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Androgenetic Alopecia Pathogenesis, Diagnosis, and Treatment

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Abstract
Background: In androgenetic alopecia [AGA], a hereditary disorder of hair loss, the pattern of hair thinning from terminal to indeterminate and finally vellus hair develops with time. The social and emotional lives of those impacted are profoundly influenced. A complex interaction between androgenic stimuli and hereditary susceptibility causes hair follicles to progressively decrease. Our goals are: The goal of this study is to provide readers a bird's-eye perspective of AGA, covering all the bases such as symptoms, causes, diagnostic tools, and treatments available today. Results: AGA may be triggered by a variety of factors and progresses gradually. In order to back up a patient's symptoms and signs that point to a diagnosis, many diagnostic tests may be conducted. Treatment options included surgical treatments, oral and topical medicines, and combination therapy, the latter of which showed encouraging results. The greatest results can only be achieved with continuous therapy and prompt action. More research is required to confirm the safety and effectiveness of newer medications, although they provide new avenues for management, such as cell-based therapies and low-level laser therapy.

Key words : Androgenetic alopecia, finasteride, minoxidil

1. Introduction:

The majority of cases of thinning hair are caused by androgenetic alopecia, which goes by a few different names. Included in this category are FPHL and MPHL, which refer to male and female pattern hair loss, respectively. Half of all women and 80% of all men will have AGA at some time in their lives. Worrying about baldness may have a devastating effect on a person's emotional and physical well-being [1].

In addition to poor self-esteem, a sense of ageing, and a lack of confidence, men with hair loss may feel annoyed by the realisation that they have the disorder. Even while AGA might be considered a physiological process [2], hair restoration techniques nevertheless cost millions of dollars annually.

Hormonal disruption is the root cause of androgenetic alopecia. The gonads create dihydrotestosterone [DHT], a sex hormone. While DHT and testosterone both bind to specific locations, DHT attaches more easily and stays bound for 53 minutes instead of 35. Excessive synthesis of dihydrotestosterone [DHT] shortens the anagen phase and lengthens the telogen phase, resulting in hair thinning and eventual hair loss. In a typical human body, the 5-alpha-reductase enzyme converts as much as 10% of the testosterone to dihydrotestosterone [DHT]. Hair loss may also be caused by inadequate blood supply. because it decreases the scalp's exposure to oxygen and nutrients [3].

The pattern that men exhibit Hamilton-Norwood Measurement: When the frontal hairline starts to recede bitmporally, men start to experience AGA. Diffuse thinning of the scalp over the vertex of the scalp follows. Before long, the bald spot might become completely bald, with just the sides and back of the scalp showing. The varying rates of hair loss in different parts of the scalp are the root cause of pattern baldness. A small percentage of males have frontal hairline retention, whereas the majority experience vertex and frontal hair loss [4].

As a representation of the typical pattern of hair loss, the Norwood-Hamilton scale classifies male pattern baldness into seven distinct groups. Type 1 does not experience hair thinning. Variant 2 has a little receding hairline in the front. Type 3 is considered more prominent and "cosmetically significant" because to the increased frontal loss it encompasses. In the "3 vertex" subgroup of type 3, patients have significant frontal recession in addition to hair loss in the vertex area of the scalp. Type 7 occurs when a large amount of hair is retained by the occipital scalp area alone, after types 4-6, which is characterised by further frontal and vertex hair loss [5].

The centroparietal region shows significant thinning, although the frontal hair line is preserved with FPHL. Even though it mostly affects women, males may also get this form infrequently [6].While females may sometimes have the "male" pattern, the Ludwig pattern is

more common and is characterised by diffuse, gradual hair loss from the crown with preserved frontal hair line.

In the past, the severity of FPHL was determined using the Ludwig scale, which categorises the degree of hair density loss across the crown into three levels.

Level 1: Noticeable crown hair thinning, with a frontal border that is 1 to 3 centimetres below the hairline.

The second grade is characterised by a noticeable rarefaction of the crown hair in the same region as the first grade.

