RESEARCH
www.commprac.com
ISSN 1462 2815

ADVANCES AND PERSPECTIVES IN UNDERSTANDING FEMALE PATTERN HAIR LOSS: A COMPREHENSIVE REVIEW

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Abstract

Female Pattern Hair Loss (FPHL) is a common condition characterized by progressive hair thinning in genetically predisposed women. The review highlights that FPHL is the most prevalent cause of hair loss in women, affecting a significant proportion of the female population across different age groups. It emphasizes the importance of differentiating between acquired and hereditary types of alopecia, with FPHL falling under the latter category. The etiology of FPHL remains complex and multifactorial, involving hormonal, genetic, and potentially environmental factors. Hormonal variables, particularly androgens like dihydrotestosterone (DHT), play a significant role in the pathophysiology of FPHL. However, the exact mechanisms by which hormonal factors induce follicle miniaturization and shorten the anagen phase are not fully understood. Clinical features of FPHL include progressive thinning of hair, typically affecting the upper parietal scalp and vertex, while maintaining the frontal hairline in many cases. Various classification systems such as the Ludwig scale, Sinclair scale, Olsen scale, and Hamilton-Norwood scale are used to assess the extent and severity of hair loss in FPHL. Diagnosis of FPHL involves thorough medical history, physical examination, and specialized tests such as pull tests, standardized wash tests, trichograms, trichoscopy, and scalp biopsy. Differential diagnosis with other forms of alopecia is crucial for accurate management. Management of FPHL includes topical and oral medications such as minoxidil, 5-α-reductase inhibitors, and antiandrogen drugs. Surgical options like hair restoration surgery may be considered for advanced cases. Psychosocial aspects of FPHL are also addressed, highlighting the significant impact of hair loss on women's self-esteem, body image, and overall quality of life. This review underscores the importance of addressing psychosocial aspects in FPHL management to improve patient outcomes and well-being.

INTRODUCTION

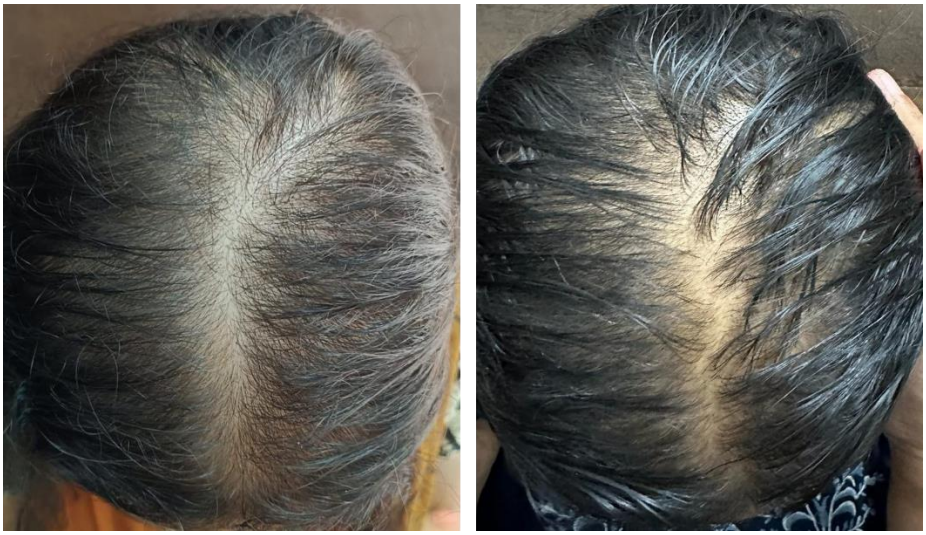
Female pattern hair loss, which is described as non-scarring progressive miniaturization of the hair follicle with a specific pattern distribution, mainly afflicts women [1]. The most prevalent cause of hair loss in women is FPHL. Initial signs and symptoms may appear in adolescence and gradual hair loss in distinct distribution occurs [2]. Since not all women with patterned, nonscarring, central scalp hair loss have a clear link with androgens or inheritance—especially considering the early and late onset subtypes—the name "FPHL" is recommended [3]. By the time they are 30 years old, 12% of young females have alopecia, and by the time they are 60–69 years old, 30–40% of them have alopecia [4].

Women are often less tolerant of hair thinning owing to cultural attitudes, in contrast to men who may embrace hair loss as a natural part of aging. Nowadays, the bulk of consultations constitute women who complain of diffuse hair loss (DHL) at a relatively younger age, taking place in the age range of 30 to 59 [5].

Alopecia may be acquired or inherited. The hereditary types are less common, impact a younger age range, and are caused by inappropriate or non-development of follicles. Diffuse, patterned, and localised hair loss are the general categories for the more prevalent acquired forms, which affect older age groups. Each of these categories is further separated into nonscarring (noncicatricial) and scarring (cicatricial) types [6,7]. Even the smallest amount of hair loss may have a catastrophic effect on a patient's

self-esteem, self-image, and general quality of life, even though alopecia is a benign illness.

