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Androgenic alopecia updates

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Abstract
Androgenic alopecia (AGA) is the predominant kind of hair loss in males, and there has been a rise in the number of documented cases in females as well. This condition is marked by the gradual reduction and noticeable absence of hair at the front of the head. AGA occurs due to a genetic predisposition and an increased sensitivity of hair follicles to androgens. This condition mostly affects men and results in the gradual transformation of thick scalp hair into fine vellus hair. While it is widespread, this condition is not lethal but may have a significant psychological and social effect, particularly on women and younger men. Considerable progress has been achieved in comprehending the epidemiology and pathophysiology of AGA. However, the FDA has only authorized two medications, namely finasteride and minoxidil. Extended use of these medications is a necessary condition for an improved therapy response. Nevertheless, this results in inadequate compliance with treatment and the occurrence of negative consequences due to prolonged use, such as the persistent "post finasteride syndrome" even after discontinuing the medicine. AGA may be treated by many methods, including the administration of medications such as Finasteride, Dutasteride, and Minoxidil, as well as surgical hair transplant and laser therapy. The current therapies for AGA seem to be constrained by many reasons, resulting in significant adverse responses.

Keywords: Androgenic alopecia, scalp hair, FDA

Introduction
Androgenic or androgenetic alopecia (AGA) is the predominant kind of gradual hair loss. AGA is a disorder caused by several genes, which may result in different levels of severity, age at which it starts, and areas of the scalp where hair loss occurs. Male hair loss often affects the temporal and vertex areas, while leaving the occipital region unaffected, resulting in a distinctive "horseshoe" pattern. Women have a widespread decrease in hair thickness throughout the top and front of the head, while still maintaining the hairline at the front [1]. Progressive hair loss occurs when terminal hairs gradually convert into vellus hairs in a consistent manner, resulting in scalp denudation and baldness [2].
The occurrence and frequency of AGA are influenced by age and race. According to the limited prevalence statistics, it is shown that up to 30% of Caucasian males will get AGA by the age of 30, up to 50% by the age of 50, and 80% by the age of 70 [3, 5]. Caucasians are more susceptible to the effects compared to Chinese, Japanese, and African American individuals [5].

Pathophysiology of AGA
Vertex scalp hair loss begins at the core region and spreads outward in a circular pattern. Hair follicle shrinking across the mid frontal scalp results in a hair loss pattern that resembles a Christmas tree [6].

Hair Cycle Dynamics and Androgenic Alopecia
Hair grows back in cycles. Growth, involution, quiescence, and regeneration are the cyclical processes that folicles go through. Spending three to five years in the anagen period is typical. The length of hair that grows out is mostly determined by how long the growth phase lasts, as hair elongation is a constant 1 cm every month. A few weeks after anagen ends, there is an involutional period called catagen. The next stage, known as telogen, is a dormant phase of the hair follicles that lasts around three months [7].

Hair Follicle Miniaturization

AGA is characterized by a gradual reduction in the size of the overall hair follicle structure, which occurs in conjunction with alterations in the hair growth cycle. The dermal papilla, which originates from mesenchyme, is situated in the center of the hair bulb at the base of the follicle. It plays a crucial role in controlling several features of the epithelial follicle and determining the specific type of hair that is generated [8]. Miniaturization and alterations to the hair cycle are likely to occur as a result of androgen-mediated processes, since the dermal papilla is essential for maintaining and controlling hair development. Given the consistent geometric link between dermal papilla and hair matrix sizes, it is reasonable to assume that dermal papilla size dictates hair bulb and shaft production [1]. Figure 1.