In Grade III, there is complete baldness [denudation] in the same region as in Grades I and II [7].

How AGA develops:

Complexity of genes

It has been shown that there is a racial disparity in the prevalence of age-related baldness and hereditary susceptibility to AGA. The response of hair follicles to circulating androgens might vary in intensity depending on genetic variables. The majority of males with AGA have a hereditary predisposition, and almost all of them have normal blood testosterone levels [8].

Changes in the regulatory sequences of the androgen receptor or changes in the DNA sequence of the gene that encodes the androgen receptor may lead to changes in the concentration or activity of the receptor, which can affect balding on the scalp. These variations may make balding people more vulnerable to DHT, which can cause hair loss to start earlier in life [9]. Another genetic factor linked to male pattern baldness is a variant in the X-chromosome androgen receptor gene [10].

Compared to occipital follicles, frontal hair follicles in AGA patients have elevated levels of the androgen receptor [AR] and the enzyme 5-α reductase. When DHT binds to certain chromosomes on Xq12, it increases AR gene activity in hair follicles, which in turn causes AGA [11].

Effects of Hormones

You can't get alopecia androgenetica [AGA] without androgens. Men in Hamilton's research who underwent castration before to reaching puberty did not have AGA. However, AGA developed in four of the twelve male castrates administered testosterone. There is no evidence that AGA is associated with full androgen insensitivity syndrome, a condition in which the body is unable to express androgen receptors [12].

One symptom of AGA can be a hormonal imbalance, such an imbalance between oestrogen and androgens. Polycystic ovarian syndrome [PCOS] in women is one hormonal imbalance-related illness that sometimes manifests as hair loss, including alopecia [13].

The activity of alpha-reductase type I and type II in the hair follicles of the frontal region is three times higher in males than in women. It is now thought that androgen signalling has a role in male androgenetic alopecia, whereas in females, this is not yet understood [14].

The fact that androgen receptors are located in the dermal papilla instead of the epithelial cells of the hair follicle indicates that this structure is the primary target of androgens. The levels of AR expression in the frontal dermal papilla vary between persons with AGA and those without, according to hormone binding tests. This suggests that increased AR expression in AGA may increase the androgen sensitivity of dermal papilla cells.

After free testosterone is converted into dihydrotestosterone [DHT] by the type II 5-alpha reductase enzyme [5-αR2], DHT attaches to an androgen receptor [AR]. Loss of hair and, eventually, AGA, are symptoms of androgen-sensitive AGA, which is caused by the miniaturisation and sensitivity of hair follicles to DHT.Additionally, environmental factors may have a role in the progression of AGA [15].

Injury Mechanism

Problems with the hair cycle caused by androgenetic alopecia

In male pattern baldness, the terminal hair, which is thick and coloured, progressively changes into little, colourless hair follicles that look like vellus. This transformation is practically invisible. Anagen, the first phase of hair growth, often lasts from two to six years; catagen, the second phase, usually lasts about two to three weeks; and telogen, the third phase, usually lasts about twelve weeks. The typical anagen to telogen ratio for scalp hair is 9:1, however this might vary with the seasons.

As the hair cycle progresses, the anagen phase of AGA becomes shorter. freshly grown anagen hair can only grow as long as the anagen phase lasts, therefore naturally, freshly grown anagen hair is shorter overall. As the percentage of telogen hair grows, the hair becomes more fine and thinning with each cycle. Less hair on the scalp indicates a longer period between hair loss and anagen regeneration [6].

What androgens do to the hair follicle:

In response to androgens, many vellus follicles that create fine, almost colourless hairs undergo a metamorphosis into larger, deeper follicles that generate thicker, longer, and more pigmented hairs. Hair becomes thicker on men's faces, upper pubic diamonds, and chests

when they take androgens, but on both sexes, the axilla and pubis get more hair. But, in other parts of the scalp, they might cause baldness [16]. This can happen even in the same individual.