Research, like the 2013 study by Zhuang XS et al. [8], has looked at how alopecia affects women's health, specifically in relation to FPHL. It found that the degree of hair loss and how it impacted the quality of life for 125 women with FPHL were significantly correlated. In a similar line, a study including 285 individuals with FPHL or telogen effluvium discovered that personality issues were more common in these women with FPHL, highlighting the significant psychological toll in the form of low self-esteem, and increased stress levels [9,10].



Two female patients with FPHL (decreased hair density over central part)

Brief History and Evolution of Understandıng Female Pattern Hair Loss:

The Greek word ἀλώπηξ (alōpēx) means "fox" in etymology, and this is where the term "alopecia" originates. It is a reference to the animals' ongoing hair loss throughout their lives [11,12].

Since ancient times, the traditional clinical pattern of male baldness has been described. Later, in 1776, Joseph Plenck named the miniaturisation of hair follicles in these situations "calvities" in his work "Doctrina de Morbis Cutaneis" (Vienna, 1776). Additionally, he classified the illness into ten categories based on its aetiology, including febrisequa, puerperarum, morborum exantematicorum, acrimoniosa, phtisicorum, a debilitate nervosa, fenum, hereditaria, a vapore mercurii, and a caufa externa. He also classified the illness into two categories based on its extent, universalem e partialem [13]. But even with modern terminology, the widespread balding process—especially in women—continues to generate misunderstanding [14]. The illness was formerly referred to as "diffuse alopecia in women" in many cases [15,16]. Androgenetic alopecia was used to highlight the genetic and hormonal aspects linked to the progress of the condition after Hamilton's 1942 demonstration of the role of male hormones in the typical progress of baldness in males [17,18]. Female androgenic alopecia, often known as female pattern baldness, is the terminology that has now come to be utilised because of these underlying similarities in genetic and hormonal aspects. The exact prevalence is difficult to ascertain owing to a lack of widely accepted criterion for diagnosis or disease definition for FPHL.

Etiology and pathogenesis:

The pathophysiology of FPHL is still unclear. There is evidence that hormonal, genetic, and potentially environmental variables have a role.

The cycle of the scalp hair follicles assumes a mosaic pattern due to the non-synchronization of the neighbouring units. Three stages are used to categorise it: telogen (resting period), catagen (regression), and anagen (growing phase). The hair sheds (exogenous phase) near the conclusion of the telogen phase and is substituted by a new hair during the early growth stage. The duration of anagen, catagen, and telogen stages is typically 2–8 years, 2–3 weeks, and 3 months, respectively.

Accordingly, eighty to ninety percent of the hair on an adult scalp is in the anagen phase, ten to twenty percent is in the telogen phase, and one to two percent is in the catagen phase, as proven by anatomopathological investigation or trichogram analysis. The length of the anagen phase is shortened and the dermal papilla shrinks in FPHL (thinning of the hair). Miniaturised hairs progressively replace thick, pigmented hairs. In addition, there is a lag between the start of the new anagen phase and the conclusion of the telogen phase. The kenogen phase is the period of rest during which the hair follicle is vacant. In afflicted locations, the capillary density gradually decreases.

These alterations also occur in male pattern alopecia (MPA), despite the fact that FPHL and MPA have distinct patterns in their clinical presentations. The hair follicle is an intricate structure that is always active. The start of the catagen phase may be determined by a disparity between cytokines and growth hormones that maintain the anagen phase and encourage apoptosis.

Follicle regression in the catagen phase is caused by the pervasive apoptosis of the follicular keratinocytes. An important feature in the history of FPHL is the early end of the anagen phase.

Apoptosis may be initiated by two mechanisms: the intrinsic route, triggered by a decrease in growth factors or keratinocyte adhesion, and the extrinsic pathway, which includes the binding of specific molecules to a set of membrane receptors from the family of necrosis factor receptors.

Apoptosis starts in the melanogenic zone during the late anagen phase of the hair follicle, then extends to the array during the premature catagen phase, and ultimately concludes in both the internal and external root sheath.

Different forms of regulation are seen when apoptosis takes place at different places within the hair follicle. The precise mechanism by which cytokines and growth hormones directly control the apoptosis of follicular keratinocytes during this phase remains uncertain.

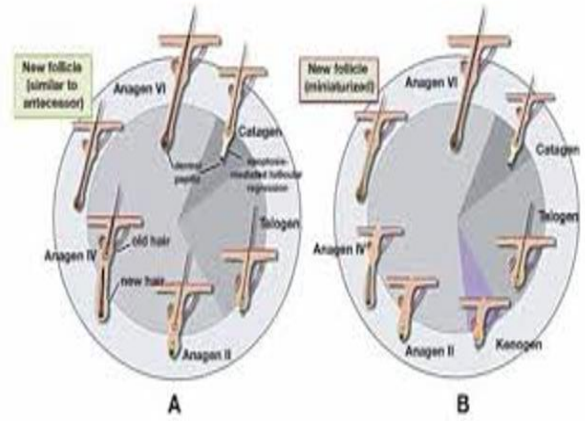
The dimensions of the dermal papilla have a direct impact on the measurement of the follicles. Therefore, the decrease in the number of papillae is the primary factor behind the process of miniaturisation. During the anagen phase, the hair does not experience thinning since it maintains a consistent thickness from the tip to the proximal portion. Miniaturisation is believed to occur during the transition from the catagen phase to the formation of new hair. Efforts to develop therapeutics and preventive measures for FPHL should mainly prioritise the factors that are recognised to induce a decrease in papillae volume.