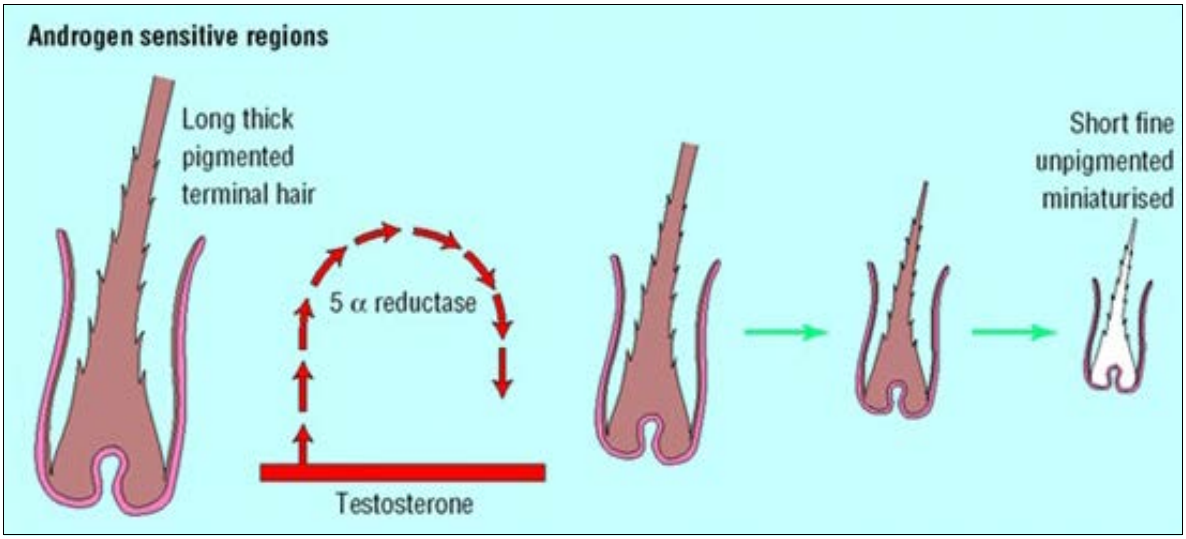


Fig 1: The hair undergoes progressive shrinkage with each cycle [9]

Pattern of Hair Loss

The microscopic pattern of hair loss refers to the precise arrangement in which hair loss takes place inside the follicular units of the scalp. Scalp hairs differ from beard hairs in that they are found in compound follicles, where many hairs develop from a single pore, often ranging from 2 to 5 hairs. The process of miniaturization that takes place inside these follicular units is carefully controlled and leads to a reduction in the number of completely grown hairs per follicular unit. This phenomena may be seen and verified via the use of dermoscopy [10]. The individual experiences this as a decrease in hair density. Baldness is characterized by the presence of a denuded scalp when all the hairs inside a follicular unit have receded [5]. Figure 2.

a) b) c)

Fig 2: Scalp dermoscopic images depicting different stages of alopecia. A: The scalp seems to be normal, with an average of 2-4 hairs in each follicular unit. B: In the early stages of AGA, there is a mix of follicular units containing both multiple and single hairs. C: In the latter stages of AGA, most follicular units consist of thin and solitary hairs [9]

Inflammation

Research indicates that inflammation is present in AGA, however its role in the development of the illness is still a subject of debate. Scalp biopsies have shown the presence of activated T-cells that had infiltrated the lower parts of follicular infundibula [11]. AGA is characterized by a moderate infiltration of lymphohistiocytes around the hair follicles, perhaps accompanied by concentric layers of collagen deposition around the follicles. This is seen in 40% of AGA patients, while only 10% of individuals without AGA show this pattern [12].

Scarring

The irreversibility of hair loss, together with the presence of fibrous tracts and the histological similarities between MAGA and lichen planopilaris (LPP), suggests the potential occurrence of a gradual inflammatory scarring process [13].

Diagnosis of Male and Female Androgenic Alopecia

Diagnosing AGA in males is often straightforward and may be accomplished by obtaining a comprehensive medical history from the patient and doing a thorough examination of their hair and scalp. AGA may be strongly indicated by

the combination of progressive and methodical hair loss, together with evidence of decreasing hair follicles and a negative outcome on a hair pull test during examination [14].