Find out:

Taking a medical history: In boys with AGA, hair loss often begins after puberty. The hair on top and at the nape of the neck gradually thins down, and there's recession in the front. Most cases of hair loss go unrecognised [17], in contrast to alopecia areata and telogen effluvium.

When diagnosing male pattern baldness, it is important to look for a pattern of progressive hair loss in men that corresponds to the Hamilton-Norwood scale [18].

The benefits of scalp trichoscopy in patients with AGA include improved diagnostics and differential diagnosis, the ability to stage the disease's severity, and the ability to monitor the disease's course and response to therapy [19].

Some examples of what trichoscopic analysis has uncovered are vascular patterns, indications of the hair follicles and their surrounding area, and characteristics of the hair shaft [20].

Simple, little red dermal papilla loops that resemble capillary loops may be discovered during a regular scalp inspection. A perifollicular pigmented network, sometimes called a honeycomb pattern, is highly prized by those with dark complexion. The nodes that make up the network include rete ridge system melanocytes as hyperchromic lines and a small number of melanocytes in the suprapapillary epidermis as hypochromic patches. In a typical scalp, follicular units include one or two vellus hairs inside and two to four terminal hairs, as stated in [22].

More histological findings including follicular ostia and infundibula, as well as a broader variety of hair issues, might be indicated by the yellow dots than was previously thought. The presence of yellow patches is a key trichoscopic characteristic that may differentiate AGA from CTE [23].

Procedure for testing hair thickness:

Additional AGA testing should be conducted using a hair pulling test [24]. What is often known as the "traction test," "Sabouraud's sign," or "pull-out sign" is really very different.

The treatment options for AGA in males include medication, hair transplants, cosmetic aids, or doing nothing and accepting the cosmetic outcome [the "wait and see" approach] [25].

Originally recommended as an antihypertensive medicine, minoxidil was later developed as a topical preparation to promote hair growth once its common adverse effect, hypertrichosis, was recognised [26].

Minoxidil has been used for about 30 years, but no one knows how it works to make hair grow faster [26].

Two different formulations of minoxidil are available for topical use: foam [MF] and liquid [MS]. To dissolve minoxidil and improve its absorption in the tissues, MS comprises two molecules: ethanol and propylene glycol [PG]. Inactive chemicals, such as water, are also included in MS. Scalp baldness is usually treated with formulations containing 2% or 5% minoxidil for patients who are 18 years old or older. Clinicians are able to provide minoxidil to children despite the fact that doing so is considered an off-label usage. Minoxidil has to be administered for a long time to keep the clinical outcomes since these advantages go back when the drug is discontinued [27].

Pharmacokinetics, Metabolism, and Elimination of Minoxidil:

The pharmacologic action of minoxidil is attributed to its sulfated metabolite, making metabolism a crucial process. The majority of minoxidil taken orally is metabolised by the liver by glucuronidation, hydroxylation, and sulfation [28].

Oral minoxidil has a three to four hour elimination half-life, and the kidneys mostly eliminate it in urine twelve to twenty hours after administration. Within four days after stopping topical minoxidil therapy, about all of the drug is flushed out of the body [29].

The work of topical minoxidil involves relaxing blood vessels, reducing inflammation, activating the Wnt/β-catenin signalling pathway, and inhibiting androgens. The duration of the anagen and telogen phases might potentially be influenced by minoxidil. That being said, minoxidil could work in more than one manner [30].In accordance with the vasodilation theory, the vasodilator minoxidil reduces both the systolic and diastolic blood pressure by relaxing the blood vessels. This results in the hair follicles receiving more oxygen-rich blood and nutrients [31].

Reduces swelling

The anti-inflammatory theory states that minoxidil increases hair growth by decreasing perifollicular microinflammation. In an in vitro study, ten healthy adults were asked to participate in which minoxidil was used to suppress T-lymphocytes. The anti-inflammatory effect of minoxidil is further shown in cell culture experiments by its

suppression of two inflammatory mediators, prostacyclin and IL-1α [30].