Although it is not a real vellus hair, the miniaturised hair resembles vellus hair quite a bit. Unlike vellus hair which lacks musculature, the thin miniaturised hair has arrector pili muscle. Apoptosis-induced reduction in papilla cell count is one potential cause of follicle miniaturisation. While papilla fibroblasts may be made to undergo apoptosis in laboratory settings, the papilla is the only part of the follicle that consistently produces substantial amounts of the antiapoptotic protein bcl-2, which theoretically provides resistance against proapoptotic stimuli. One important aspect of the pathophysiology of FPHL that has to be looked into is the failure of antiapoptotic mechanisms [19].

Although hormonal variables have a significant role in the progress of baldness, the processes by which they cause the follicles to miniaturise and the anagen phase to shorten remain unclear.

Hormonal Factors: Male androgenetic alopecia has long been associated with androgens; Hippocrates first recognised this connection, and Hamilton confirmed it in 1942. It is important to recognise the significance of dihydrotestosterone (DHT), a byproduct of testosterone (T), in those who have a genetic predisposition to hair follicle miniaturisation. Owing to its higher affinity for androgen receptors, DHT stimulates the transcription of genes, which modifies tissue architecture. Nonetheless, one effective strategy for treating MPA has been to target type-2 5α-reductase (5αR), the enzyme that converts T to DHT.

The precise function of androgens in female pattern hair loss (FPHL) is still being investigated, despite years of study, and the efficacy of finasteride and dutasteride in treating FPHL has produced mixed findings [20]. The first peak of FPHL is observed in reproductive age group and the second peak post-menopause [21]. Chaikittisilpa et al. reported FPHL prevalence of 52.2% among 178 postmenopausal women which indicates that the disease is closely related to hormonal influences [22]. Jiang et al. described an association of FPHL with polycystic ovarian syndrome and hyperandrogenism [23].



Representative schemes of the hair cycle. A - Normal cycle of the follicle. B - Alterations occurring in baldness: shortening of the anagen phase, increase in the latency period (kenogen phase) and hair follicle minituarization. These alterations may occur together or individually both in FPHL and MPA. *Adapted from Ramos et al, 2015 [19]*

Microinflammation: A microinflammatory process may occur and lymphohistiocytic infiltration in the peri-infundibular area leads to hair follicle miniaturisation. It is not the same as the inflammation linked to scarring alopecia. Skin bacteria, environmental contaminants, and UV radiation are examples of extrinsic variables that might affect the frequency of microinflammation [19,20]. Peyravian et al. noted that inflammation and CD4+ T cells infiltration in hair follicles was responsible for hair cycle changes in both MPA and FPHL [24].

Genetics and Environmental Factors: Twin studies and family history both reflect polygenetic patterns, which indicate a substantial hereditary influence on MPA and FPHL. Ohn et al. described a higher positive family history of patterned alopecia (mainly maternal family history) in FPHL patients compared to healthy individual (25). Dickkopf WNT signaling pathway inhibitor 1 and R-spondin 1 are found to play a role in pathogenesis (26). Environmental variables including stress, hormone swings, and personal lifestyle decisions may also play a role in the development of FPHL. Association of nutrient deficiencies with FPHL is being explored with evidence showing lower serum and tissue VDR levels (27). Detailed investigation has shown particular genetic susceptibility markers for MPA and FPHL, suggesting differentiating variables. To fully understand the complicated relationship between genetics and environmental factors in the development of female pattern hair loss, further study is necessary.

Clinical features:

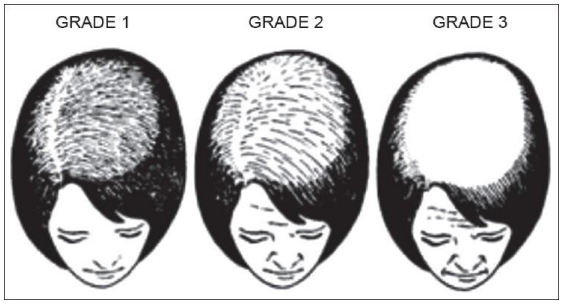
Female pattern hair loss (FPHL) often manifests as a progressive thinning of hair, particularly affecting the upper parietal scalp and vertex. This may or may not be followed by an increase in hair shedding. In contrast to men, the frontal hairline is often maintained, and the degree of miniaturisation is also less pronounced. Conversely, in some females, hair thinning is more widespread and affects the back and sides of the scalp in a diffuse pattern. Female pattern hair loss (FPHL) often presents with three discernible patterns:

The 3-point Ludwig scale and the 5-point Sinclair scale are used to describe the first pattern of diffuse thinning in the crown region while preserving the frontal hairline. The Olsen scale characterises the progressive reduction and widening of the central region of the scalp, accompanied by a receding hairline that resembles the shape of a Christmas tree. This hairstyle has a prominent centre part that extends into a triangular shape at the front hairline, along with overall thinning of the hair.