A- Clinical assessment

AGA is characterized by the early manifestation of follicular miniaturization. Miniaturized hairs may be detected by observing the thickness of hair fibers against a backdrop with contrasting colors or by using a portable microscope or dermatoscope. Dermoscopy may detect anisotrichosis, which refers to differences in the diameter of hair follicles, well in advance of hair loss being noticeable in a clinical setting [15]. The miniaturized hairs might vary in length and width due to the independent actions on each individual follicle. The occurrence of disparate hair lengths and texture is indicative of AGA [5].

Men with AGA frequently have a familial history of hair loss. Additional assessment is necessary for sudden hair loss or hair loss followed by itching, burning, or scalp discomfort, since these characteristics are not often linked to AGA [16].

Dermoscopy of the scalp in AGA

Scalp dermoscopy, commonly referred to as trichoscopy, might be valuable for diagnostic purposes.

Features present in all grades of androgenic alopecia [17]

- Hair diameter diversity >20% which corresponds to vellus transformation of hair, this finding is documented in all patients, it is a hallmark of the disease [18].
- **Yellow dots:** round or polycyclic dots which represent empty hair follicles filled with sebum, the frequency of this finding is affected by different skin phenotypes and shampoo habits between cultures.
- **Scalp honeycomb pigmentation:** formed of hypo pigmented areas bordered with hyper pigmented lines, corresponding to melanin of rete ridges.

Features present in early grades of androgenic alopecia [17]

Brown peripilar sign: brown halos around the follicular opening of about 1mm diameter.

Features present in sever and late grades of androgenic alopecia [17]

- **White peripilar sign:** larger white halo around the follicular osteum, it is related to peripilar fibrosis.
- **Focal atrichia:** areas of complete hair loss on scalp, mostly in a size of a pencil eraser, it has appositive correlation with disease severity.

Major and minor trichoscopic criteria for diagnosis of AGA [19]

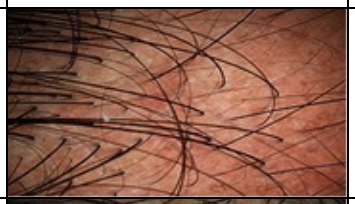
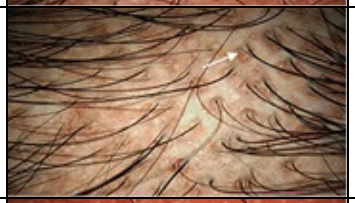
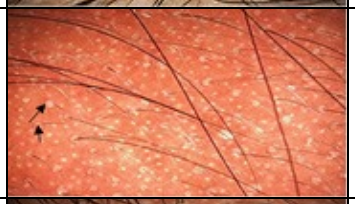
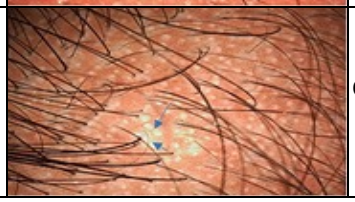
Major criteria are

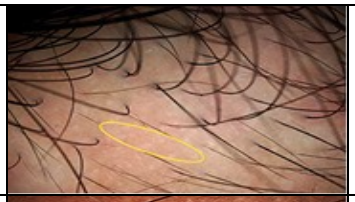
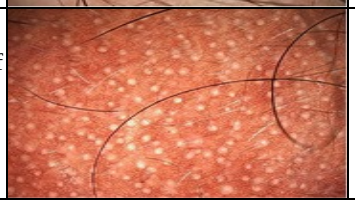
1. There are over 4 yellow dots observed in the frontal area of 4 photos when magnified 70 times.
2. The hair in the frontal area has reduced density in comparison to the hair in the occipital region.
3. Over 10% of vellus hair is present in the frontal area.

Minor criteria are

1. The ratio of the front to back of a single hair unit has increased.
2. Heightened quantity of vellus hair.
3. Discoloration around the hair follicles.
4. The presence of 2 major criteria or 1 major and 2 minor is diagnostic to AGA.