Signalling inducer of Wnt/β-catenin

The β-catenin signalling pathway that is associated with vascular endothelial growth factors [VEFG] may be activated and VEFG secretion in dermal papilla cells may be enhanced by minoxidil. The cytoplasm becomes enriched with β-catenin via the Wingless-Int [Wnt] pathway, which then acts as a transcriptional factor and promotes hair follicle regeneration [32].

Antagonists against male hormones

It is possible that minoxidil has antiandrogen effects. In human keratinocyte [HaCaT] cells, this trait has been shown to significantly reduce 5a-reductase type 2 gene expression in vitro. the number 33.Proliferation of cells and DNA synthesis

Thanks to minoxidil's ability to speed up DNA synthesis in the anagen bulb, which prompted telogen follicle secondary germ cells to activate [34].

How minoxidil works throughout the anagen and telogen stages

You can get minoxidil to extend the anagen phase, shorten the telogen phase, or do both at once. Using a rat model, we found that telogen phase duration was reduced in rats given 3% or 5% minoxidil solutions, while anagen phase duration was unaffected. Thanks to this, the anagen phase started sooner than expected [30].

The Impact of Minoxidil on Living Things

For a long time, minoxidil has been the go-to medication for thinning hair. The medicine reduces hair loss and increases hair growth by acting on follicular cells. Stopping therapy causes hair loss to worsen after 12–24 weeks [26].

Results showed that both the 2% and 5% MS formulations significantly reduced hair loss and increased hair growth in AGA patients treated with the former, with the latter showing superior results [26].

The drug extended the human anagen interval, as seen by hypertrichosis in untreated regions. According to one experiment, minoxidil lengthened the anagen phase in the dermal papilla [DP] by promoting follicular proliferation and differentiation and inducing β-catenin activity. Under the microscope, both the proportion of anagen follicles and the size of those follicles increased [26].

Perifollicular vascularization is regulated by vascular endothelial growth factor [VEGF], which experiences a dramatic upregulation during the anagen phase and a subsequent downregulation during the catagen and telogen phases. There was a dose-dependent increase in VEFG mRNA expression in response to minoxidil; a six-fold increase was seen upon application of minoxidil. Topical minoxidil also generated hypoxia-inducible factor-1-alpha, an essential component for VEFG synthesis [35].

Furthermore, via activating prostaglandin endoperoxide synthase-1, minoxidil enhanced prostaglandin E2 synthesis while decreasing prostacyclin creation. Minoxidil also enhanced the expression of the prostaglandin E2 receptor, which is the gene primarily targeted by the β-catenin pathway in DP cells. Because of this, the anagen phase of hair follicle development may be prolonged [36].

Dosage of Minoxidil

You may get minoxidil as a topical treatment without a prescription in the US. Each day, take one millilitre as directed. You may skip massaging your scalp after usage. Half of the minoxidil is absorbed within one hour, and three quarters of it is absorbed after four hours. Although further study is needed to determine the importance of this potential link, some practitioners find that using topical minoxidil with microneedling increases its efficacy [37].

There is evidence that 5% minoxidil is more effective than 2% minoxidil in treating alopecia. A more pronounced clinical response to minoxidil is seen in cases when the hair follicles are not drastically reduced and alopecia starts within five years, which is common in young individuals [38].

Minoxidil Side Effects

Although most people have no problems with minoxidil, there are several side effects to be aware of when using it topically: Due to the drug's effect of shortening the telogen phase, minoxidil may cause substantial hair loss in men. Additionally, minoxidil has the potential to induce pulmonary hypertension due to an increase in cardiac output and pulmonary artery pressure. Some other side effects may include frequent pulsating headaches, hypertrichosis, itchy eyes, skin rashes [including bullous eruptions], and polymenorrhea. Redness, soreness, and a burning sensation on the scalp are signs of skin irritation. The condition referred to as "scaly changes of the scalp" may irritate the scalp or worsen seborrhoeic dermatitis. Localised itching appears in the affected regions.Skin redness, inflammation, and itching are signs of allergic contact dermatitis. Allergenic contact dermatitis is most often caused by two primary allergens: minoxidil and propylene. glycol. Patch testing is a useful tool for identifying the root cause. For those who have allergic contact dermatitis due to propylene glycol, a foam

formulation of topical minoxidil might be a suitable alternative [39].