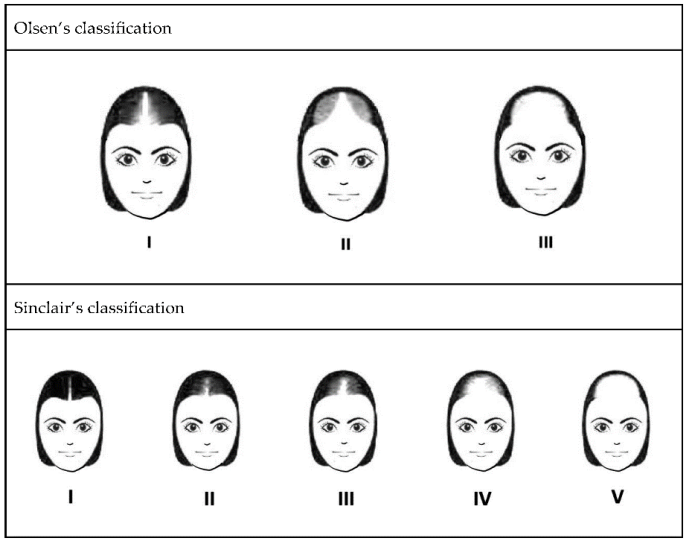
The Hamilton-Norwood scale is used to assess the degree of thinning of the hairline and recession of the temples. In contrast to the neural crest origin of the dermis in the frontal/parietal scalp, these patterns do not affect the occipital area and may be attributed to the mesodermal development of the dermis in the occipital region [28].

Classifications:

The Ludwig classification categorises female pattern hair loss (FPHL) into three distinct stages, as outlined in Ludwig's 1977 classification. Grade 1 is defined by a line that is 1-3 cm behind the frontal hairline and results in significant thinning on the crown. Grade 2 is characterised by a noticeable decrease in hair density on the crown within the Grade 1 region. Grade 3 is characterised by complete baldness within the areas affected by Grades 1 and 2.



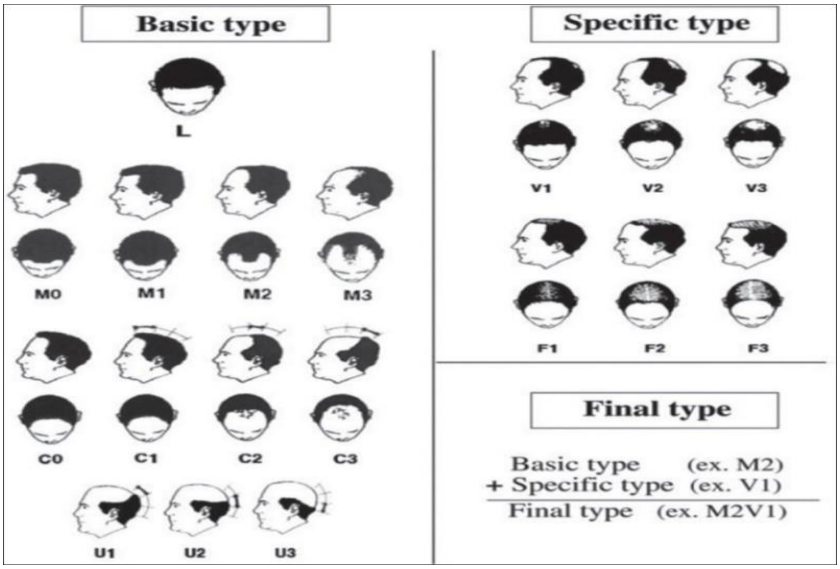
Ludwig classification of FPHL



Olsen classification and Sinclair classification of FPHL

Olsen's classification: Olsen's classification is distinguished by a frontal accentuation with anterior widening that creates a "Christmas tree" pattern near the hairline at the front of the head.

The BASP Classification is a categorization system. The classification system consists of four categories indicated by letters and determines the basic type (BA) according to the form of the anterior hair implantation line. The severity grading scale for early female pattern hair loss (FPHL) developed by Kaneko et al. use a five-level grading system. This approach primarily examines changes in the surface-reflected light of flash in global photographs (GP). The FPHL-SI (FPHL severity index) evaluates four factors: the amount of hair shedding, the density of hair along the midline, the variation in hair diameter, and the change in the proportion of single hairs / hair follicle unit in the frontal and occipital scalps. When grading, factors such as pull test results, midline hair density measured using Sinclair's scale, changes in hair diameter seen in trichoscopy images, and differences in the composition of hair follicular units among the forehead and occipital area are considered [28].