Table 1: Trichoscopic patterns and their clinical correlations [20]

Trichoscopic pattern	Description	Trichoscopic picture Correlate	Clinical correlation
1. Hair shaft thickness heterogenesity	It is the characteristic feature of AGA and corresponds to the change of vellus hair.		Observed in males and females of all age groups with AGA.
2. brown peripilar sign	In the first phases, the presence of a 1mm wide brown halo around the emerging hair shaft is observed.		Initial stages of AGA in males and females
3. White peripilar sign	A white halo is seen at the opening of the hair follicle.		Observed in cases of great severity
4. Yellow dots	In AGA, circular or polycyclic yellow spots are seen, which indicate empty hair follicles that are filled with sebum.		Observed in both initial and advanced stages

5. Focal atrichia	Localized alopecia refers to the occurrence of small, circular areas of complete hair loss on the scalp, often resembling the size of a pencil eraser.		Observed at advanced stages of AGA
6. Scalp honeycomb pigmentation	The hypomelanotic zones are characterized by a lack of pigmentation and are surrounded by hyperchromic lines, which are caused by an excess of melanin in the rete ridges.		Typically seen in all stages of AGA in regions where hair loss has occurred.

B- Histopathology

Histopathologic analysis is often unnecessary for diagnosing male AGA. Nevertheless, because to the characteristic histological characteristics of AGA, biopsies might serve to validate the diagnosis in rare instances when there is uncertainty about the diagnosis [21].

The most recommended method for acquiring a tissue sample for diagnostic purposes is by using a 4 mm punch biopsy. Typically, two biopsies are acquired, one for horizontal (transverse) sectioning and another for vertical sectioning. Performing a horizontal sectioning of the tissue samples enables a clearer view of a greater number of follicles and often produces more valuable outcomes compared to vertical sectioning [22].

Horizontal sections

The histological features of AGA are more readily seen in horizontal segments. The clinical symptoms may vary in intensity and might display a mixture of terminal, vellus, and vellus-like hair follicles in the dermis. The vellus and vellus-like hairs possess a diameter less than 0.03 mm. Terminal hairs, with a diameter above 0.06 mm, are observable inside the reticular dermis [23]. The ratio of anagen to telogen (A: T) hair follicles and terminal to vellus (T: V) hair follicles is altered in AGA and may be readily evaluated using horizontal segments. During AGA, the A to T ratio goes from 12:1 to less than 5:1, whereas the T to V ratio declines from 7:1 to less than 2.5:1 in more severe cases. The alterations in the ratio of terminal to vellus hairs might be quite advantageous in the diagnosis of and AGA [23].

Vertical sections

The vertical sections show terminal hair follicles deeply embedded in the subcutaneous and reticular dermis, whereas vellus hair follicles are positioned nearer to the surface in the papillary dermis. Apparent are vertical columns of connective tissue known as follicular stelae or follicular streamers. The follicular stelae in AGA, found in the deep dermis and providing support to vellus hairs in the upper dermis, might perhaps represent remains of connective tissue that formerly surrounded fully grown hair follicles before they reduced in size [24].

Despite being categorized as a non-inflammatory type of hair loss, histologic specimens from AGA often show inflammation. One-third of biopsies show mild perifollicular lymphohistiocytic inflammation, whereas around 40 percent of specimens exhibit moderate inflammation. This inflammation exhibits notable distinctions from the severe peribulbar inflammation often seen in cases of alopecia areata. Perifollicular fibrosis of a mild kind might also be identified as an extra characteristic. This fibrosis is distinct from the significant scarring that occurs in cicatricial alopecias, which leads to the complete destruction of hair follicles [25].

The clinical diagnosis of FPHL is often established. Scalp biopsies are useful tools for diagnosing conditions when other scalp problems make it difficult to interpret the clinical symptoms, or when the diagnosis is uncertain. When there are clinical indications of hyperandrogenism, laboratory tests are conducted to evaluate its presence [26].

