechanism causing this decrease is unknown, but possibilities include apoptotic cell death, decreased keratinocyte proliferation, cell displacement due to loss of cellular adhesion,

which causes dermal papilla fibroblasts to drop off into the dermis, or migration of dermal papilla cells into the dermal sheath connected to the outer root sheath of the hair follicle.

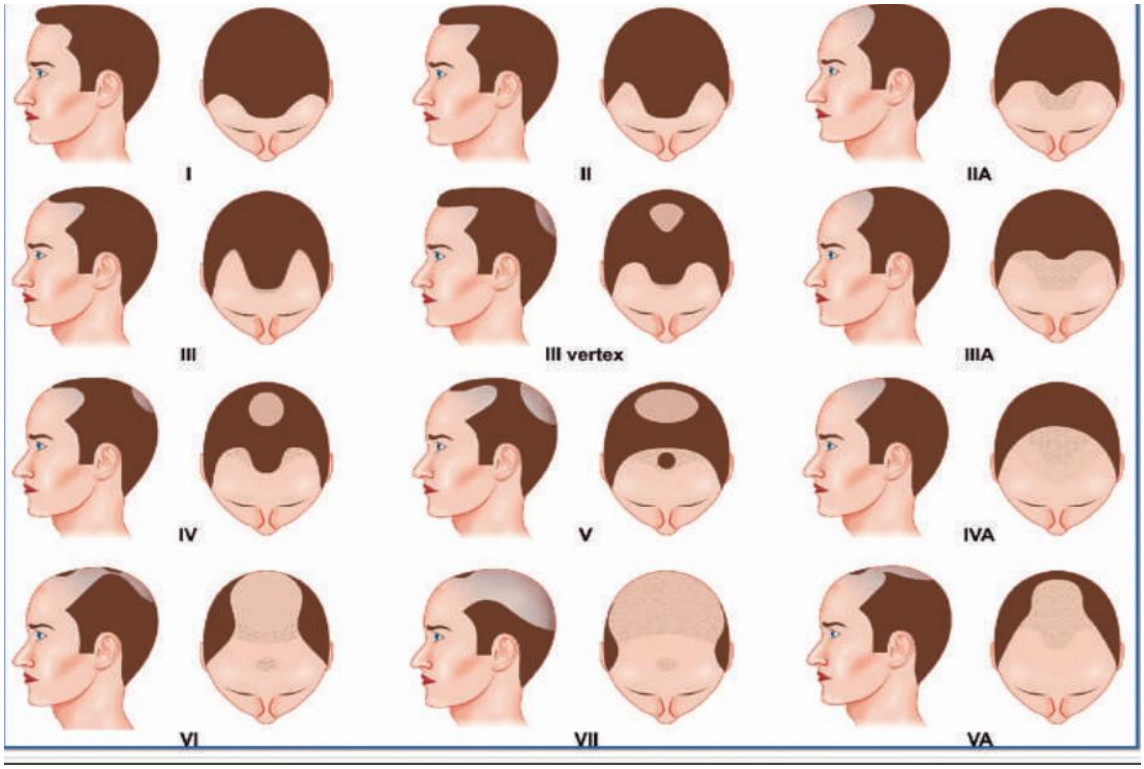
Rather than during the anagen phase, follicular shrinkage takes place in between anagen cycles. The extended interval between the onset of medication and the clinical response could be explained by the limited duration of the androgen impact, since pharmacological interventions become effective only when they reach the point of miniaturization.[57]

A decrease in anagen duration results in lower hair length, whereas an increase in telogen duration delays regeneration, in addition to the shrinkage of hair follicles that causes thin fibers in androgenetic alopecia. As a result, the hairs become so thin and short that they are unable to grow long enough to cover the scalp.[56]

CLINICAL FEATURES:

In the majority of cases, the clinical presentation of MAA is promptly and widely recognized. According to Hamilton and Norwood's, the hair loss progresses in an ordered manner. For male pattern hair loss, these authors employ an adjusted grading system. Miniaturization and decreased hair density are the results of affected hairs. Before complete baldness, there is a general decline in hair density in the impacted areas due to the progressive replacement of terminal hairs by vellus hairs.[58,59]

Although hair loss is usually natural, there may be times when a positive "hair pull" is detected during periods of excessive shedding. When hair can be easily removed from the scalp, the hair pull test is positive. About 80% of patients have a family history of MAA on either side of the family, whereas 20% of cases have no family history.[58]



Norwood-Hamilton classification.

Modified Norwood Hamilton categorization divides MPHL into seven kinds according to severity.[59]

Type I: Minimal recession of the hairline along the anterior border in the fronto-temporal (FT) region.

Type II: The anterior border of the hair in the FT region has triangular areas of recession that tend to be symmetrical. These regions stop at a line drawn in a coronal plane between the external auditory meatus on both sides, and they don't go any farther posterior than around 2 cm anterior. Along the mid-frontal boundary of the scalp, there is either sparse or absent hair.[59]

Type III: Characterized by a deep FT hair recession, usually symmetrical and either bald or sparsely covered with hair. Beyond a point of about 2 centimeters anterior to a line drawn in a coronal plane between the external

auditory meatus on either side, these patches of hair recession stretch farther posterior.[59]

Type IIIv (Vertex): The vertex is where most hair loss occurs. Although there might be some frontal recession, it is not as severe as type III.

Type IV: Compared to type III, the frontal and FT recession is more significant. The vertex area also shows signs of hair sparsity or absence. These patches of baldness are widespread, but they are divided from one another by a band of hair that is relatively dense and connects the fully haired fringes on each side of the head.[59]

Type V: The hair loss over the vertex and FT areas is larger than in type IV and the band of hair between them is narrower and sparser.

Type VI: The hair loss over the FT and vertex regions is confluent and the bridge of hair that crosses the crown is absent.[59]

Type VII: There is only a narrow horseshoe-shaped band of hair that begins laterally just anterior to the ear and extends posteriorly on the sides and fairly low on the occipital area. Variants (Type variants ‘a’): Constitutes 3% of all cases of AGA: (i) The entire anterior border of the hairline progresses posteriorly without the normal island of hair in the mid-frontal region and (ii) there is no simultaneous development of a bald area on the vertex. Instead, the anterior recession just advances posterior to the vertex. Type IIa: The entire anterior border of the hairline lies high on the forehead. The usual mid-frontal island of hair is represented by only a few sparse hairs. The area of denudation extends no farther than 2 cm from the frontal line. Type IIIa: The area of denudation reaches the mid-coronal line. Type IVa: The area of denudation extends beyond the mid-coronal line, and there may be considerable thinning of hair posterior to the actual hairline. Type Va: Most

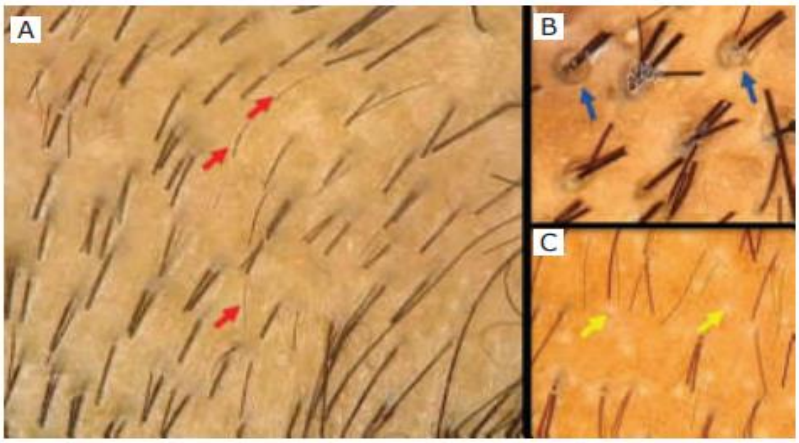
advanced degree of alopecia; however, the bald area does not reach the vertex. This pattern appears to be the most common type in clinical practice.[59]

CARDINAL FEATURES OF MPHL:

- The balding pattern with miniaturized hairs
- Gradual onset with progression
- Onset after puberty
- Family history of baldness
- Thinning with or without gradually developing bald patches
- Negative hair pull test[60]

TRICHOSCOPY:

Trichoscopic observations that are typical of AGA include hair diameter diversity (>20%), reduced number of hairs per FU, peripilar sign or brown halo (observed in early AGA and indicative of perifollicular inflammation), yellow dots (>4 in 4 areas of the frontal area suggests severe miniaturization), white dots, and honeycomb pattern pigment (seen in advance cases).[61]



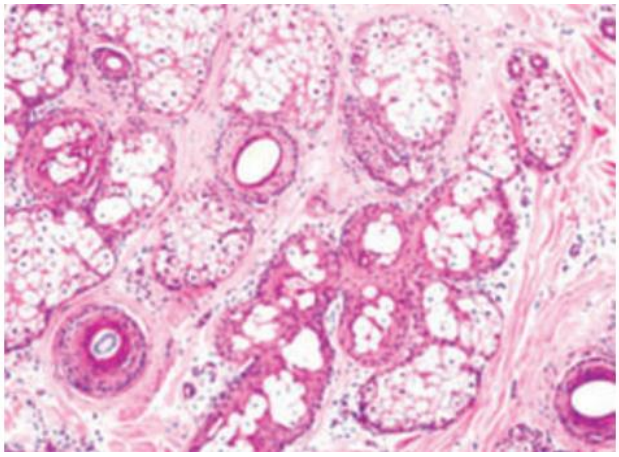
Trichoscopy: (A) Hair diameter diversity (>20%), miniaturized hair (red arrows); (B) Peripilar sign (blue arrows); and (C) White dots (yellow arrows)

HISTOPATHOLOGY:

For MAA, a histological diagnosis is rarely required. The best specimen for patients with uncertain diagnoses is a 4mm punch vertex scalp biopsy. Compared to vertical biopsies, horizontal scalp biopsies provide greater diagnostic data.[62]

The main characteristic observed in scalp biopsies is a decrease in the number of terminal anagen hairs.[63] This decrease appears to be caused by the progressive replacement of terminal hairs with secondary pseudo-vellus hairs, along with residual angiofibrotic tracts.[66]The ratio of terminal to vellus hairs decreases from more than 6:1 to less than 4:1, and the anagen to telogen hair ratio decreases from 12:1 to 5:1.[64]

Given that hair follicle miniaturization is an essential element in the onset and progression of androgenic alopecia, histological diversity in hair diameter represents a significant characteristic, reflecting the various stages of miniaturization and accurately corresponding with the diversity of hair diameter observed in clinical settings.[65]



Transverse section showing variation in hair follicle diameter primarily displaying vellus hairs.