Basic and specific classification for patterned hair loss

Diagnosis:

Every patient at FPHL should have a thorough medical history obtained and a physical examination. This will support the identification and management of further coexisting medical issues [12].

Category	Method
Non-invasive	Questionnaire, daily and 60-s hair counts, standardized and modified wash test, global photographs, dermoscopy, phototrichogram, TrichoScan, polarizing, and surface electron microscopy
Semi-invasive	Trichogram and unit area trichogram
Invasive	Scalp biopsy

Classification of methods to evaluate hair loss. *Adapted from Singal et al, 2013 [46]*

Pull test: If the test results indicate FPHL, it is only noted in the affected areas such as the vertex. The identification of FPHL is highly indicated by the existence of telogen hairs measuring less than 3 cm, which indicate the telogen phase of the miniaturised follicles.

Standardised wash test*:* The standardised wash test distinguishes between FPHL and TE. Less than 100 hairs are lost in FPHL or AGA, with at least 10% of those constituting telogen vellus hairs.

Modified wash test*:* The test, referred to as the androgenetic/telogen effluvium wash test, distinguishes between telogen effluvium (TE) and female pattern hair loss (FPHL). Hair length is used for quantifying the number of hairs and classifying them into three distinct categories: long hair (measuring over 5 cm); intermediate-length hair (measuring over 3 cm but less than 5 cm); and short vellus hairs (measuring less than 3 cm). Telogen vellus hairs have a length of less than 3 cm. The final results are calculated by determining the overall count of telogen hairs and the proportion of telogen vellus hairs.

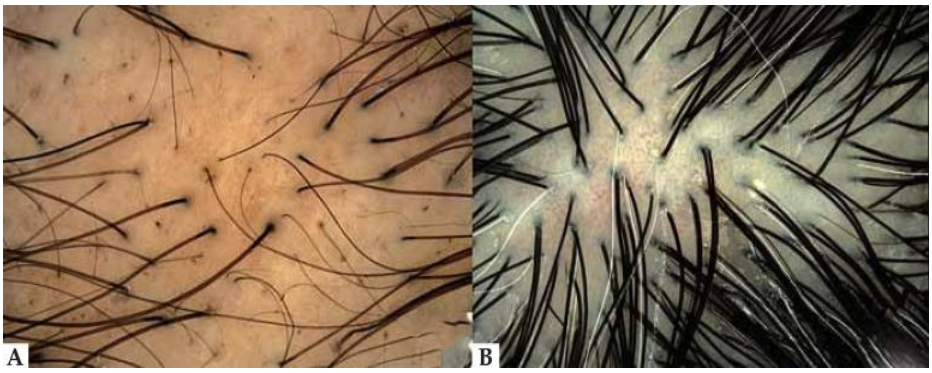
Trichogram: Trichograms are a minimally invasive microscopic tool used to evaluate the hair cycle and hair follicles. By using this method, it is possible to compute the anagen/telogen ratio and determine the quantity of hair follicles present at each stage of the hair cycle. Abnormal trichograms were seen in 62% of individuals with early female pattern hair loss (FPHL) and 84.2% of those with advanced FPHL. The unit area trichogram may be used to sample a set region.

Phototrichogram: By employing a light microscope or a dermoscope, this method enables the capture of magnified images of the scalp. This allows for the evaluation of the density and percentage of anagen and telogen hair in a particular area of the scalp.

Trichoscopy*:* Particularly in the early stages of the illness, dermoscopy may aid in the diagnosis of FPHL. The primary dermoscopic observation is the variation in hair thickness accompanied by a greater quantity of miniaturised hairs, particularly in the frontoparietal area. A hair diameter density more than 20% indicates FPHL. The

presence of short vellus hair on the frontal scalp is a very helpful indicator for FPHL and indicates significant miniaturisation. An additional significant factor is a reduction in the quantity of hairs per follicular unit.

The peripilar sign, which corresponds with the inflammatory infiltration, is a light brown region that is somewhat atrophic surrounding the follicle and often appears in the early stages of FPHL. In the early stages of FPHL, it may be the brown sign; in the later years, it can be the white peripilar sign. In more severe instances, yellow spots may appear, most likely because to keratin and sebum buildup in dilated follicles. Increased hair thinning increases UV light penetration into the scalp and may lead to photoaging-related alterations including the formation of a honeycomb-colored network. When these symptoms are assessed together, FPHL may be identified early. Rakowska standardised the criteria in 2009, based on dermoscopic findings, for the diagnosis of FPHL.