Patient history

The patient's medical history might provide indications that either support or refute a diagnosis of FPHL and help uncover an underlying condition of excessive testosterone levels. The sudden and widespread loss of hair might indicate the presence of acute TE, especially if there has been a recent change in health or medication in the last few months. It is important to mention that TE may occur along with FPHL, and an occurrence of TE can reveal an underlying FPHL condition. Assessing the gynecologic history and looking for symptoms of virilization may assist identify women who should have an examination for hyperandrogenism [27].

FPHL is characterized by the gradual thinning of the ponytail and the increased exposure of the scalp in the frontal and vertex regions of the head. These characteristics are often seen over a period of months to years. While the development and advancement of FPHL often occur gradually, some individuals report experiencing distinct bouts of heightened hair loss before seeing a substantial decrease in the density of their terminal hair. Women may also have a heightened susceptibility to sunburns on the scalp due to less hair covering [28].

Physical examination

It is recommended to do a comprehensive examination of the skin, including the scalp, face, body, and nails, for individuals who report a new issue of hair loss. When examining the scalp and scalp hair, it is important to specifically determine the pattern of hair loss and the thickness of hairs in the affected regions. Indications that align with FPHL include seeing the loss of fully developed hair largely at the front and/or top of the scalp, as well as the presence of miniaturized hairs (shorter, thinner, or vellus hairs). Utilizing a piece of paper as a contrasting backdrop helps enhance the clarity of individual strands of hair [29].

When examining the scalp, it is important to specifically look for signs of inflammation, scarring, or scaling. The

existence of these characteristics clearly indicates that the hair loss is attributed to an alternative ailment or if FPHL is concurrently present with another scalp problem [30].
Examining different areas of the body may lead to the discovery of characteristics that might be helpful in diagnosing and treating the patient. Patients with diffuse alopecia areata (AA) may exhibit nail abnormalities and have patchy hair loss on various areas of the body. These observations are not often seen in cases with FPHL. Furthermore, the existence of excessive hair growth, acne, and obesity indicates the potential occurrence of a hyperandrogenic condition associated with polycystic ovarian syndrome or another hormonal irregularity [31].

Pull test: The pull test is a method used to assess the amount of hair that is lost after a gentle pulling of the scalp hair. This test provides an approximate estimation of the degree of hair loss in everyday clinical practice, although it is subject to significant variation in interpretation among different observers. The user should hold a cluster of about 50 to 60 strands of hair between their thumb, index finger, and middle finger, starting at the root near the scalp [32].
The hair is forcefully pulled away from the scalp as the fingers smoothly glide over the hair shaft. Subsequently, the quantity of extracted hairs is tallied. A positive pull test occurs when more than 10% of the grabbed hairs (specifically, six hairs) are forcibly removed from the scalp, indicating active hair loss. Normal physiologic shedding is characterized by the presence of less than six readily removable hairs [32].
McDonald *et al*. conducted a study to measure the standard values of hair pull test by investigating the impact of pretest hair washing and brushing. They performed the pull test on a total of 181 individuals. The research demonstrated that the acceptable threshold for the hair pull test should be lowered to 2 hairs or less [33].

Standardized wash test
During the standardized wash test, women delay from bathing their hair for a period of 5 days. Afterward, they proceed to shampoo and rinse their hair in a basin, ensuring that the hole is closed with gauze. The hairs that are present in the water and gauze are gathered and sent for analysis. There are 34 hairs that need to be counted and categorized based on their length, either being less than or equal to 3 cm or more than or equal to 5 cm. This approach is crucial for distinguishing between TE and FPHL [32].

Modified wash test
The AGA/TE modified wash test involves counting and categorizing the hairs into three categories according on their length.
1. Long hair is defined as hair that is longer than 5 cm.
2. Intermediate-length hair is defined as hair that is between 3 and 5 cm in length.
3. c- Short vellus hair is defined as hair that is less than 3 cm in length.

Hairs with a length less than 3 cm are classified as telogen vellus hairs. The AGA/TE modified wash test provides the ultimate outcomes in terms of the overall count of telogen hairs and the proportion of telogen vellus hairs [34].