DIFFERENTIAL DIAGNOSIS

Diffuse alopecia areata is the diagnosis from which differentiation may be challenging. Random patches, a diffuse pattern, or a generalized pattern can all be seen in alopecia areata. It is relevant to all age groups. It usually has sudden onsets and remissions interspersed with relapses.[67]

Shedding is evident, and both dystrophic anagen and telogen hairs test positive for hair pull. According to the authors' observations, there is frequently a correlation between AGA and alopecia areata

It can occasionally be challenging to distinguish between diffuse alopecia areata and diffuse unpatterned alopecia (DUPA). Diffuse miniaturization without patchy loss is observed in DUPA. Differentiating between the two disorders is aided by trichoscopic inspection. In these types of diagnostic situations, some people would rather obtain a scalp biopsy for the transverse region.[68]

MANAGEMENT

Balding males have a lot of options at their disposal. First off, it makes sense to forego therapy and let the baldness advance spontaneously because the condition is not life-threatening and the morbidity is variable. In actuality, the great majority of men choose to act in this manner.[69] An adequate explanation of the disease's pathophysiology, its prevalence in the community, and the range of treatment choices available should be part of every patient's support and counselling, regardless of whether they choose to pursue therapy.

Before beginning medical therapy, it is crucial to determine whether patients' expectations on the course of their treatment are realistic. Additionally,

patients should be informed about the benefits of starting treatment early and the need for ongoing care.[82]

Camouflage and Wigs

The simplest and most straightforward method of treating mild MAA is camouflage. Adding tiny fibers held in place electrostatically, coloring the scalp the same color as the hair, and altering hairstyle to hide the balding scalp are all crucial and affordable steps that can produce results that are aesthetically pleasing. Contemporary wigs are natural-looking, easily styled, and cleaned.[83]

Medical Management

The therapeutic choices in medical management can be classified into two groups:

1. Treatments based on evidence
2. Therapies based on evidence

1.TREATMENTS BASED ON EVIDENCE

Currently, the Food and Drug Administration (FDA) has only approved topical minoxidil and oral finasteride as therapies for androgenetic alopecia in men. Although they can only partially correct the baldness, these two drugs can stop more hair loss. To keep the effects going, both need to be used consistently.

Before determining whether to continue treatment, these medications should be administered for at least a year, as clinical response may take six to twelve months to manifest. In the context of androgenetic alopecia, HairMax LaserComb, sometimes referred to as low level laser therapy, has received

FDA approval.The FDA has approved just these therapies to treat androgenetic alopecia.[81]

MINOXIDIL:

One derivative of a piperidino-pyrimidine is minoxidil. The sulfotransferase enzyme transforms it in the ORS of the hair follicle into minoxidil sulfate, which is its active molecule.Its precise mode of action is unclear. However, a few of the hypotheses put forth are as follows:

Anagen phase activation and lengthening; increased VEGF (vascular endothelial growth factor) secretion leading to an increase in perifollicular angiogenesis; accelerated cell proliferation; and immune regulatory activity.[70]

• Ca2+ influx induction, which causes stem cell differentiation

At a dose of 10–40 mg, minoxidil in an oral formulation has been used in general medicine to treat severe and uncontrolled hypertension.[74,75] Inadvertently, the early trials of oral minoxidil as an antihypertensive medication indicated the drug's propensity to stimulate hair growth and noted side effects such as hypertrichosis and hirsutism with chronic use.[73]A topical minoxidil formulation was subsequently created for the treatment of AGA as a result of these findings.

Topical minoxidil comes in foam, aerosol, and lotion forms in concentrations of 2%, 5%, 10%, 12.5%, and 15% (in propylene glycol). Lotion, foam, and aerosol with concentrations of 2% and 5% are FDA-approved, nevertheless. In 4–8 months, hair growth begins to show, and by 12–18 months, it stabilizes.[71]

Facial hypertrichosis and irritating contact dermatitis are frequent adverse effects.[72] There are now many topical minoxidil combinations available,

including minoxidil plus peptide, finasteride with minoxidil, and peptide alone, however there is still a dearth of supporting data.

However, the adverse effects at recommended dosages have restricted the use of oral minoxidil for AGA. However, the use of oral minoxidil at low doses (0.25–5 mg once daily) for androgenetic alopecia is receiving more and more attention.[76,77]

5-ALPHA REDUCTASE INHIBITORS

A class of antiandrogenic medications known as five-ARIs is equivalent to dihydrotestosterone (DHT) blockers that are authorized for use in benign prostatic hyperplasia.The three different metabolic pathways of bile, androgens, and estrogen are regulated by the 5-ARI enzyme family (types I, II, and III).[78,79]

FINASTERIDE:

The FDA has approved finasteride as a pharmacologic treatment for male pattern hair loss and benign prostatic hyperplasia.Finasteride was approved in 1997 at a dosage of 1 mg to treat male pattern hair loss.[85]

Synthetic azo-steroid finasteride is a strong and extremely specific inhibitor of type II 5 alpha reductase. It does not oppose androgens. It binds to the enzyme permanently, preventing testosterone from being converted to dihydrotestosterone. As a result, the biological action lasts far longer than the pharmacokinetic half-life, which is roughly eight hours.

Finasteride only inhibits 5-alpha reductase type 2, but dutasteride inhibits both isoenzymes (types 1 and 2), making it a dual blocker. Finasteride was authorized for use in AGA by the US Food and Drug Administration (FDA) in 2007.[84]

For oral administration, finasteride is available as a 1 mg or 5 mg tablet. Adults with androgenetic alopecia should take 1 mg every day. Loss of libido, erectile dysfunction (2%–4%), decreased ejaculatory volume, and gynecomastia are common side effects associated with finasteride. Orthostatic hypotension is also a problem with finasteride, especially in the early stages of treatment.[86]

LOW-LEVEL LASER THERAPY

Low-level laser treatment (LLLT) emits light at a 5 mW power level with a wavelength of 650–900 nm. According to the FDA, it is safe to use in AGA. By lowering inflammation and producing vasodilatation through nitric oxide release, it encourages the development of hair. It can only be taken in addition to other AGA therapies that are now available.[87]

HAIR TRANSPLANTATION

In advanced cases of hair loss (AGA IV–VI), it is recommended in addition to the medical course of treatment. The most crucial element of a good hair transplant outcome is careful case selection and counselling. A patient who has no comorbidities, a stable, high-quality donor location, healthy recipient area, and reasonable expectations would be considered excellent. It is important to carefully consider your options before undergoing surgery since certain disorders, such as DUPA, occipital cicatricial alopecia, and advanced AGA cases (VI–VII), may not be suitable candidates for the procedure and have a very low donor pool. The most crucial stage in guaranteeing a patient's satisfaction is customizing a decent hairline for each particular patient.

Depending on the needs for the graft, transplantation can be completed using the follicular unit transplantation technique, the follicle unit extraction

technique, or both in a single session. The hairline, frontotemporal recession, mid-scalp, and vertex are areas that are frequently addressed.

THERAPIES BASED ON EVIDENCE

DUTASTERIDE:

Inhibits both Type I and Type II 5α-reductase enzyme.This medication was first created in the early 2000s to treat benign prostatic hyperplasia (BPH). It is a patterned hair loss treatment that is not FDA-approved. When compared to finasteride, it has three times the potency to inhibit type II 5α-reductase and 100 times the potency to inhibit type I 5α-reductase.[80,88]

Soft gelatin capsules with 0.5 mg, 2.5 mg, and 5 mg of it are available. It is administered twice a week at different doses of 0.5 mg.A common side effect is a decrease in libido, though most of the time this may be reversed.Eventually, with persistent treatment, the side effects go away.

At 24 weeks, dutasteride 0.5 mg was found to be statistically better to finasteride 1 mg and placebo in a recent phase III experiment. Finasteride and dutasteride, two five-ARIs, work well to treat AGA. Unlike finasteride, dutasteride is a dual reductase inhibitor that can reverse shrinkage. The safety profiles of finasteride and dutasteride are comparable, and there aren't any significant issues with neurotoxicity, teratogenicity, fertility, or sexual side effects. These medications are essential to the treatment of AGA.[89]

AMINOACIDS

By lowering blood reactive oxygen species, oral L-cysteine promotes hair growth, albeit it may only benefit those who eat a diet low in essential nutrients.[90]

CAFFEINE

A derivative of methylxanthine, caffeine is an alkaloids found mostly in coffee plants. By inhibiting phosphodiesterase and adenylyl cyclase inhibitor, it raises intracellular cyclic adenosine monophosphate (AMP) and stops hair follicle shrinking. Caffeine-infused shampoos have been demonstrated to reduce hair loss and improve hair texture.[91]

MELATONIN

It has been demonstrated that topical 0.1% melatonin lotion, with its antioxidant qualities and ability to suppress free radical toxicity, increases anagen hairs and modulates hair development.[92]

PLATELET-RICH PLASMA (PRP)

It is an autologous preparation made with the blood of the patient. Three to five times as many platelets will be present as there are in the circulation. Numerous cytokines and growth factors, including VEGF, PDGF, transforming growth factor, and other bioactive proteins, are present in it.[93]

Injections of platelet-rich plasma (PRP) have become a viable regenerative treatment for androgenetic alopecia because of its autologous origin, which reduces the possibility of adverse effects. Growth factor concentrate (GFC) therapy, a variation on platelet-rich plasma (PRP) therapy, has shown promise in the treatment of androgenetic alopecia and hair restoration.[94,96]

HAIR CAMOUFLAGE

Patients with an advanced grade of MPHL or those who choose not to pursue medicinal or surgical treatments may consider it as a therapy option. Hair fibers, powder cakes, scalp lotions and sprays, wigs, and scalp

micropigmentation are just a few of the alternatives available. It is crucial that dermatologists talk with patients about their needs and expectations before suggesting the best course of action.[95]

MATERIALS & METHODS

MATERIALS AND METHODS

Source of data:

All male patients with non-scarring hair loss attending the outpatient clinic of Dermatology, Venereology and Leprosy in R L Jalappa Hospital and Research centre attached to Sri Devaraj Urs Medical College, Tamaka, Kolar will be screened and those satisfying the inclusion criteria will be included in the study between October 2022 and August 2024.

