

An Analysis of Several Aspects of Hair Fall

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ABSTRACT: The most prevalent kind of alopecia in women is female pattern hair loss (FPHL). Women who are affected may suffer psychological anguish as well as social impairment. Early detection and therapy are preferable since therapies are more successful at preventing hair loss rather than promoting regrowth. A diagnosis of FPHL may usually be established just by reviewing a patient's medical history and doing a physical examination. A scalp biopsy may be used for diagnostic purposes, although it is seldom necessary. A search for ovarian or adrenal diseases should be done in women who show symptoms of hyperandrogenism. Myths about FPHL treatment obfuscate the situation. The goal of FPHL therapy may be twofold: to reverse or stabilize the shrinkage of hair follicles. In many instances, women with mild-to-moderate FPHL may benefit from oral antiandrogen treatments (cyproterone acetate and spironolactone) and/or topical minoxidil. In most individuals with mild-to-moderate FPHL, current medicinal therapies may stop the illness from progressing and reverse miniaturization if taken properly. In certain instances of severe FPHL, hair systems and surgery may be explored.

KEYWORDS: Fall, Growth, Hair, Loss, Symptom.

1. INTRODUCTION

Due to the obvious ambiguous connection between androgens and this entity, female pattern hair loss (FPHL) has evolved as the preferred name for androgenetic alopecia (AGA) in women. The most prevalent hair loss condition in women is FPHL. Initial signs and symptoms may appear throughout adolescence, leading to gradual hair loss with a distinct pattern distribution. FPHL is a non-scarring diffuse alopecia that develops when hair follicles shrink and the quantity of hairs decreases, particularly in the central, frontal, and parietal scalp areas. There are three major clinical symptoms of FPHL. The initial symptom is widespread thinning of the upper biparietal and vertex areas, with the anterior hair implantation line remaining intact. There are many hair loss scales that try to classify FPHL, each with its own set of benefits and drawbacks[1].

Thinning of the upper bitemporal region and vertex with frontal accentuation that forms a triangle or Christmas tree shape with hair loss in a triangular shape in the frontal-vertical area is another symptom. A third symptom is a severe recession of the frontal-temporal hairline and genuine vertex baldness, which is most often observed in males but may also be seen in women. There are many therapy options for FPHL, but each one takes a long time to show a significant improvement. As a result, a significant amount of time should always be spent on doctor-patient counseling in order to promote compliance[2].

1.1 Epidemiology:

The prevalence of FPHL varies by demographic group and often rises with age. However, the absence of generally recognized illness definitions makes it difficult to compare prevalence across research. Approximately 6 percent to 38 percent of healthy women have some degree of frontal and/or frontal-parietal hair loss. FPHL manifests itself throughout the reproductive years, which is later in women than in males. By the age of 29, 12% of women have clinically detectable FPHL, 25% by the age of 49, 41% by the age of 69, and > 50% have some form of FPHL by the age of 79. During puberty, more severe instances of the illness are seldom reported. Nonetheless, individuals aged 25 to 40 years old have a higher need for therapy. In the United Kingdom, FPHL affects 6% of women under the age of 30 and 42% of women over the age of 70. Only 43% of women over the age of 80 had no indication of FPHL.

1.2 Pathophysiology:

Although FPHL and male AGA have a similar end route that leads to follicular regression, recent evidence indicates that the pathogenesis is not the same in both sexes. Although the function of androgens in the pathogenesis of male hair loss is well understood, the involvement of androgens in the pathogenesis of FPHL is less apparent. In fact, even in the absence of androgens, FPHL may develop. Other nonandrogenic

variables, which are presently unknown, are likely to have a role in the etiology of FPHL. As a result, the role of these genes in the etiopathogenesis of FPHL cannot be ruled out entirely[3].

A genetic tendency may be implicated in women with FPHL who do not have high testosterone levels. Normal amounts of circulating androgen may act on follicular target cells, which are particularly sensitized by attaching to specific intracellular androgen receptors, according to this hereditary propensity. In certain instances, the formation of FPHL may be aided by an androgen-independent process.