Trichogram: The trichogram is a minimally invasive technique that involves plucking hair and using a microscope to evaluate the hair root and its growth cycle. The trichogram relies on the hair cycle and measures the number of hair follicles in various stages of development. Using a rubber-armed forceps, a total of 60 to 80 hairs are extracted from two specified areas of the scalp, depending on the kind of hair problem being investigated. Hair is extracted by applying a single, swift, vigorous push in a path perpendicular to the scalp, consistently following the natural growth pattern of the hair. Hair bulbs are immediately placed with their roots on a glass slide and analyzed using a magnification lens or low-power microscope to determine the quantity of hairs in the various stages of the hair cycle. The findings are expressed as a percentage relative to the total count of extracted hairs [35].

Dermoscopy/Videodermoscopy
The dermoscopic aspects of FPHL are identical to the dermoscopic features of male AGA. (Figure 3).

Fig 3: Yellow dots in dermoscopy of FPHL patient [36]

Video dermoscopy is a noninvasive method that was originally used to assess pigmented lesions in living tissue, but it has also shown its utility in examining scalp and hair abnormalities in living subjects. This method enables doctors to differentiate between FPHL and acute and chronic TE, particularly during the first phases of the condition [37].
A video microscope is used, which is equipped with a range of objective lenses ranging from × 20 to × 1000. Magnification improves the clarity of the scalp and hair pictures and identifies the hair shaft inside the follicle (if it exists), as well as its dimensions, diameter, and any potential irregularities. All digital photographs may be kept for future scrutiny [37].

Scalp biopsy
Scalp biopsies are often unnecessary for diagnosing FPHL, although they may be useful when the clinical assessment fails to offer a conclusive diagnosis. Cases when a biopsy might be especially advantageous include [38].
- Differentiating FPHL from TE or Diffuse Alopecia Areata.
- To differentiate between FPHL and scarring alopecia when it is difficult to clinically distinguish between alopecia that causes scarring and alopecia that does not cause scarring.
- Identifying concurrent scalp diseases.

Scalp biopsies should be conducted within the region that is affected and should penetrate into the layer of fat under the skin. Avoiding the bitemporal area is advisable, since

follicular shrinkage is often seen in persons without hair loss in this region [39]. Figure 4.

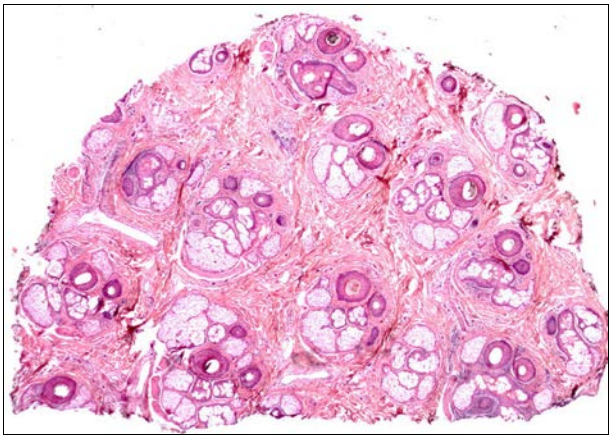


Fig 4: Histopathology of female AGA with increased number of miniaturized (vellus-like) hairs [40]

The typical histopathologic features of FPHL are identical to those of male AGA and they include [41]

- The number of hair follicles has increased in a smaller size, but the sebaceous glands have decreased in size.
- Reduced ratio of adenine (A) to thymine (T).
- There is a higher quantity of follicular stelae.
- There is inflammation surrounding the top part of the hair follicle, either with or without fibrosis around the hair follicle.

Laboratory tests

While serologic testing may not provide a conclusive diagnosis of FPHL, they are useful in detecting an underlying hyperandrogenic condition. Hyperandrogenism testing is conducted in women with FPHL if they exhibit clinical signs that indicate hyperandrogenism, such as hirsutism, irregular menstrual cycles, moderate to severe acne, adult acne that does not respond to therapy, acanthosis nigricans, or galactorrhea [29].