Study design: Randomized Control Trial

Sample size calculation:

Sample size was estimated based on the marked improvement proportion seen in the recent research article- "Efficacy and Safety of Oral Minoxidil 5 mg Once Daily in the Treatment of Male Patients with Androgenetic Alopecia" by Ratchathorn Panchaprateep, Suparuj and "5-alpha Reductase Inhibitors in Androgenetic Alopecia: Shifting Paradigms, Current Concepts, Comparative Efficacy and Safety" by Rachita Dhurat.

The marked improvement proportion in the treatment of male androgenetic alopecia with oral minoxidil is 43.3% and the marked improvement proportion seen with oral dutasteride is 77.4%.So with 95% Confidence interval, Alpha error 5% and Power value of 80% - the estimated total sample size for the study is 64 and the sample size per group is 64. Sample size has been calculated using OPEN EPI data version 3.01.

Proportion in group Ⅰ - 77.4%

Proportion in group Ⅱ - 43.3%

Power (%) - 80%

Alpha error - 5%

Required sample size for each arm = 32

Total sample size for the study = 64

Method of collection of data (including sampling procedure)

SELECTION CRITERIA

Inclusion criteria

1. Male patients aged 18-30 years with clinical features suggestive of androgenic alopecia (MPHL).
2. Patients with AGA Grade 1-5 of modified Hamilton-Norwood scale.
3. Person who has not taken medical treatment for AGA in any form for the past 3 months.

Exclusion criteria

1. Male patients with alopecia secondary to known endocrinologic disorder (Cushing's disease, Addison's disease, etc.)
2. Male patients with other non scarring alopecias
3. Elderly patients with severe hypertension associated with other cardiovascular problems.

Sample size:

Total sample size for the study is 64.

Sample size per group is 32. 32 participants will be treated with oral minoxidil 5 mg once daily and other 32 participants will be treated with oral dutasteride 0.5 mg thrice a week for a period of 12 months.

Randomization:

Total sample size for the study is 64 and the sample size per group (Group A and Group B)is 32.

To avoid the SELECTION BIAS in the study,randomization of the sample selection is essential.

Hence randomization is done using COMPUTER GENERATED BLOCK RANDOMIZATION.

COMPUTER GENERATED BLOCK RANDOMIZATION PLAN is generated from www.randomization.com

A Randomization Plan
from
http://www.randomization.com

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57. A
58. B
59. A
60. B
61. A
62. B
63. A
64. A

Seed: 10600

Treatment groups: Group A, Group B
Block size: 2
List length: 64

Assessment of efficacy and safety profile in both the groups were done by using Global Photographic Assessment(GPA) scale.

GPA SCALE:

Patients of this study had undergone serial photography of the hair loss areas at baseline and at subsequent sittings.Lighting and positioning were kept identical for all the serial photographs.

The serial photographs were assessed independently by a blinded third observer.The blinded third observer gave the grading of efficacy of the treatment modality based on the Global Photographic Assessment (GPA) scale. GPA score was calculated for all the patients of the study at the baseline presentation and at subsequent follow up sittings.

The blinded third observer assigned a score of 0,1,2,3 depending on whether the response was less than 25%, between 25% and 50%, between 51 and 75% or greater than 75 %.

STATISTICAL ANALYSIS:

Data will be entered into a Microsoft Excel Data Sheet and will be analyzed using SPSS 22 version software. Categorical data will be represented in the form of Frequencies and proportions. Chi-square will be used as the test of significance. Continuous data will be represented as mean and standard deviation. p value< 0.05 will be considered as statistically significant.

RESULTS

RESULTS

Table 1: Distribution of patients according to their Age Group (N=64)

Age Group	Frequency	Percent	
18-23 years	26	40.62	
24-30 years	38	59.38	
Mean ± SD	25.25 ± 3.73		
	Group A	Group B	Total
18-23 years	14	12	26
24-30 years	18	20	38
Total	32	32	64

The mean age of the patients included in our study population was 25.25 ± 3.73. The majority of patients belong to 24-30 years of age(59.38%) followed by 18-23 years of age (40.62%)

Figure1: Bar diagram depicting distribution of patients according to their age groups in both the study groups

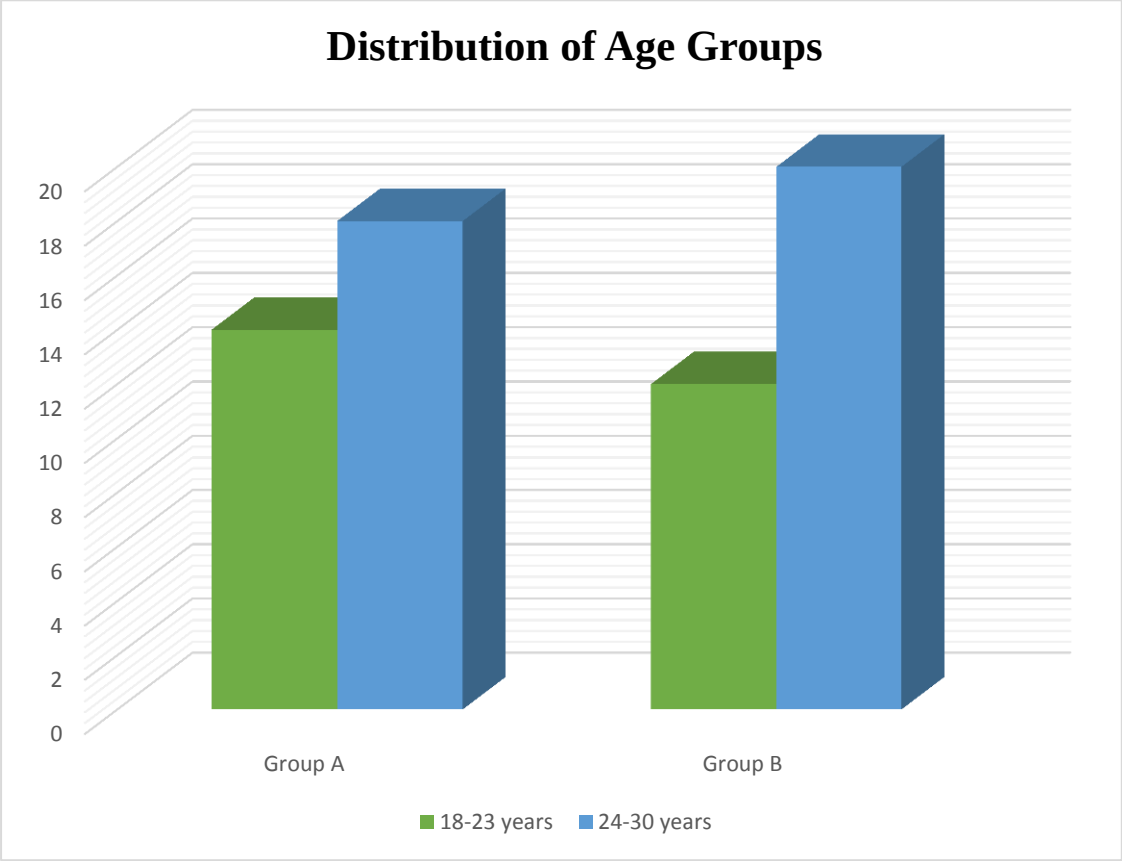


Table 2: Association between occupation and Study Group.

Occupation	Group A	Group B	Total	P value
Student	8 (12.50%)	7 (10.94%)	15 (23.44%)	0.382
Skilled professionals	16 (25.00%)	22 (34.38%)	38 (59.38%)	
Semi-skilled professionals	5 (7.81%)	2 (3.13%)	7 (10.94%)	
Unskilled workers	1 (1.56%)	3 (4.68%)	4 (6.25%)	
Total	30 (46.87%)	34 (53.13%)	64 (100%)	

Overall, among 64 study participants, commonly observed occupation was skilled professionals (59.38%) followed by students (23.44%), semi-skilled professionals (10.94%) and unskilled workers (6.25%).

Figure 2: Bar diagram depicting the distribution of patients according to their occupation in both the groups.