Hair loss in women is polygenic and complex, with environmental factors also playing a role. Several research have looked at the significance of a number of genes linked to alopecia. FPHL includes the gradual shrinkage of hair follicles and the eventual conversion of terminal follicles to vellus-like follicles. Because the anagen phase is reduced in these vellus-like follicles, the hair cycle is shortened, resulting in the creation of short and fine hair shafts. Women's shrinking is not consistent and severe like men's, therefore there are no full regions of baldness unless in very rare instances. Furthermore, a mild-to-moderate lymphohistiocytic infiltrative infiltration in the peri-infundibular area may accompany the shrinkage process. To distinguish this infiltration from the inflammation that occurs in scarring alopecia, the term "microinflammation" has been employed[4].

1.3 Comorbidities:

Polycystic ovarian syndrome is the most frequent endocrinologic comorbidity linked with FPHL. Obesity, insulin resistance, hypertension, hyperprolactinemia, and elevated aldosterone levels are all symptoms of metabolic syndrome, which has been linked to FPHL. There has also been a link to an increased risk of carotid and coronary artery disease. Systematic investigations in larger population-based samples are required to better define the comorbidity profile of FPHL. The existence of a link between ferritin levels and FPHL is debatable. Individuals with FPHL had lower ferritin levels than controls in certain trials, and antiandrogen treatment seems to function better in patients with ferritin levels > 40 g/l[5].

1.4 Diagnosis:

Women who have increased hair shedding but little or no loss of hair volume across the mid-frontal scalp may be suffering from a variety of illnesses, including acute and chronic telogen effluvium (TE). To obtain the proper diagnosis, you'll need an anamnesis and a physical examination. Anamnesis should concentrate on when the hair loss began, whether it was gradual or included clumps of hair, and any physical, mental, or emotional stresses that happened in the preceding 3 to 6 months. Detecting indications of hyperandrogenism such as hirsutism, ovarian abnormalities, menstrual irregularities, acne, and infertility should be the goal of a history and physical examination. In women with FPHL but no symptoms of hyperandrogenism, laboratory test findings are seldom examined[6].

Furthermore, hair loss may occur in individuals who are using oral contraceptive medicines containing progesterone with a high androgenic potential, such as norethindrone, or who have just stopped taking an estrogenic oral contraceptive pill. The occipital area, which will reveal a widening of the center portion with a widespread decrease in hair density across the frontal scalp rather than baldness per se, should be evaluated during a physical examination. Although these regions have the most noticeable loss of hair density, evidence of a worldwide loss of hair density can typically be seen across the whole scalp[7].

1.5 Pull test:

The quantity of hairs lost following a little tension on the scalp hair is measured in the pull test. With a high interobserver variability, this test may be used to assess the degree of hair loss in everyday practice. From the root close to the scalp, a bundle of 50 to 60 hairs is held between the thumb, index finger, and middle finger. As the fingers glide down the hair shaft, the hair is firmly but not forcefully pulled away from the scalp. The number of removed hairs is then counted. The pull test is affirmative if more than 10% of the gripped hair (six strands) is pulled away from the scalp. This indicates active hair loss. Normal physiologic shedding is defined as less than six hairs that are readily taken out.

1.6 Standardized wash test:

The standardized wash test requires women to go without washing for 5 days before shampooing and rinsing their hair in a basin with the hole covered with gauze. All hairs left in the water and gauze are

collected and submitted to be examined. A total of 34 hairs must be counted and sorted into three length categories of 3 cm and 5 cm. This is a crucial method for distinguishing TE from FPHL[8].

1.7 Trichogram:

The trichogram is a microscopic technique for evaluating hair root and cycle that is semi-invasive (plucking). The trichogram is based on the hair cycle and measures the growth stages of hair follicles. Depending on the hair condition, 60 to 80 hairs are plucked using a rubber-armed forceps at two particular scalp sites. Hair is pulled out in one swift, powerful motion perpendicular to the scalp and always in the direction of hair development. Hair bulbs are immediately implanted with their roots on a glass slide and analyzed with a magnification lens or a low-power microscope to determine the quantity of hairs in each phase of the hair cycle. The proportion of the total number of plucked hairs is used to calculate the results[9].