Types of hypertrichosis: localised and generalised Applying orally or topically applied minoxidil may cause hypertrichosis. However, this effect is more prevalent with the 5% topical minoxidil solution and the oral formulation than with the 2% solution. Researchers have found a link between hypertrichosis and using minoxidil to lengthen the anagen phase. Furthermore, hypertrichosis in infants has been associated with unintentional skin-to-skin contact [40].

Metanolone cream

The synthetic 17-lactone steroid spironolactone protects potassium while acting antagonistically on the androgen and progesterone receptors. It is authorised by the US Food and Drug Administration [FDA] to treat hypertension, oedema and ascites, heart failure, and primary hyperaldosteronism. Also, hidradenitis, acne, AGA, and hirsutism are common conditions treated with spironolactone off-label due to its antiandrogenic properties [41].

Reversible and competitively antagonistic to the aldosterone receptor, spironolactone reduces androgen production. This medicine is poorly absorbed when given orally due to the rapid breakdown it undergoes in the liver by its active metabolites, canrenoic acid and 6β-hydroxy 7α-thiomethyl spironolactone [42].

A Review of Spironolactone's Metabolism and Excretion:

Spironolactone is a protein-bound, extensively metabolised, enterohepatic recirculated, and partly absorbed [about 65%] pharmaceutical. Canrenone, 6-beta-hydroxy-7-alphamethylthiospironolactone, and at least seventeen more metabolites are generated during its rapid and broad liver metabolism. Two spironolactone metabolites that have pharmacological activity are 7-alphamethylthiospironolactone. When taken orally, spironolactone takes 2.6 hours to reach its peak concentration, but the active metabolite canrenone takes 4.3 hours. When taken with food, its bioavailability increases to around 95%. The reason why spironolactone's biological effects don't wear off as quickly is that its metabolite, canrenone, has a half-life of 16.5 hours instead of 1.6 hours [43].

The Way Spironolactone Works

Spironolactone often exerts its effects by binding competitively to receptors in the distal convoluted renal tubule, namely at the aldosterone-dependent sodium-potassium exchange site. Because of this, it acts as a mineralocorticoid receptor and an antagonist of aldosterone. Spirolactone, a synthetic steroid, competes with the aldosterone receptor in the cytoplasm. It raises water and sodium secretion and decreases potassium outflow by competing for the aldosterone-sensitive Na+/K+ channel in the distal tubule of the nephron. A reduction in gene expression and synthesis of protein mediator that activates Na+ channels in the apical membrane and a decrease in numbers of Na+/K +ATPase pumps in the basolateral membrane are consequences of spironolactone and eplerenone competing with aldosterone for binding to intracellular receptors. Urine finally excretes around 5% of the filtered Na+ load [44].

The capacity of spironolactone to bind and inhibit androgen receptors is advantageous for female patients suffering from hirsutism and acne, particularly those who have polycystic ovarian syndrome [PCOS] [45].

The fact that it reduces sebum production makes it useful for treating acne vulgaris in women. Due to its anti-androgen properties, it is used off-label to treat female pattern hair loss [FPHL] exclusively. It is believed that oral spironolactone may halt the course of FPHL by reducing blood androgen levels, since it inhibits both the generation of adrenal androgens and the activity of androgens in the hair follicle [46].

Similar local suppression of androgens on hair is achieved with topically administered spironolactone. hair follicles without experiencing the systemic adverse effects of spironolactone taken orally [47].

The Impact of Spironolactone on the Body

Originally used as a potassium-sparing diuretic, spironolactone is now known to be an effective adjunctive therapy for heart failure. benefits people with heart failure and a reduced ejection fraction, as well as those who have had a myocardial infarction, significantly reducing mortality and morbidity. Additionally, it significantly affects those suffering from resistant hypertension [48].