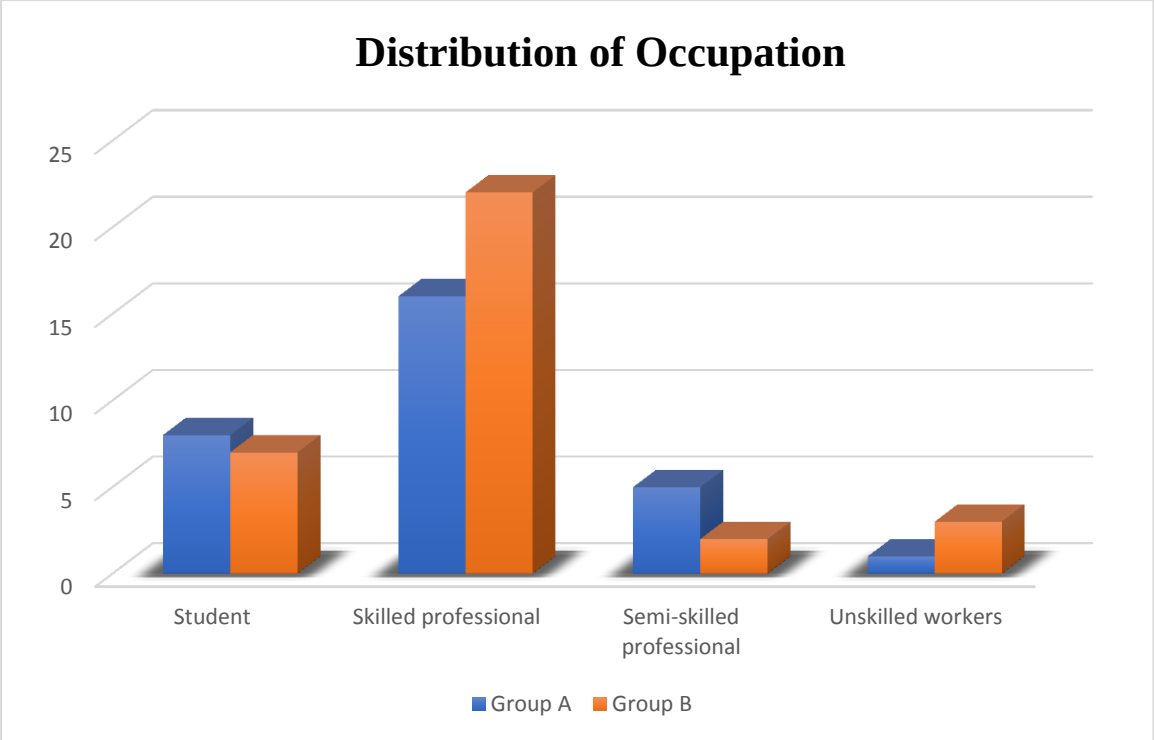


Table 3: Descriptive analysis of baldness in family members in the study population (N=64)

Baldness in family members	Frequency	Percent
Yes	39	60.94
No	25	39.06
Total	64	100

Among the study population, 39(60.94%) participants had history of baldness in family members and 25(39.06%) participants had no history of baldness in family members.

Figure 3: Bar diagram depicting baldness in family members in the study population(N=64)

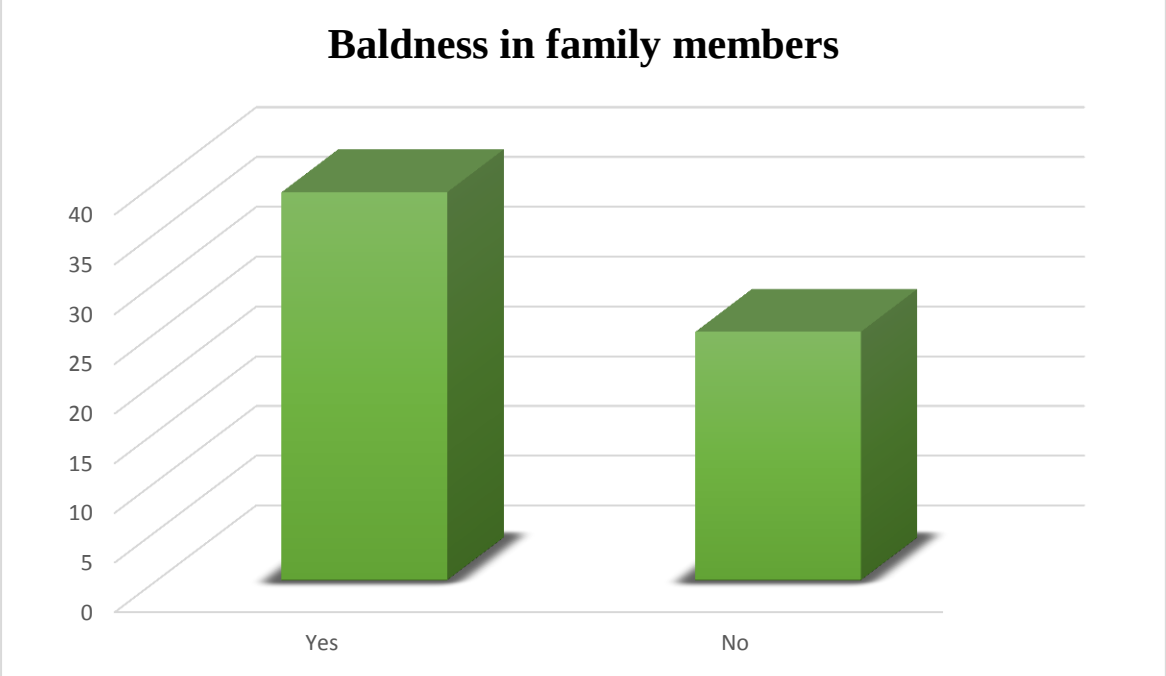


Table 4: Association between Norwood Hamilton scale and Study Group.

Norwood Hamilton scale	Group A	Group B	Total
I	1 (1.56%)	0 (0.00%)	1 (1.56%)
II	3 (4.69%)	2 (3.13%)	5 (7.81%)
III	9 (14.06%)	9 (14.06%)	18 (28.13%)
IV	12 (18.75%)	13 (20.31%)	25 (39.06%)
V	8 (12.50%)	7 (10.94%)	15 (23.44%)
Total	33 (51.56%)	31 (48.44%)	64 (100%)

Among the study population, the most common Norwood-Hamilton classification was Type IV (39.06%) followed by Type III (28.13%) then, Type V (23.44%) ,Type II (7.81%) and Type I (1.56%).

Figure 4: Bar diagram depicting the distribution of Norwood Hamilton Scale in patients in both the groups.

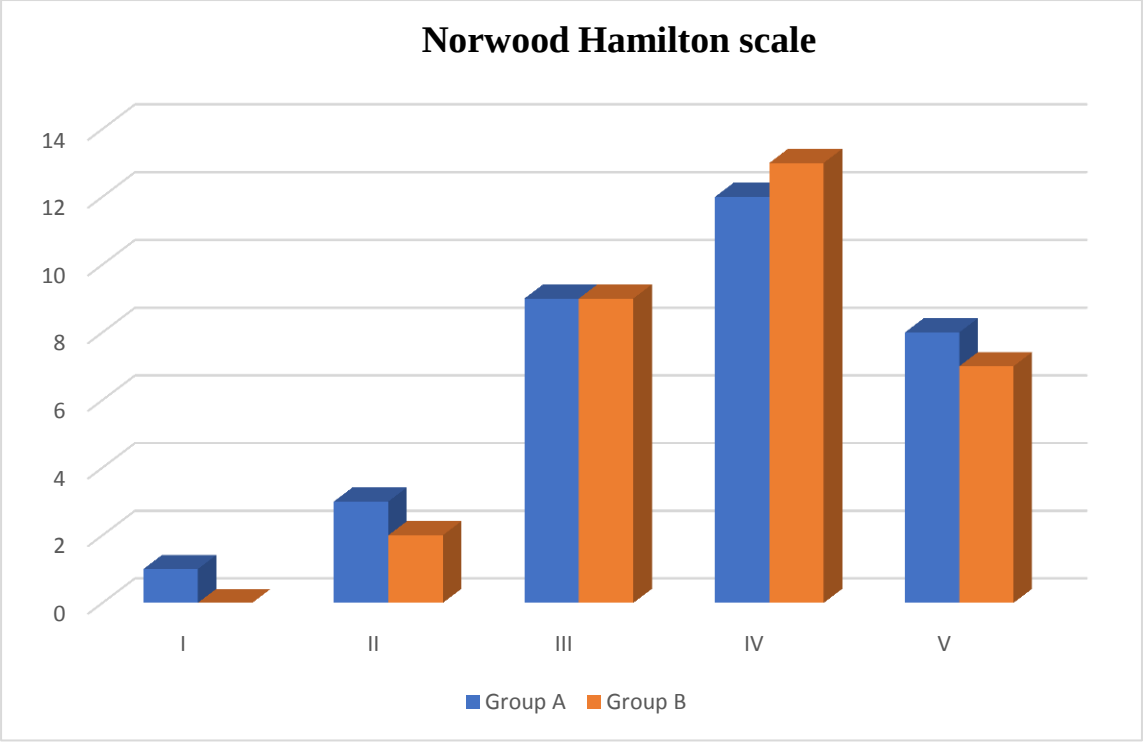


Table 5: Association between Norwood Hamilton scale and Age group.

Norwood Hamilton scale	Total	Age 18-23 years	Age 24-30 years
I	1 (1.56%)	1 (1.56%)	0 (0.00%)
II	5 (7.81%)	2 (3.13%)	3 (4.69%)
III	18 (28.13%)	6 (9.38%)	12 (18.75%)
IV	25 (39.06%)	10 (15.63%)	15 (23.44%)
V	15 (23.44%)	7 (10.94%)	8 (12.50%)
Totals	64 (100%)	26 (40.62%)	38 (59.38%)

Overall among the study participants, majority of the study participants 15(23.44%) who had Type IV Norwood-Hamilton scale belonged to the age group of 24-30 years followed by 12(18.75%) who had Type III Norwood-Hamilton scale are of 24-30 years.