Adjunctive tests

Trichograms and phototrichograms are additional methods that may be used to evaluate and monitor FPHL. A trichogram is a method that involves using a needle holder to pluck 25 to 50 hairs off the scalp. These hairs are then examined at their base to determine the percentage of hairs in the telogen phase. The use of trichograms for diagnosing FPHL has significantly declined [42].

Phototrichograms are imaging methods used to meticulously analyze the characteristics of hair loss for the purpose of diagnosis and subsequent monitoring. A tiny section of hair is cut and then analyzed using imaging techniques to determine the proportions of regrowing (anagen) hairs, resting (telogen) hairs, and shed hairs, as well as the rate of hair growth and hair density. Computerized methods for conducting phototrichograms have been developed, namely TrichoScan and Folliscope. Phototrichograms are mainly employed in clinical research and specialized hair clinics [43].

Differential diagnosis of androgenic alopecia

Diffuse alopecia areata (AA), TE, and fibrosing alopecia with a pattern distribution are the most often encountered hair abnormalities while distinguishing and diagnosing AGA. It is crucial to bear in mind that AGA may concurrently present with these disorders [27].

Diffuse alopecia areata

Scatter Alopecia areata (AA) often has a more sudden beginning compared to AGA, and it normally does not follow a specific pattern of hair loss. Additionally, AA may be accompanied by hair loss on other areas of the body. AA may present with nail pitting, while AGA does not exhibit this characteristic. Both AA and AGA exhibit the presence of miniaturized hairs. A punch biopsy is advantageous in challenging instances [21].

- **Telogen effluvium:** Unlike the predictable hair loss seen in AGA, TE often causes hair loss over the whole scalp, leading to a decrease in hair volume. TE often occurs in response to a sudden incident, and the outcomes of the hair pull test are affirmative. Knowledge of a preceding incident or medication is helpful in understanding the situation. A biopsy may aid in differentiating between AGA and TE [44].
- **Fibrosing alopecia in a pattern distribution (FAPD);** FAPD, or Fibrosing Alopecia Pattern of Scarring, is a newly recognized kind of hair loss characterized by scarring. The characteristic feature of this condition is the reduction in size of hair follicles and the presence of an inflammatory infiltration that affects the isthmus and infundibular area in the early stages. These traits include elements of both AGA and lichen planopilaris (LPP) [45].

Management of androgenic alopecia

Topical treatment of AGA

The Food and Drug Administration has only authorized topical minoxidil and oral finasteride as therapies for AGA. Both drugs inhibit additional hair loss but can only partly restore baldness. Both need consistent use in order to sustain the desired outcome [9].

1. **Minoxidil:** Minoxidil topical preparations are offered in 2% and 5% solutions. Both strengths are presently utilized for treating males, however, the 5% minoxidil solution has exhibited greater effectiveness compared to the 2% solution. The recently developed topical hydroalcoholic foam is free of propylene glycol and can be applied more conveniently to specific areas of interest. Placebo-controlled double-blind trials have proven that the hydroalcoholic foam is effective, safe, and aesthetically pleasing to patients [46].
2. **Fluridil:** It is a specific kind of medication that inhibits the activity of androgen receptors, and it has been intentionally designed for use in MAGA. The design of this substance aims for local metabolism rather than systemic absorption, and it may be broken down into inactive metabolites without causing any systemic anti-androgenic effects [47].
3. **Prostaglandin analogues:** Latanoprost, a prostaglandin analogue, is thought to stimulate hair growth by prolonging the anagen phase of the hair cycle. The use of latanoprost on the skin has been linked to the lengthening of both eyelashes and eyebrows in individuals with glaucoma [48].
4. **Topical Antifungal and Antibiotics:** The exact function of inflammation in the development of AGA is uncertain, and the importance of inflammatory cells at the lower part of the hair follicles is not well

understood. Studies have shown that the use of ketoconazole shampoo directly on the affected area has resulted in a significant increase in hair growth, as compared to a placebo. Ketoconazole shampoo is an effective supplementary therapy that is believed to possess anti-inflammatory and anti-androgenic effects. It may also aid in the treatment of seborrheic dermatitis if it is present [49].