Astoundingly, spirolactone has the potential to treat primary hyperaldosteronism and manage heart failure in both adults and newborns. Refractory oedema, which reduces lung congestion in premature newborns, is another use for this medicine. Hypertension, bronchopulmonary dyspepsia, and furosemide are all treated with thiazide diuretics and spironolactone, respectively [49].

To alleviate hypokalaemia caused by amphotericin or other diuretics, spironolactone is often used as a diuretic in cases of congestive heart failure, ascites, oedema, and nephritic syndrome [44].

Applications of Spironolactone in AGA

To maximise the efficacy of other conventional medications, such as minoxidil, spironolactone is a safe and effective addition to the therapy regimen for AGA. As a safer alternative to oral administration, topical spironolactone is expected to gain popularity as a treatment for individuals who do not have a satisfactory response to minoxidil. This drug is suitable for both males and females. When applied topically, it reduces overall testosterone levels and blocks androgenetic receptors in specific tissues, allowing dermatologists to reduce testosterone's effects on hair and skin [50].

When used daily at dosages of 50-200 mg for a minimum of six months, it has been shown to prevent progression in 90% of instances. It is recommended for long-term use since it does not have any major side effects [51].Spironolactone 1% gel and minoxidil 5% topical are proposed as a new therapy. The recommended dosing schedule is twice day for 12 months, and clinical trials have shown a response rate of up to 100%, suggesting that it may improve medication penetration to the disease's active site [52].

Although it has shown less side effects than systemic spironolactone, more long-term trials are needed to confirm these results and evaluate its safety and effectiveness in a larger group of patients [53].

Spironolactone Side Effects

Patients with impaired renal function, those using potassium supplements, and those receiving concomitant ACE inhibitor treatment are at increased risk of hyperkalaemia, the main adverse effect. Taking the medicine alongside other diuretics increases the risk of hyponatraemia and dehydration. Gynaecomastia, decreased libido, and relative impotence are negative effects that men may suffer if they take excessive amounts. In addition to hyperestrogenemia, women may notice atypical breast soreness and menstrual irregularities [51].

No endocrine systemic side effects are reported in men who take spironolactone topically. The primary metabolite of spironolactone, canrenone, has been tested in the blood during the 72 hours after the ointment's application. Salivary and plasma testosterone levels have also been measured. After 48 hours of applying the lotion, the researchers checked the urine for canrenone levels. During the 72-hour topical treatment period, there were no measurable changes in plasma canrenone levels or any of the other hormones. It seems that topical spironolactone only penetrates the epidermis in a localised manner [54].

Spironolactone has fewer side effects when applied topically, and it reaches the active site more effectively than when taken orally [55].Topical Ketoconazole: AGA treatment options include oral finasteride and shampoo containing ketoconazole. The exact method by which ketaconazole exerts its antiandrogenic effects remains unknown, although it does so by blocking the DHT route. On top to that, it might lessen skin irritation. To confirm its efficacy, clinical studies are required [36].

Topical Azelaic Acid: Acne and other skin disorders are the most prevalent uses for azelaic acid. Researchers in France have been looking at azelaic acid as a possible therapy for AGA since the late 1980s, when they found that it is a strong inhibitor of 5 α- reductase. When combined with zinc and vitamin B6, it decreases 5 α-reductase activity by 90% [56].

Understanding the differences in the sequence of the men's AR gene between balding and non-balding individuals would pave the way for future gene therapy techniques that transfer the non-balding AR gene to hair follicles, thereby stopping hair loss without causing any systemic side effects [57].

The creation of a topical lotion that employs liposomes to transport encapsulated DNA to the hair follicles of mice has expanded this possibilities. This work shows that hair follicles may be safely and selectively targeted to address genes critical to AGA. The lacZ reporter gene was effectively delivered to the hair follicles of mice after a topical application of the gene-encapsulated liposomes [57].