Figure 5: Bar diagram depicting the distribution of Norwood Hamilton scale in different age groups

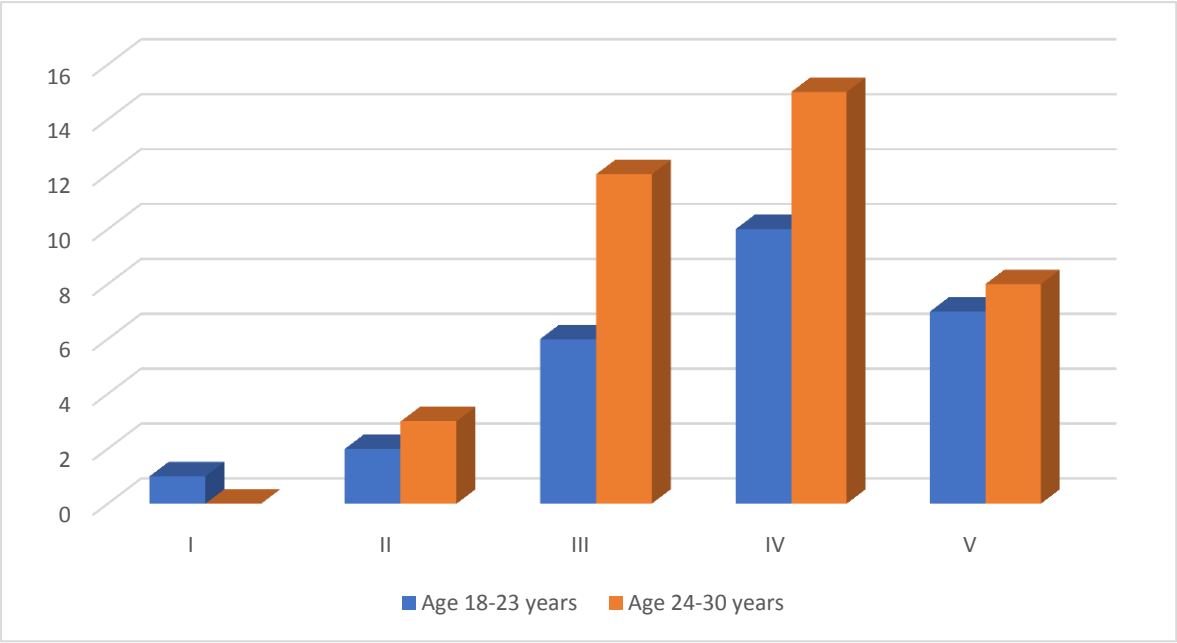


Table 6: Efficacy assessed using GPA-Scale

	MEAN SCORE				
GROUP	BASELINE	12 WEEKS	24 WEEKS	36 WEEKS	48 WEEKS
GROUP A	0.00	0.81	1.72	2.41	2.69
GROUP B	0.00	1.00	1.97	2.56	2.94
P Value	NA	<0.001	<0.001	0.436	<0.001

The Blinded Third observer assessed the efficacy of the treatment modalities using GPA-Scale. At the end of 48 weeks, the Blinded Third Observer gave a score of 3(>75% improvement) to all cases in Group B(93.75%-30) and 68.75% of cases in Group A (22 cases).The Blinded Third Observer gave a score of 2(50-75% improvement) to 2(6.25) in Group B and 10(31.25%) in Group A.

Figure 6: Line diagram showing efficacy assessed using GPA-Scale

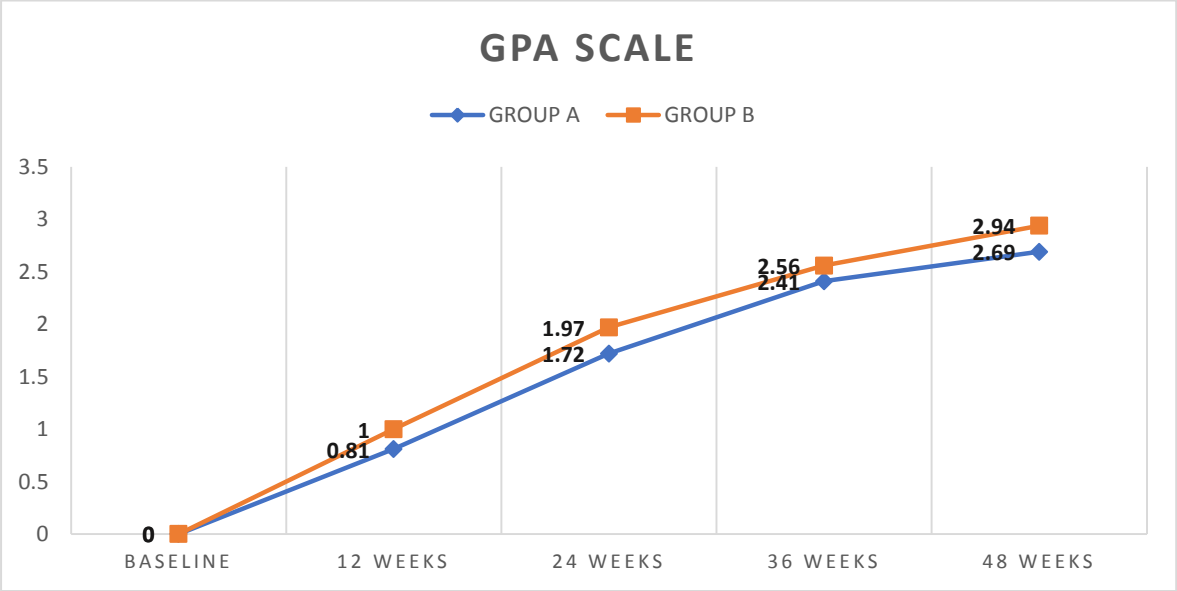


Table 7: Adverse Effects observed in both the groups.

	Erectile Dysfunction	Pedal Edema	Orthostatic Hypotension	Decreased libido	Hypertrichosis
Group A	0 (0.0%)	4 (12.5%)	6 (18.8%)	0 (0.0%)	14 (43.8%)
Group B	6 (18.8%)	0 (0.0%)	0 (0.0%)	9 (28.1%)	0 (0.0%)

The most common adverse effect observed in Group A was hypertrichosis (43.8%) followed by orthostatic hypotension (18.8%) and pedal edema (12.5%).The most common adverse effect observed in Group B was decreased libido(28.1%) followed by erectile dysfunction(18.8%)

Figure 7: Bar diagram showing the distribution of Adverse effects in patients in both the groups.

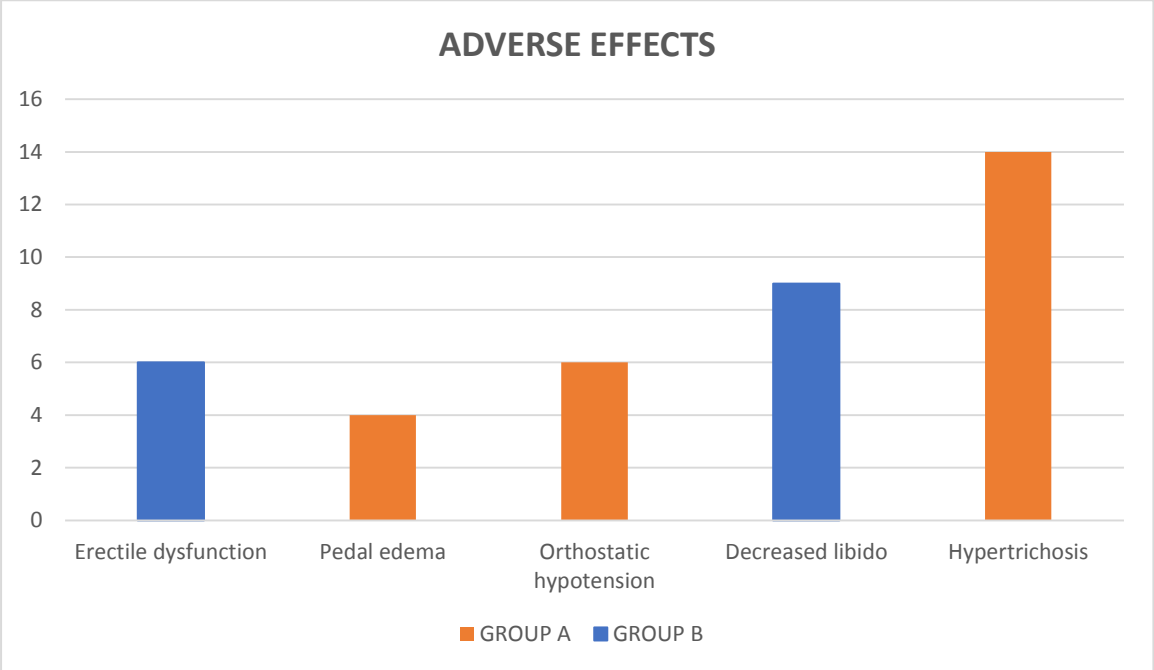
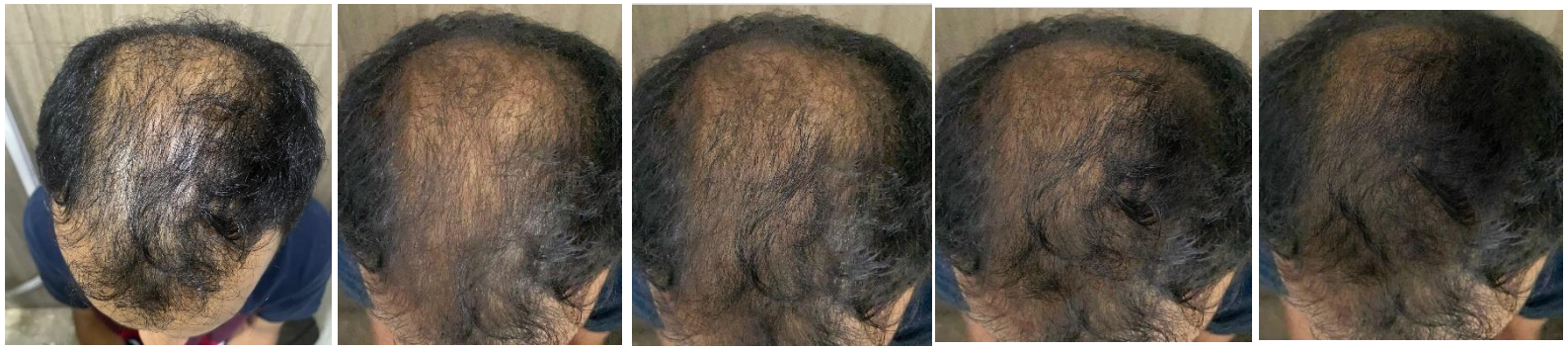


FIGURE 8: GROUP A -PATIENT 1

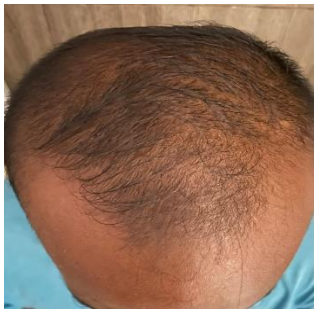
PATIENT 1:



BASELINE	12 WEEKS	24 WEEKS	36 WEEKS	48 WEEKS
Assessment of hair growth using GPA Scale= 0	GRADE 0(15%) Mild improvement	GRADE 1 (30%) Mild improvement	GRADE 2(60%) Moderate improvement	GRADE 3(80%) Marked improvement

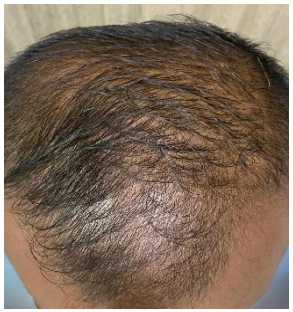
FIGURE 9: GROUP A -PATIENT 2

PATIENT 2:



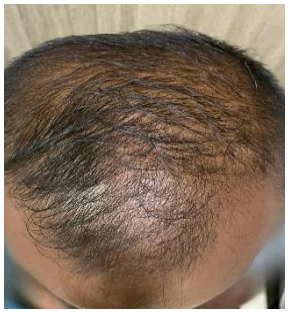
BASELINE

Assessment of hair growth using GPA Scale= 0



12 weeks

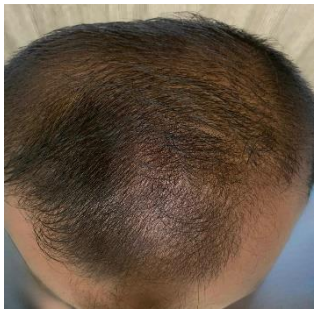
GRADE 1(25%) Mild improvement



24 weeks

GRADE 2 (30%)

Moderate improvement



36 weeks

GRADE 2 (50%)

Moderate improvement

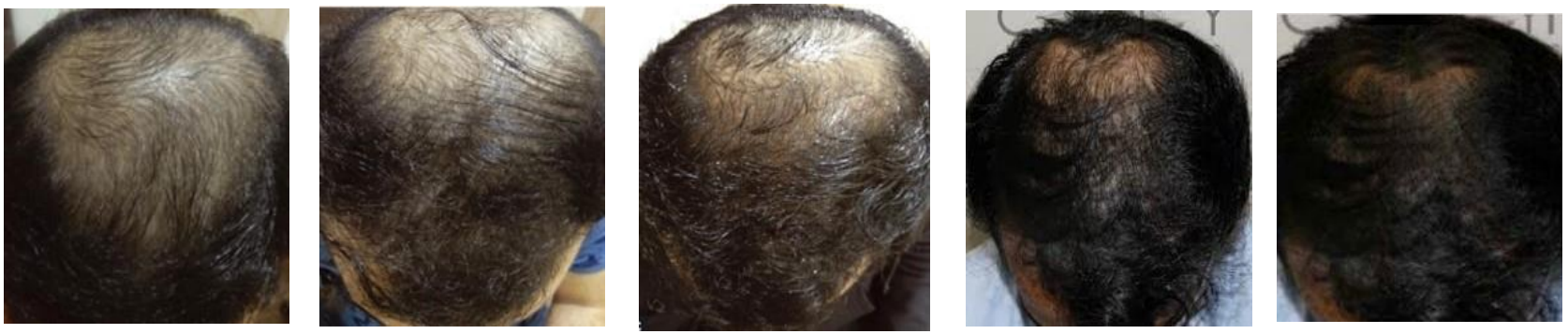
48 weeks

GRADE 3(75%)

Marked improvement

FIGURE 10: GROUP B -PATIENT-1

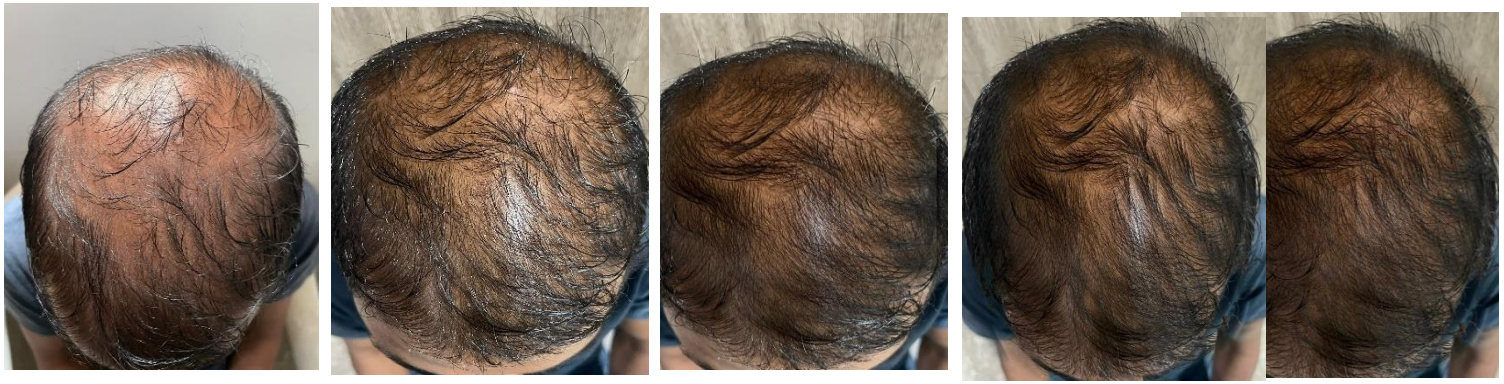
PATIENT 1:



BASELINE	**12 WEEKS**	**24 WEEKS**	**36 WEEKS**	**48 WEEKS**
Assessment of hair growth using GPA Scale= 0	GRADE 1(15%) Mild improvement	GRADE 2(30%) Mild improvement	GRADE 3(75%) Moderate improvement	GRADE 3(90%) Marked improvement

FIGURE 11: GROUP B – PATIENT 2

PATIENT 2:



BASELINE	12 WEEKS	24 WEEKS	36 WEEKS	48 WEEKS
Assessment of hair growth using GPA Scale= 0	GRADE 1(15%) Mild improvement	GRADE 2(30%) Mild improvement	GRADE 3(75%) Moderate improvement	GRADE 3(90%) Marked improvement

DISCUSSION

DISCUSSION:

AGA is a multifactorial disorder caused by interactions between several genes and environmental factors. The pathogenesis of patterned baldness differs between men and women. AGA remains a source of psychological distress to the balding men.[97]

There are various treatment modalities for male AGA, but there are only limited effective modalities among which low dose oral Minoxidil and low dose oral Dutasteride is gaining popularity in recent times since these drugs demonstrated a promising safety and efficacy profile in the treatment of male AGA.

Topical minoxidil is approved by FDA to treat female-pattern hair loss (FPHL) and male androgenetic alopecia (AGA) while oral minoxidil is off-label. Low-dose oral minoxidil (LDOM), with a daily dosage ranging from 0.25 to 5 mg, has a good safety and efficacy profile for treating both male AGA and FPHL, based on a number of studies.

Minoxidil acts as a vasodilator, a Wnt/β-catenin signaling pathway inducer, an anti-inflammatory agent, and possibly an antiandrogen.Minoxidil promotes hair growth in a number of ways.

Minoxidil stimulates the ATP-sensitive potassium channel, which eventually leads to vasodilation. More oxygen and nutrients can reach the hair follicles through dilated blood vessels, which may promote the growth of new hair.[103] Additionally, minoxidil may decrease inflammatory mediators like interleukin-1 alpha, which promotes hair growth, by downregulating the production of the interleukin-1 alpha gene.[105]This may potentially decrease the perifollicular microinflammation.

Minoxidil may function as an antiandrogen which promotes hair growth by suppressing the expression of the 5 alpha-reductase type II gene. By promoting the production of VEGF in the dermal papilla cell, minoxidil also stimulates Wnt/b-catenin signaling pathway, which results in the regeneration of hair follicles.[104]

Minoxidil has an essential role in the anagen phase, which could stimulate hair growth. The drug may accelerate the anagen phase by increasing DNA synthesis in anagen bulb. Moreover, minoxidil may alter both the anagen and telogen phases, or it may lengthen the anagen phase. Hence, oral minoxidil may be an effective substitute[100], especially for male AGA and FPHL patients who have not seen positive results from topical minoxidil.[106]

The most common side effect of oral Minoxidil is hypertrichosis, which was reported as mild and is dose dependent. Other less common side effects include postural hypotension/dizziness, lower limb edema, mild blood pressure changes, and EKG changes. No severe cardiopulmonary side effects were noted.[107]

Dutasteride is a type I and II 5-ARI that is FDA approved for treatment of benign prostatic hyperplasia and is used off label to treat AGA. In clinical practice, finasteride and dutasteride are used to treat males with AGA.

Both finasteride and dutasteride are thought to be effective in reducing male pattern hair loss because they inhibit the function of hair follicles by lowering DHT levels. Dutasteride is more effective than finasteride since it inhibits both 5-α-reductase types I and II, which results in a 90% decrease in serum DHT levels as opposed to a 70% decrease with finasteride.[109]

The most often reported sexual side effects with 5-ARIs are Erectile dysfunction, ejaculatory dysfunction, and loss of libido; these rates are

comparable for finasteride and dutasteride use. According to several studies, 5-ARI sexual side effects are uncommon but considerable when compared to placebo, and frequently resolve on their own without the need to discontinue the treatment.[101]

In our study, we compared the treatment of Male AGA with low dose oral minoxidil (Group A) versus low dose oral Dutasteride (Group B)

In this study, majority of AGA patients (59.38%) belonged to 24-30 years of age followed by 18-23 years of age (40.62%). This finding is consistent with observations of Ratchathorn Panchaprateep et al in their study where they concluded that age range above 24 years are having the highest prevalence.[98]

Many studies have reported the family history of baldness in patients with male AGA. In previous studies,Cameron et al suggest that the probability of male pattern hair loss is dependent on family history and age.[108]These findings are consistent with our study where the history of family members with baldness is present in 60% of study population and hence we infer that expression of male pattern hair loss can be influenced by familial prevalence. In this study, the most common Norwood Hamilton classification was Type Ⅳ(39.06%) followed by Type Ⅲ (28.13%) and Type Ⅴ (23.44%). This finding is consistent with the observations of Suparaj Lueangarun et al.[98]

In our study, the efficacy of both treatment modalities was assessed using GPA scale. At the end of 48 weeks, GPA scale showed a score of 3(>75% improvement) to all cases treated with oral Dutasteride 0.5mg thrice weekly and 68.75% cases treated with oral minoxidil 5 mg. This finding is consistent with the observations of Aditya K Gupta et al and Rachita S Dhurat.[99]

In this study, the patients who were treated with Oral Minoxidil 5 mg had adverse effects such as Hypertrichosis(43.8%) followed by orthostatic

hypotension (18.8%) and pedal edema (12.5%). This result is in line with the observations of Mesbah Talukder, Amer Shemar in their research.[102]

Those patients in Group B who were treated with oral Dutasteride had minimal adverse effects compared to Group A. The side effects were decreased libido (28.1%) and erectile dysfunction (18.8%) which were reversible. This finding is consistent with the research of Yunbu Ding et al.[101]

CONCLUSION

CONCLUSION

Male AGA is a multifactorial disease which occurs due to interplay of several environmental and genetic factors. Also, AGA in young men can significantly impact self-esteem, confidence and mental health. Early treatment with options like oral Minoxidil and oral Dutasteride can slow hair loss, promote hair regrowth and improve the quality of life.