1.8 Videodermoscopy:

Videodermoscopy is a noninvasive method that was originally developed to evaluate pigmented lesions in vivo but has now proved to be an effective tool for studying scalp and hair problems in vivo. This method enables doctors to tell the difference between FPHL and acute and chronic TE, particularly in the early phases of the illness. A videomicroscope with a variety of objective lenses is used. The magnification improves pictures of the scalp and hair, allowing for the detection of the hair shaft in the follicle (if present), as well as its length, diameter, and any abnormalities. All digital pictures may be saved for later use.

1.9 Treatment:

As FPHL may resemble and frequently runs simultaneously with other illnesses, the patient should have a thorough medical history review and physical examination. Different concomitant conditions should be explored and treated if relevant, using other diagnostic instruments and methods. The treatment options for FPHL may be divided into two categories: Drugs that are applied topically and those that are administered systemically. Because FPHL is a biological condition characterized by a genetically mediated sensitivity to androgens, most of these medicines target androgen activity by changing androgen synthesis, transport, or metabolism, or by blocking androgen binding to androgenic receptors. Furthermore, androgen-dependent medicines are prohibited in pregnant women because they may induce abnormalities in the genitalia of the male baby. As a result of this discovery, several doctors advise using an oral contraceptive throughout the whole treatment period[10].

1.10 Prostaglandin analog treatments:

When the development of eye lashes was discovered as a side effect, latanoprost and bimatoprost were created for ocular glaucoma. In fact, the PG-F2 analog therapies latanoprost and bimatoprost are known to promote hair growth by extending the anagen phase among prostaglandins (PG). A small placebo-controlled study in males with moderate AGA found that 0.1 percent latanoprost improved hair density and pigmentation substantially after 24 weeks when compared to baseline and the placebo-treated site. Despite this, just 16 male patients were involved in the research, and the drug was only administered to a tiny region of the scalp. However, a case study of postmenopausal women with FPHL failed to show that 0.03 percent bimatoprost injected locally for 16 weeks was effective. Other investigations have shown that an elevated PG-D2 level is linked to hair follicle shrinkage, and that topical administration of PG-D2 also inhibits hair growth. Other medicines that may block the PG-D2 receptor (GPR44), which has an inhibitory impact on hair development and is known to be increased in the scalp of AGA patients, are being researched in recent studies. In a clinical study for asthma, setipiprant (KITH-105) is an orally given GPR44 receptor inhibitor that may have a potential use for individuals with AGA.

1.11 Ketoconazole:

AGA's pathobiology isn't fully understood. Androgen influence and genetic predisposition have been found to have a role in AGA. These variables, however, do not account for the existence of a large lymphoid infiltration on the infra-infundibulum and isthmus of transitional hair follicles. Exacerbation of AGA hair state following intercurrent episodes of various inflammatory dermatoses, particularly seborrheic dermatitis, confirms the detrimental impact of inflammation on AGA hair status.

Ketoconazole (KCZ) is a prescription-strength antibiotic used to treat dandruff and seborrheic dermatitis. KCZ is an anti-inflammatory agent that also functions as an androgen receptor antagonist. The effectiveness

of topical KCZ may be explained by these factors. The presence of certain members of the microflora that usually characterizes seborrheic dermatitis may possibly be linked to the inflammation that abuts on the AGA hair follicles. As a result, KCZ promotes hair health by decreasing AGA inflammation. 2 percent ketoconazole shampoo was proven to be beneficial in the treatment of FPHL with hyperandrogenism.

1.12 *Melatonin:*

Melatonin is a neurohormone produced by the pineal gland that controls many physiological processes such as seasonal biorhythms and daily sleep-wake cycles, all of which affect the aging process. Melatonin is known for its anti-apoptotic and anti-inflammatory characteristics, as well as its capacity to aggressively trap free radicals.