5. **Topical finasteride:** Topical finasteride has been studied as a possible alternative method of administering the medicine. Although the application of a 0.05% finasteride solution to the scalp resulted in a significant absorption rate and a 40% decrease in serum DHT levels, it did not have any impact on hair regrowth. An explanation for this discovery is that the suppression of prostatic dihydrotestosterone (DHT) synthesis plays a crucial role in avoiding hair loss while using finasteride [50].
6. **Caffeine:** Caffeine stimulates the development of human hair follicles by promoting the elongation of hair shafts, lengthening the duration of the anagen phase, and encouraging the proliferation of keratinocytes in both males and females [51].

Systemic treatment for AGA

1. **Finasteride:** Finasteride has been shown to decelerate the advancement of AGA and induce partial regrowth in around two-thirds of male individuals. The weight of hair is influenced by factors such as hair density, hair growth rate, and hair thickness. These studies demonstrate finasteride's capacity to counteract the shrinking process, resulting in longer and thicker hair, maybe with an increased growth rate [52].
2. **Dutasteride:** Dutasteride effectively blocks the activity of both type I and type II 5 alpha reductase enzymes. It has been shown that it is around 3 to 100 times stronger than finasteride in blocking the activity of type I and II 5 alpha reductase enzymes, as indicated by research [53]. Dutasteride has the ability to reduce serum DHT levels by over 90%, whilst finasteride reduces serum DHT levels by 70% [54, 55].
3. **Cyproterone acetate:** It inhibits the androgen receptors and reduces testosterone levels by decreasing the production of luteinizing hormone (LH) and follicle-stimulating hormone (FSH). Additional applications include prostate cancer, hirsutism, and severe acne. It may be taken either on its own or in combination with ethinylestradiol. The regimen consists of administering a dosage of 50mg ethinylestradiol and 2mg cyproterone acetate on days 1-14 of each monthly cycle, for a duration of one year [56].
4. **Spironolactone:** To treat FPHL, a daily dosage of 50-200 mg is administered for a minimum duration of 6 months. Spironolactone may lead to postural hypotension and electrolyte imbalance [56]

Minor surgical procedures for AGA treatment

1. **Mesotherapy:** A recent study found that using a combination of minoxidil, fenastride, biotin, and D-panthenol in mesotherapy demonstrated significant improvement in hair growth when used with other treatments for AGA. [57]
2. **Platelet rich plasma (PRP):** The combination therapy of hormone medication and topical minoxidil 5% is efficacious for AGA. Nevertheless, the use of PRP enhances its effectiveness and reduces the duration required for optimal enhancement [58].
3. **Microneedling:** Furthermore, there is a hypothesis suggesting that MN might potentially facilitate the activation of growth factors, such as platelet-derived growth factor (PDGF), epidermal growth factors (EGF), VEGF, B catenin, Wnt3a, and Wnt10 b, by causing micro injuries [58].

Laser Treatment

Furthermore, laser light has the ability to modify cell metabolism via the process of photo-dissociation, which involves separating inhibitory nitric oxide from cytochrome c oxidase (a component of unit IV in the mitochondrial respiratory chain). This leads to a boost in the generation of ATP and overall cellular activity [59].

Combination therapy: Topical minoxidil used as a premedication in hair transplant surgery has the advantage of stabilizing hair loss, increasing the number of hairs in the anagen phase, and decreasing post-surgical TE. It is recommended to cease the use of minoxidil for a duration of 2 to 3 days before undergoing surgery. This is done to minimize the risk of skin irritation and reduce the chance for intraoperative bleeding caused by vasodilation. Therapeutic sessions should be recommenced within a timeframe of 1 to 2 weeks [60].

Conclusion

AGA is an extremely common disorder affecting both men and women. The treatment approach for AGA includes use of drugs like Finasteride, Dutasteride, Minoxidil, surgical hair transplant and laser therapy. Current available treatment for hair loss seems to be limited by many factors with substantial adverse reactions.

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