In this study, we compared and documented the efficacy and safety of low dose oral Minoxidil and low dose oral Dutasteride in the treatment of Male AGA.

In this study, it is evident that both the drugs oral Minoxidil and Oral Dutasteride showed significant improvement in the initial few weeks of therapy but patient satisfaction and greater treatment efficacy is seen with low dose oral Dutasteride in the subsequent follow up. Also, the side effects are minimal in patients treated with oral Dutasteride than the patients treated with oral Minoxidil.

Hence, we conclude that oral Dutasteride is a better treatment modality compared to oral Minoxidil for AGA with respect to efficacy, safety and adverse events.

SUMMARY

SUMMARY

A total of 64 AGA patients participated in the study as a part of RCT. The patients were equally divided into 2 groups: Group A and Group B, each of which had 32 individuals. An informed consent was procured from AGA patients about the participation in the study, disease nature and necessity to follow-up.

Male patients aged 18-30 years who had not taken any treatment for the past 3 months were enrolled in the study. Male patients with other non- scarring alopecia, systemically ill patients were excluded from the study.

Patients in Group A was treated with low dose oral Minoxidil 5 mg once daily for 12 months and patients in Group B was treated with low dose oral Dutasteride 0.5 mg thrice weekly for 12 months.

The efficacy and safety of treatment methods in both the groups were assessed using GPA scale and adverse effects were documented. Follow-up was done at 12 weeks, 24 weeks, 36 weeks and 48 weeks. Majority of the patients in Group A and Group B belonged to the age group of 24-30 years (59.38%) followed by 18-23 years of age (40.62%)

In this study, there was a familial history of AGA in 60.94% of the individuals. Also 59.3% were skilled professionals followed by students (23.44%), semi-skilled professionals (10.94%) and unskilled workers (6.25%).

The most common Norwood Hamilton classification was Type Ⅳ (39.06%) followed by Type Ⅲ (28.13%), Type Ⅴ (23.44%), Type Ⅱ (7.81%), Type Ⅰ (1.56%)

The mean GPA Scale at baseline, 12 weeks, 24 weeks, 36 weeks and 48 weeks in Group A was 0, 0.81, 1.72, 2.41 and 2.49 respectively. The mean GPA scale at baseline, 12 weeks, 24 weeks, 36 weeks, 48 weeks in Group B was 0,1.00,1.97,2.57 and 2.94 respectively.

P value was <0.001 for GPA after 12 weeks, 24 weeks, 36 weeks and 48 weeks. However, GPA Scale at the end of 36 weeks was not <0.001(p value=0.436). Hence, the data of efficacy of both treatment modalities assessed at the end of 48 weeks using GPA Scale were statistically significant.

43.8% of patients in Group A treated with oral Minoxidil had hypertrichosis as an adverse event, whereas 28.1% of the patients in Group B treated with oral Dutasteride had decreased libido which was reversible.

In the first few weeks of treatment, both oral Dutasteride and oral Minoxidil had shown substantial improvement, however oral Dutasteride was associated with greater therapeutic efficacy and patient satisfaction in the subsequent follow-up. Therefore, based on efficacy, safety and adverse effects, we conclude that oral Dutasteride is a better therapeutic option for AGA than oral Minoxidil.

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ANNEXURE

ANNEXURE-1

PROFORMA

Name:

Age:

Sex:

Occupation:

UHID number:

Phone number:

Address:

Date :

Chief complaints:

History of Presenting Illness:

Age of Onset:

Site of onset:

Duration:

Any Associated Symptoms: itching.

Mode of spread: static/ growing/ receding

Use of any drugs before onset of illness

Aggravating factors: Occupational/ hobbies/ trauma/ drug/ work/ sunlight/ emotional factors/ food/ cosmetics/ chemicals/ any other:

Recovery: Some/ good/ poor/ no response.

Past history:

Associated systemic diseases: DM/ HTN/ Thyroid disease.

Associated cutaneous diseases:

Family history:

Similar complaints:

Other skin problems:

Personal history:

Diet: veg/ non veg/ mixed

Bowel/ Bladder habits: regular/ altered.

Sleep- adequate/ disturbed

Appetite-

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 :

CVS

RS

PER ABDOMEN

CNS

LOCAL EXAMINATION :

INVESTIGATIONS :

FINAL DIAGNOSIS :

TREATMENT :

REMARKS OF THE GUIDE :

INFORMED CONSENT FORM

I Mr. _____ have been explained in my own understandable language, that I will be included in a study which is **COMPARISON OF ORAL MINOXIDIL WITH ORAL DUTASTERIDE IN THE TREATMENT OF MALE ANDROGENETIC ALOPECIA - A RANDOMIZED CONTROL TRIAL.**

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.

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:	Relation to patient:
Place:	

ನಾನು ಶ್ರೀ/ಶ್ರೀಮತಿ. _____ ಅನ್ನು ನನ್ನ ಸ್ವಂತ ಅರ್ಥವಾಗುವ ಭಾಷೆಯಲ್ಲಿ ವಿವರಿಸಲಾಗಿದೆ, ಪುರುಷ ಆಂಡ್ರೊಜೆನೆಟಿಕ್ ಅಲೋಪೆಸಿಯಾ ಚಿಕಿತ್ಸೆಯಲ್ಲಿ ಬಾಯಿಯ ಮಿನಾಕ್ಸಿಡಿಲ್ ಅನ್ನು ಓರಲ್ ಡ್ಯುಟಾಸ್ಟರೈಡ್‌ನೊಂದಿಗೆ ಹೋಲಿಸುವ ಅಧ್ಯಯನದಲ್ಲಿ ನನ್ನನ್ನು ಸೇರಿಸಲಾಗುವುದು - ಒಂದು ಯಾದೃಚ್ಛಿಕ ನಿಯಂತ್ರಣ ಪ್ರಯೋಗ.

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.

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-2

PATIENT INFORMATION SHEET

STUDY TITLE: COMPARISON OF ORAL MINOXIDIL WITH ORAL DUTASTERIDE IN THE TREATMENT OF MALE ANDROGENETIC ALOPECIA - A RANDOMIZED CONTROL TRIAL.

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

OBJECTIVES:

1. 1.To compare the efficacy of low dose oral minoxidil with that of low dose oral dutasteride.
2. To document the adverse effects associated with that of oral minoxidil and oral dutasteride.

Androgenetic alopecia is a genetically predetermined disorder due to excessive response to androgens. MPHL is the most common hair loss disorder in the men. The MPHL is a non-scarring progressive thinning of hair which causes psychological distress affecting quality of life of the patient.

Androgenetic alopecia can be diagnosed by clinical examination. In this study two treatment modalities for androgenetic alopecia are documented.

In this study, Group A Participants - will be treated with oral minoxidil 5 mg once daily for a period of 12 months.

Group B participants - will be treated with oral dutasteride 0.5 mg thrice a week for a period of 12 months.

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 investigations will be taken care of by the principal investigator. This study has been reviewed by the Institutional Ethics Committee and you are free to contact the members 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 a 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. Anjana. G

Mobile no: 9487087687

E-mail id: anjanambbs.0207@gmail.com

ರೋಗಿಯ ಮಾಹಿತಿ ನಮೂನೆ

ಅಧ್ಯಯನದ ಶೀರ್ಷಿಕೆ: ಮೌಖಿಕ ಮಿನಾಕ್ಸಿಡಿಲ್ ಜೊತೆಗೆ ಚಿಕಿತ್ಸೆಯಲ್ಲಿ ಓರಲ್ ಡ್ಯೂಟಾಸ್ಟರೈಡ್ ಪುರುಷ ಆಂಡ್ರೊಜೆನೆಟಿಕ್ ಅಲೋಪೆಸಿಯಾ - ಯಾದೃಚ್ಛಿಕ ನಿಯಂತ್ರಣ ಪ್ರಯೋಗ.

ಅಧ್ಯಯನದ ಸ್ಥಳ: ಆರ್.ಎಲ್ ಜಾಲಪ್ಪ ಆಸ್ಪತ್ರೆ, ಟಮಕ, ಕೋಲಾರ

ಉದ್ದೇಶಗಳು:

1.ಕಡಿಮೆ ಡೋಸ್ ನ ಪರಿಣಾಮಕಾರಿತ್ವವನ್ನು ಕಡಿಮೆ ಪ್ರಮಾಣದ ಮೌಖಿಕ ದ್ಯುತಿರಾಸನದೊಂದಿಗೆ ಹೋಲಿಸಲು.

2. ಬಾಯಿಯ ಮಿಕ್ಸಿಡೈಲ್ ಮತ್ತು ಬಾಯಿಯ ಮೂಲಕ ಉಂಟಾಗುವ ಪ್ರತಿಕೂಲ ಪರಿಣಾಮಗಳನ್ನು ದಾಖಲಿಸಲು

ದ್ಯುತಿರಂಧ್ರ.