Many neurohormones, neuropeptides, neurotrophins, and neurotransmitters target the hair follicle, which also generates many of these chemicals. As a result, the pilosebaceous unit is most often referred to as a neuroendocrine organ. Melatonin regulates hair development, pigmentation, and molting in a variety of animals, including humans. In a controlled research, topical administration of melatonin 0.1 percent solution substantially increased anagen hair in male and female AGA with excellent compliance.

1.13 *Micro-needling:*

Micro-needling is a minimally invasive dermatological treatment in which tiny needles pierce the stratum corneum by rolling them over the skin. Micro-needling initiates a wound healing cascade with little damage to the epidermis, resulting in collagen creation, neovascularization, and growth factor synthesis in the treated regions, thanks to the physical stress caused by needle penetration. Micro-needling has showed promise in the treatment of atrophic scars, AGA, alopecia areata, and pigmentation disorders including melasma as an adjuvant therapy for improved medication delivery.

Micro-needling has been effectively used with other hair-growth boosting treatments such as minoxidil, platelet-rich plasma, and topical steroid medicines, despite the fact that there are only a few studies that have looked at this technique in the context of hair loss. The penetration of such first-line medicines via micro-needling may be aided, and this is one mechanism that stimulates hair growth. Micro-needling therapy has shown the greatest outcomes in AGA to far. Indeed, micro-needling has the potential to improve hair growth, particularly when used in conjunction with other methods.

1.14 *Hair transplantation:*

Hair transplantation is a viable option for individuals with FPHL over the age of 25 who have not had success with medicinal treatments and whose hair loss has stabilized. The relocation or transfer of hairs from the occipital to the bald region is known as hair transplanting. Women with significant hair density at the donor site over the occipital scalp and severe hair loss or thinning of the frontal scalp are ideal surgical candidates for hair transplantation. Follicular unit hair transplantation is a baldness surgical therapy in which follicular units of hair are dissected under a stereomicroscope and implanted into the bald region for a natural appearance. The operation is done under local anaesthetic and includes the transplanting of 800 to 1200 grafts in one session. The procedure has become considerably less intrusive because to the use of follicular unit transplantation, which provides natural, undetectable, and repeatable outcomes.

2. DISCUSSION

Hair loss is a very frequent problem. It may be caused by a number of inherited or acquired diseases. The capacity of treating doctors to comprehend the etiological aspects as well as the distinctions between various diseases aids in the proper diagnosis and therapy. To arrive at a diagnosis, a comprehensive clinical examination of the patient with hair loss is required. Physicians must stay up to speed on new therapeutic developments. The emphasis of this review article will be on the most frequent acquired reasons of hair loss. It will provide general practitioners a fundamental understanding of hair loss and baldness, as well as how to treat it. Hair loss and baldness are not difficult to diagnose. The importance of collecting a history and doing a scalp examination in the early stages of diagnosis and treatment cannot be overstated. Acute hair loss may be a sign of telogen effluvium, while persistent hair loss could be a sign of androgenetic alopecia.

3. CONCLUSION

Despite the significant incidence of FPHL, dermatologists' clinical practice is still hampered by its treatment. The research must be enhanced in order to discover possible pathogenic factors (other than genetic and hormonal) in FPHL. These discoveries will aid in the development of new and more effective treatments to prevent and reverse the progression of the illness. New data on the epidemiology, genetics, and pathophysiology of FPHL may assist to enhance the quality of life of people afflicted by this illness, since the response to therapy is a problem for dermatologists. The goal of FPHL therapy may be twofold: to reverse or stabilize the shrinkage of hair follicles. In many instances, women with mild-to-moderate FPHL may benefit from oral anti-androgen treatments (cyproterone acetate and spironolactone) and/or topical minoxidil. In most individuals with mild-to-moderate FPHL, current medicinal therapies may stop the illness from progressing and reverse miniaturization if taken properly. In certain instances of severe FPHL, hair systems and surgery may be explored.

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