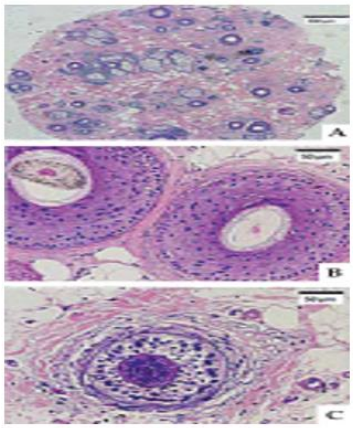


Dermoscopy images of scalp: A) FPHL- variable thickness of the hair shaft, decreased follicle density, incipient pigment network. B) Normal scalp- uniform thickness of hairs, many hairs emerging from single ostium. *Adapted from Ramos et al, 2015* [19]

TrichoScan: TrichosScan is a computerised system that uses epiluminescence microscopy to analyse digital images automatically. Through the use of a mathematical approximation, it allows for the evaluation of hair density and quantity, as well as the ratio of terminal and vellus hairs, and the amount of hairs in telogen and anagen phase.

Scalp biopsy*:* By assessing the terminal:vellus hair ratio, this test is the most dependable method for distinguishing between FPHL and CTE. A ratio less than 4:1 indicates FPHL, whereas a ratio more than 8:1 indicates CTE.

Histopathological examination of FPHL. **a)** Transverse section evidencing wide variability in diameter of the follicles. **b)** terminal follicle in detail. **c)** miniaturized follicle, perifollicular fibrosis and sparse mononuclear inflammatory infiltrate in detail. (HE 40x). *Adapted from Ramos et al, 2015 [19]*



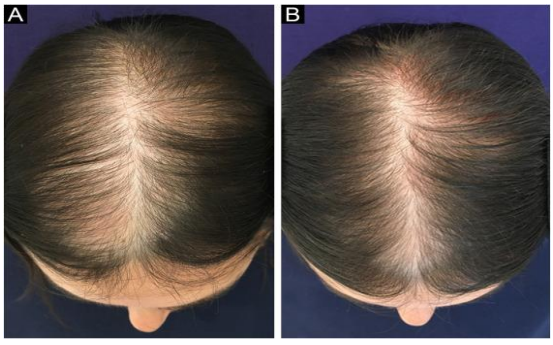
Laboratory investigations: Prolactin levels and the free androgen index are recommended as screening assays by the 2011 European Consensus. The connection [total testosterone (nmol/L)/sex hormone-binding globulin (nmol/L) × 100] between total testosterone and SHBG is known as the FAI. Polycystic ovarian syndrome is indicated by an FAI of 5 or above. SHBG levels change when hormonal contraceptives are used. As a result, laboratory testing needs to be done after atleast a two-month break from hormonal contraception. Evaluation of thyroid function is necessary as thyroid dysfunction may be a factor.

Alopecia Areata Incognito (AAI), Frontal Fibrosing Alopecia (FFA), Permanent Alopecia after Chemotherapy (PAC), and Chronic Telogen Effluvium (CTE) are the most significant illnesses to take into account while making a differential diagnosis for FPHL.

Management And Treatment [29]

I) Topical Minoxidil:

Minoxidil is an ATP-sensitive potassium channel opener and first introduced as a blood pressure medication, but it has since gained popularity as a topical therapy for androgenetic alopecia. In addition to increasing blood flow via its vasodilator qualities, minoxidil stimulates the development of dermal papilla cells, which affects hair growth genetically. A meta-analysis of randomized controlled trials by Van Zuuren et al. noted the proven efficacy of 2% and 5% minoxidil solution and 5% foam for FPHL [30]. The use of a 2% solution in women has been the main focus of clinical study; after 24 weeks of usage, the average number of hairs per square centimetre increased by 12.4 [29]. Adverse effects such as local irritation and increased facial hair growth may occur and alternate formulations that do not contain propylene glycol can be used in these instances. It is imperative to note that an initial increase in hair loss may occur in the first few weeks. However, topical minoxidil remains the leading treatment for female pattern hair loss, and it must be used consistently and indefinitely to achieve long-term results.



Clinical improvement in a FPHL patient on minoxidil 5% solution od. (A) Before treatment. (B) After six months of treatment. *Adapted from Ramos et al, 2015 [19]*

II) Oral Minoxidil:

Although there have been some reported side effects, such as headache, increased body hair growth and transient hair loss, it is generally well-tolerated. A low dose of 0.25 mg is currently used for FPHL patients [31]. But some studies have noted no significant variation in outcome comparing topical (5%) and oral minoxidil (1mg od)

[32]. Comprehensive guidelines for the use of oral minoxidil to treat AGA need further investigation with larger clinical studies.

III) 5- α-reductase inhibitors:

A major factor in the development and progression of androgenetic alopecia (AGA) in both males and females is dihydrotestosterone (DHT), which is a byproduct of testosterone generated by the enzyme 5α-reductase (5αR). Finasteride and dutasteride, two 5αR inhibitors, have shown promise in treating male AGA; however, there is insufficient evidence of their efficacy in treating female pattern hair loss (FPHL). Although finasteride at the recommended daily dosage of 1 mg is known to be successful in treating male AGA, its effects on postmenopausal women have been less than favourable. Higher dosages of finasteride, such as 2.5 mg or 5 mg daily, have been demonstrated in some studies to enhance hair density in women; Limiting factor for use of finasteride in FPHL is the teratogenic potential. Topical finasteride may be an alternative with lesser systemic effects [33,34]. There isn't much evidence to support the use of dutasteride, a very effective 5α-reductase inhibitor, in treating FPHL. This might be because non-hormonal components implicated in FPHL limit the female response to 5α-reductase inhibitors.