The synthetic azosteroid finasteride inhibits type II 5 α-reductase with a high degree of selectivity and potency. It blocks the conversion of testosterone to dihydrotestosterone [DHT] by binding irreversibly to the enzyme, a property it has as a non-competitive antagonist. Its effectiveness is restricted to the hair follicles on the scalp since it blocks the production of DHT, which is the basis for its usage [58].The vitamin zinc was shown to completely inhibit 5 α-reductase activity in studies conducted in France in the late 1980s, when the amounts of zinc were high enough. Consequently, zinc may be useful for issues related to excess DHT [59].

Tiny needles

The use of microneedling to stimulate hair regrowth in alopecia has recently gained popularity. There has been a shift away from using it for cosmetic reasons and in the direction of progress in the administration of topical drugs. Microneedling involves making small punctures in the skin using a series of needles that are usually linked to a roller. These punctures stimulate the production of

Wnt proteins, which in turn release growth factors and lead to the formation of new blood vessels. When the hair bulge is activated, it releases growth factors that stimulate the hair follicles to produce new hair. Researchers have discovered that Wnt proteins promote hair growth by activating stem cells in the dermal papillae. Combining microneedling with topical medications, such as platelet-rich plasma [PRP] or minoxidil, is common practice. Investigations assessing the efficiency of microneedling in collaboration with.

Weekly microneedling with 5% minoxidil solution vs minoxidil solution alone was the subject of a 12-week trial. Half of the males in each group took minoxidil twice a day. Due to the minor erythema that occurred in the treated region, the microneedling group was given minoxidil 24 hours after the operation. The total number of hairs in a specified region with a diameter of 1 cm was one of the outcomes of the 12-week study. Patients in the treatment group had 91.4 hairs/cm2 [P=.039], much higher than the 22.2 hairs/cm2 seen in the control group. The patients reported no adverse effects after treatment, and the effects lasted for eight months.While some studies have shown inconsistent effects, microneedling seems to improve the absorption of topical medications into the scalp.Upon treating individuals for whom conventional topical treatments have failed, healthcare providers may want to take this into consideration [36].

The impact of low-level light radiation on cells is usually referred to as photobiomodulation [PBM] or low-level light therapy [LLLT]. We don't know how PBM halts or reverses hair loss in either men or women. Several theories attempt to account for the cellular alterations brought forth by LLLT. Increasing blood flow at the dermal papilla is one potential action mechanism, according to one notion [60].

The process of hair transplantation involves removing hair follicles from one area of the scalp and placing them in another, usually the frontal or bald vertex, or occipital region. It is routinely possible to achieve graft survival rates of 90% by making use of modern approaches. Surgery cannot proceed unless there is a sufficient number of healthy occipital hair donors and the hair loss has been stabilised with medicinal treatment [61].

Scalp reduction: Compared to hair transplantation, this procedure is less popular. It entails removing the alopecia-damaged core scalp in order to strengthen the hair-bearing skin. It is sometimes used in conjunction with hair transplantation to provide the best possible cosmetic results. The risks of the surgery have increased with time. There are a few drawbacks to consider when getting a scalp reduction: the possibility of scar widening over time from stretching the surrounding scalp skin, the increased visibility of excision scars for cosmetic reasons, the fact that subsequent individual hair loss is unpredictable, and the need for multiple reductions to effectively address hair loss [62].The use of wigs and camouflage are two non-invasive cosmetic options for men and women experiencing hair loss when medical treatments are not necessary, desired, or advised. In addition, they have the potential to be used as an adjunctive treatment alongside other medical or surgical operations. Lotions, dyed powders, and hair sprays are all part of this category. On top of that, you may utilise wigs, extensions, and hair pieces [63].

Alternatives to AGA therapy are the subject of ongoing research and development, and new therapies including JAKs and PRP injections are among them. There is currently no data on the use of JAKs in AGA, although they are being studied as potential new treatments for alopecia [36].

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