ಆಂಡ್ರೊಜೆನೆಟಿಕ್ ಅಲೋಪೆಸಿಯಾ ಒಂದು ಆನುವಂಶಿಕ ಪೂರ್ವನಿರ್ಧರಿತ ಅಸ್ವಸ್ಥತೆಯಾಗಿದೆ, ಇದು ಆಂಡ್ರೊಜೆನ್ಸ್ಗೆ ಅತಿಯಾದ ಪ್ರತಿಕ್ರಿಯೆಯಿಂದ.mphl ಪುರುಷರ ಸಾಮಾನ್ಯ ಕೂದಲು ನಷ್ಟ ರೋಗವಾಗಿದೆ. mphl ಎಂಬುದು ಕೂದಲಿನ ಪ್ರಗತಿಪರವಲ್ಲದ ತೆಳುಗೊಳಿಸುವಿಕೆ, ಇದು ರೋಗಿಯ ಜೀವನದ ಮಾನಸಿಕ ಒತ್ತಡಕ್ಕೆ ಕಾರಣವಾಗುತ್ತದೆ.

ಆಂಡ್ರೊಜೆನೆಟಿಕ್ ಅಲೋಪೆಸಿಯಾವನ್ನು ಕ್ಲಿನಿಕಲ್ ಪರೀಕ್ಷೆಯಿಂದ ನಿರ್ಣಯಿಸಬಹುದು. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಆಂಡ್ರೊಜೆನೆಟಿಕ್ ಅಲೋಪೆಸಿಯಾಕ್ಕೆ ಎರಡು ಚಿಕಿತ್ಸಾ ವಿಧಾನಗಳನ್ನು ದಾಖಲಿಸಲಾಗಿದೆ.

ಈ ಅಧ್ಯಯನದಲ್ಲಿ, ಗ್ರೂಪ್ ಎ ಭಾಗವಹಿಸುವವರು - 12 ತಿಂಗಳ ಅವಧಿಗೆ ದಿನಕ್ಕೆ ಒಮ್ಮೆ 5 ಮಿಗ್ರಾಂ ಮೌಖಿಕ ಮಿನೊಕ್ಸಿಡಿಲ್ನೊಂದಿಗೆ ಚಿಕಿತ್ಸೆ ನೀಡಲಾಗುತ್ತದೆ. ಗ್ರೂಪ್ ಬಿ ಭಾಗವಹಿಸುವವರು - 12 ತಿಂಗಳ ಅವಧಿಗೆ ವಾರಕ್ಕೆ ಮೂರು ಬಾರಿ 0.5 ಮಿಗ್ರಾಂ ಮೌಖಿಕ ಡುಟಾಸ್ಟರೈಡ್ ನೊಂದಿಗೆ ಚಿಕಿತ್ಸೆ ನೀಡಲಾಗುತ್ತದೆ.
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 ತನಿಖೆಗಳಿಗೆ 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 ಒಪ್ಪಿಕೊಂಡರೆ ಮಾತ್ರ ನೀವು ಹೆಬ್ಬೆರಳಿನ ಗುರುತನ್ನು ಸಹಿ ಮಾಡುವ ಅಗತ್ಯವಿದೆ.

ಎಡ ಹೆಬ್ಬೆರಳಿನ ಅನಿಸಿಕೆ/ರೋಗಿಯ ಸಹಿ ಎಡ ಹೆಬ್ಬೆರಳಿನ ಅನಿಸಿಕೆ/ಸಾಕ್ಷಿಯ ಸಹಿ
ಯಾವುದೇ ಹೆಚ್ಚಿನ ಸ್ಪಷ್ಟೀಕರಣಕ್ಕಾಗಿ ನೀವು ಅಧ್ಯಯನ ತನಿಖಾಧಿಕಾರಿಯನ್ನು ಸಂಪರ್ಕಿಸಬಹುದು:
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KEY TO MASTER CHART

1.GRP- Group

2.GPA- Global Photographic Assessment scale

Score-0: <25% improvement

Score-1: 25-50% improvement

Score-2: 51-75% improvement

Score-3: >75% improvement

3.BL- Baseline

4.HT- Hypertrichosis

5.PE- Pedal Edema

6.OHT- Orthostatic Hypertension

7.↓libido- decreased libido

8.ED- Erectile dysfunction

MASTER CHART

							GPA Scale					ADVERSE EFFECTS		
SLNO.	AGE	SEX	UHID	GRP	BL	12 WEEKS	24 WEEKS	36 WEEKS	48 WEEKS	HT	PE	OHT	↓LIBIDO	ED
1	28	M	179929	A	0	1	2	2	3	1	0	0	0	0
2	22	M	176298	A	0	1	2	3	3	0	1	0	0	0
3	20	M	180107	B	0	1	2	2	3	0	0	0	0	0
4	19	M	185581	B	0	1	2	3	3	0	0	0	0	0
5	28	M	952701	B	0	1	2	3	3	0	0	0	1	0
6	27	M	186046	A	0	1	2	3	3	1	0	1	0	0
7	23	M	173850	B	0	1	2	2	3	0	0	0	0	0
8	27	M	180107	B	0	1	2	3	3	0	0	0	1	0
9	19	M	133031	B	0	1	2	3	3	0	0	0	0	0
10	30	M	129697	A	0	0	1	2	2	0	0	1	0	0
11	29	M	186391	A	0	1	2	3	3	1	0	0	0	0
12	29	M	136465	B	0	1	2	3	3	0	0	0	1	0
13	28	M	186410	A	0	1	1	2	3	0	1	1	0	0
14	29	M	159921	A	0	1	2	3	3	0	1	0	0	0
15	27	M	186874	B	0	1	2	3	3	0	0	0	0	1
16	20	M	187285	B	0	1	2	2	3	0	0	0	0	0
17	29	M	122829	A	0	1	2	2	2	1	0	0	0	0
18	25	M	187271	A	0	0	1	2	2	0	0	0	0	0
19	29	M	177338	B	0	1	2	2	3	0	0	0	0	1
20	28	M	178370	B	0	1	2	3	3	0	0	0	1	0
21	22	M	188096	A	0	1	2	3	3	0	0	1	0	0
22	20	M	188199	A	0	1	2	2	2	0	0	0	0	0
23	27	M	167017	B	0	1	2	3	3	0	0	0	0	1
24	23	M	188394	B	0	1	2	3	3	0	0	0	0	0

							GPA Scale					ADVERSE EFFECTS		
SLNO.	AGE	SEX	UHID	GRP	BL	12 WEEKS	24 WEEKS	36 WEEKS	48 WEEKS	HT	PE	OHT	↓LIBIDO	ED
25	20	M	184017	B	0	1	2	2	3	0	0	0	0	0
26	30	M	179053	A	0	1	2	3	3	1	0	0	0	0
27	30	M	181234	A	0	1	2	3	3	0	0	0	0	0
28	28	M	183403	B	0	1	2	3	3	0	0	0	1	0
29	25	M	190161	A	0	1	2	3	3	1	0	0	0	0
30	23	M	190270	A	0	0	1	2	2	0	0	1	0	0
31	22	M	190160	A	0	1	2	3	3	0	0	0	0	0
32	21	M	190253	B	0	1	1	2	2	0	0	0	0	0
33	29	M	198615	A	0	0	1	2	2	1	0	0	0	0
34	19	M	190763	B	0	1	2	2	3	0	0	0	0	0
35	26	M	191192	B	0	1	2	3	3	0	0	0	0	0
36	21	M	189136	A	0	1	2	2	3	0	0	0	0	0
37	26	M	134129	B	0	1	2	2	3	0	0	0	1	0
38	30	M	253301	B	0	1	2	2	3	0	0	0	1	0
39	29	M	247744	B	0	1	2	2	2	0	0	0	0	1
40	26	M	247739	B	0	1	2	3	3	0	0	0	0	0
41	27	M	247742	A	0	0	1	1	2	1	0	0	0	0
42	23	M	247842	A	0	1	2	2	3	0	0	0	0	0
43	22	M	247178	A	0	1	1	2	2	0	0	0	0	0
44	20	M	248156	B	0	1	2	2	3	0	0	0	0	0
45	30	M	253356	B	0	1	2	2	3	0	0	0	1	0
46	29	M	253286	B	0	1	2	3	3	0	0	0	0	0
47	28	M	253306	A	0	1	2	2	3	1	0	0	0	0
48	30	M	253342	A	0	1	1	2	2	1	0	0	0	0

							GPA Scale					ADVERSE EFFECTS		
SLNO.	**AGE**	**SEX**	**UHID**	**GRP**	**BL**	**12 WEEKS**	**24 WEEKS**	**36 WEEKS**	**48 WEEKS**	**HT**	**PE**	**OHT**	**↓LIBIDO**	**ED**
49	30	M	253318	B	0	1	2	2	3	0	0	0	0	0
50	21	M	398988	A	0	1	2	3	3	0	0	0	0	0
51	26	M	399269	B	0	1	2	3	3	0	0	0	0	1
52	22	M	399398	B	0	1	2	2	3	0	0	0	0	0
53	30	M	401198	A	0	1	2	3	3	1	0	0	0	0
54	23	M	401491	A	0	1	2	3	3	1	0	0	0	0
55	20	M	402855	A	0	1	2	2	3	0	0	0	0	0
56	21	M	69781	B	0	1	2	3	3	0	0	0	0	0
57	30	M	135566	A	0	0	1	2	2	1	0	0	0	0
58	25	M	130624	B	0	1	2	3	3	0	0	0	0	0
59	22	M	253280	A	0	1	2	2	3	1	0	0	0	0
60	25	M	144820	B	0	1	2	3	3	0	0	0	1	0
61	30	M	253301	A	0	1	2	3	3	0	1	0	0	0
62	26	M	151912	B	0	1	2	3	3	0	0	0	0	1
63	23	M	187275	A	0	1	2	3	3	0	0	1	0	0
64	20	M	170398	A	0	1	2	2	3	0	0	0	0	0