IV) Antiandrogen drugs:

Antiandrogen medications, such as spironolactone, cyproterone, flutamide, and bicalutamide, are often used to treat female pattern hair loss (FPHL). These medications are especially useful when there is an excess of androgen in the body. Among them, studies have revealed that spironolactone, a diuretic with antiandrogen qualities, improves hair density [35]. Clinical studies suggest a daily dose of 100–200 mg, with the best response after 1 year or more of treatment [36]. However, it comes with moderate side effects, such as exhaustion and irregular menstrual cycles. Although cyproterone, a GnRH inhibitor and androgen receptor antagonist, has shown effectiveness in treating hyperandrogenism, studies have revealed minoxidil to be more potent. Due to reports of serious liver damage, flutamide, a once-commonly used therapy for FPHL, has been stopped. Nonetheless, bicalutamide has shown some encouraging outcomes in recent retrospective research, suggesting it may be a viable therapy choice for FPHL.

V) Other drugs:

Nutraceuticals, prostaglandin analogues, and alpha estradiol are among other treatments for FPHL. Vitamin D, biotin, caffeine, melatonin, and zinc have shown to be useful, although their efficacy in treating FPHL needs further ananlysis [37-41]. Latanoprost and bimatoprost (PG-F2 analogs) and Setipiprant (PG-D2 receptor inhibitor) have shown some efficacy but limited by their high cost [42]. Botanical preparations like pumpkin seed oil and Leontopodium alpinum extracts and water-soluble peptides from egg yolk (hair growth peptide) have been tried.

Procedures [29]:

VI) Mesotherapy:

The term "mesotherapy" describes a minimally invasive method of injecting small doses of diluted medication intradermally or subcutaneously. Growth factors, panthenol, biotin, finasteride, dutasteride, minoxidil, and steroids are a few examples of these active substances. Although the results of clinical studies have been

inconsistent, they have shown a rise in the proportion of thick to fine hair, but with only modest gains in density. On the other hand, there are issues with pain and a lack of standardisation.

VII) Microneedling:

Rolling pins, stamps, and electric pens are some of the tools used in microneedling, a technique that uses sterile microneedles to make tiny holes in the skin. Although research has shown some advantages for hair growth, there is presently little high-quality data. The best devices and puncture depths are still up for discussion, which emphasises the need for further study to create standardised procedures.

VIII) Microinfusion of Drugs into the Skin (MMP):

Using a specialised tattoo machine, the novel MMP approach stimulates the skin and delivers medicine. As a result, medications are consistently delivered to the dermal layers above. The folding method provides a solution to the often voiced worry over discomfort. Nevertheless, there isn't much research on MMP's effectiveness pertaining to FPHL. Moreover, pharmaceutical actives meant especially for MMP usage now have very little regulatory clearance.

IX) Platelet-Rich Plasma (PRP):

Injections of highly concentrated platelets from the patient's own blood causes release potent growth factors locally. PRP is utilized in treating hair loss related to MPA, FPHL, and alopecia areata. It has been indicated that PRP increases the hair thickness and density in FPHL through systematic reviews and meta-analyses [43,44]. However, there is a lack of standardisation on platelet concentration, blood preparation method, and application technique which needs to be addressed.

X) Botulinum Toxin:

In order to improve blood circulation, reduce tissue DHT, and relax scalp muscles, botulinum toxin has been proposed as a therapy for AGA. Even while some research has hinted at mild improvement in hair density, additional investigation is still required to determine the best application and timing procedures. While promising, more comprehensive and methodical research is needed to determine the efficacy of botulinum toxin.

XI) Photobiomodulation:

The FDA has authorised photobiomodulation, sometimes referred to as low-level laser therapy or LLLT, as a treatment for alopecia. These gadgets employ various light sources and function by promoting the development of fibroblasts and keratinocytes. There is evidence to show that it stimulates the electron transport chain in mitochondria [45]. LLLT has been shown to be well tolerated and may result in both reduced hair loss and enhanced hair density. Nevertheless, there is considerable disagreement over which kind of light is more efficient: LEDs or coherent lasers. LLLT is often considered a backup plan or an alternative for patients who have decided against pursuing clinical or surgical procedures.

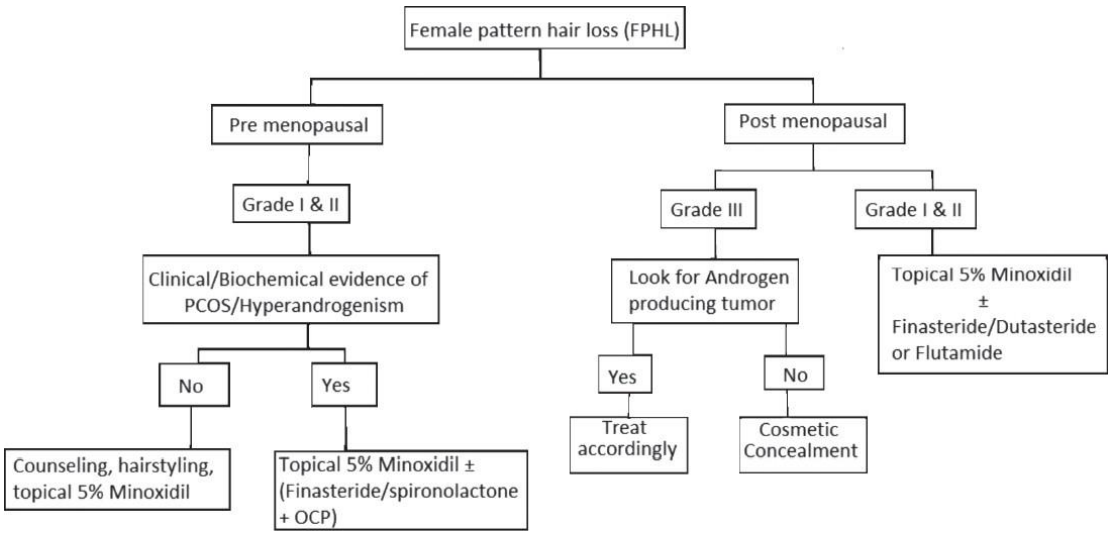
XII) Hair Restoration Surgery (Hair Transplant):

For patients with more complex situations or those who have not responded adequately to previous therapies, hair restoration surgery is often advised. The FUT technique results in a single horizontal scar after the removal of a skin strip. In contrast,

FUE leaves fewer, less obvious scars since it removes follicular units directly. Surgery is not a guaranteed treatment for female pattern hair loss, and this should be made clear in any advertising. When deciding whether to proceed with surgery, it's critical to consider the expectations of the patient, assess the donor site, and set reasonable objectives.

XIII) Camouflage Methods:

Prosthetics aid concealment of hair loss, since they effectively improve psychological well-being. These medically-specific prostheses are available in a variety of fashions, including various hair kinds, base materials, fastening techniques, and extension possibilities. Dermatologists must have a thorough awareness of these distinctive qualities in order to effectively help patients make the best decision.



Algorithm depicting practical approach to management of a case of FPHL.
Adapted from Singal et al, 2013 [46]

Psychosocial Impact:

The psychological and social well-being of women is significantly impacted by FPHL. The extreme value that our culture places on beauty, especially on women, exacerbates this. Comprehending these impacts completely is necessary. Such women more susceptible to issues including poorer self-esteem, less confidence, body image worries and frequently feel inadequate and ugly as a result of the cultural beauty standards that are linked to having a full head of hair. Women with FPHL often struggle with anxiety, despair, a general decline in their quality of life.

Women may experience changes in how they are seen as feminine and beautiful, which might cause them to retreat socially or avoid certain hobbies. Social interactions may be hampered by the fear of stigmatisation or judgement, which can have an impact on both the personal and professional domains.

Importance of Addressing Psychological and Social Aspects in Management:

Recognising the psychological toll that hair loss takes and how it affects treatment compliance is crucial. Providing body image and self-esteem-addressing therapies together with psychological support may make a big difference in the patient's experience. We can boost treatment adherence and eventually enhance clinical results by giving them the appropriate psychosocial care.

Including social and psychological aspects in FPHL treatment aligns with the fundamental principles of patient-centered care. It recognises the complexity of the illness and stresses the need of treating not just the physical symptoms but also the psychological and emotional effects on the person. Healthcare professionals may make a significant contribution to lessening the stigma attached to hair loss by candidly recognising these elements of FPHL. By bringing attention to the psychological domain, we might encourage a more sympathetic and understanding attitude in both the medical community and the general public.

FUTURE DIRECTIONS AND RESEARCH

Although there have been great advances in the research of Female Pattern Hair Loss, there are still areas of uncertainty. Although genetic and hormonal associations are recognised, the exact aetiology and illness processes remain unclear. A deeper understanding of the complex relationships involved is essential for managing FPHL.

Finding the whole spectrum of risk factors linked to it and establishing prediction indicators for susceptibility and progression need further research. Accurate risk assessment and the development of individualised management strategies depend on a thorough knowledge of the contributions made by the environment, lifestyle, and genetics. Further research on the psychological effects of FPHL is also necessary. It is critical to measure the psychological and social repercussions, understand coping strategies, and evaluate the effect on overall quality of life in order to ensure comprehensive treatment.

CONCLUSION

In conclusion, this thorough analysis explores the complex aspects of Female Pattern Hair Loss, including physical manifestations due to interaction of hereditary, hormonal, and environmental variables and the profound psychological repercussions. In the treatment of FPHL, it is critical to acknowledge and treat all these factors thereby encouraging mental and emotional resilience in addition to the patient's bodily well-being.

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