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President's Message

From the desk of Dr. David H. Kingsley – President, World Trichology Society

Welcome to the inaugural edition of the Times of Trichology.

I am delighted and honoured to be asked to be Editor-in-Chief as trichology has been so important to me for all my working life. I became a certified trichologist in London in 1980 and have practiced ever since.

I subsequently moved to New York where I run a busy trichology clinic. After receiving my doctorate in hair-loss research, I was instrumental in establishing the World Trichology Society as an organization whose mission is to help promote excellence in educational standards for student-trichologists worldwide, as well as continuing education for certified trichologists.

The publishers of the Times of Trichology shared my vision in bringing the most current trichological research and information to specialists in the hair-loss field, and so I was thrilled to associate myself with the publication of this journal as a way to help continue the World Trichology Society's educational goal.

Enjoy!

Dr. David H. Kingsley
President, World Trichology Society

Efficacy of liposomal caffeine for improving hair growth and hair density in alopecia

Dr Narendra Patwardhan
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Introduction

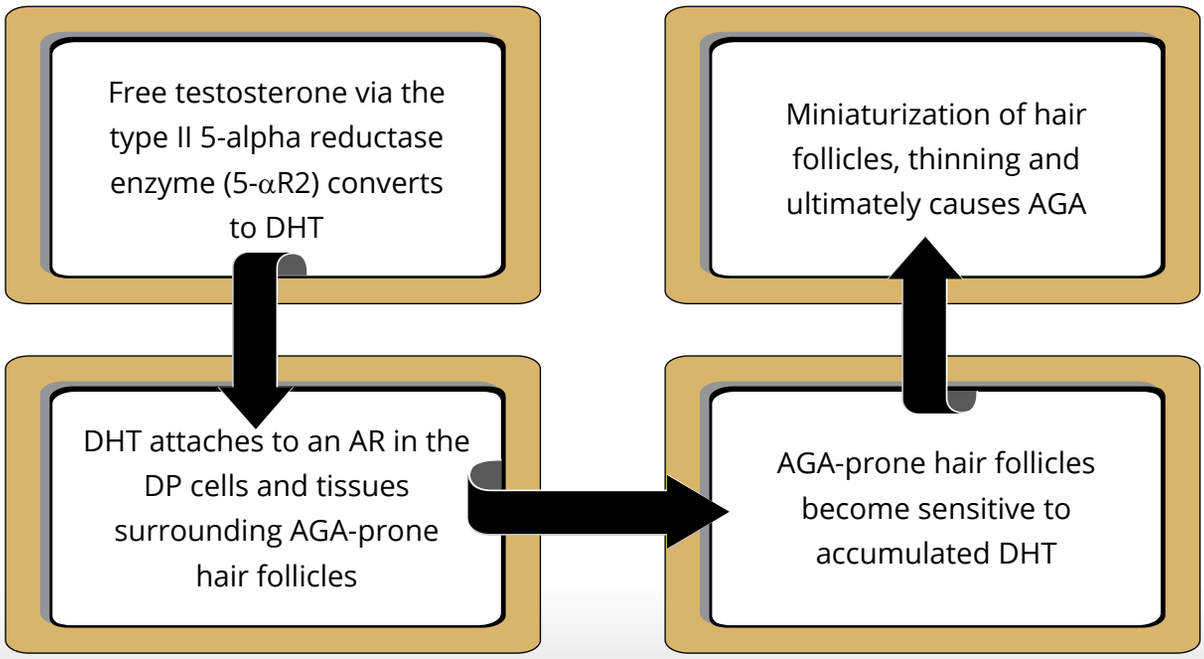
Androgenetic alopecia (AGA) is considered to be the most common type of baldness characterized by progressive hair loss. It occurs with a progressive conversion of terminal hair into indeterminate hair and vellus hair.[1] Hair loss is common among those over 50 years of age, where 95% of hair loss is attributed to AGA.[2]

In women suffering from hair loss, part width increases progressively, most prominently anteriorly, and demonstrates thinning rather than baldness. Temporal recession occurs to a lesser degree in women as compared to men.[3] Due to the high prevalence of alopecia, several methods such as topical, oral, implants treatments, etc have been used to restore hair loss.[1]

Strategic approach to slow hair loss

Androgenic pathway is the first target to slow hair loss. The objective is to slow the production of dihydrotestosterone (DHT) by 5α -reductase. This metabolite has a higher affinity for androgen receptors in the dermal papilla than testosterone. DHT acts by causing follicular atrophy, and through a pro-apoptotic mechanism via caspase according to a recently advanced hypothesis.[4] AGA results from androgen-dependent miniaturization of the scalp hair follicle, with DHT being a major cause as shown in Figure 1.[2]

Figure 1. Mechanism of androgen-dependent miniaturization of the hair follicle[2]



AR: androgen receptor; DP: dermal papilla; AGA: androgenetic alopecia; DHT: dihydrotestosterone.

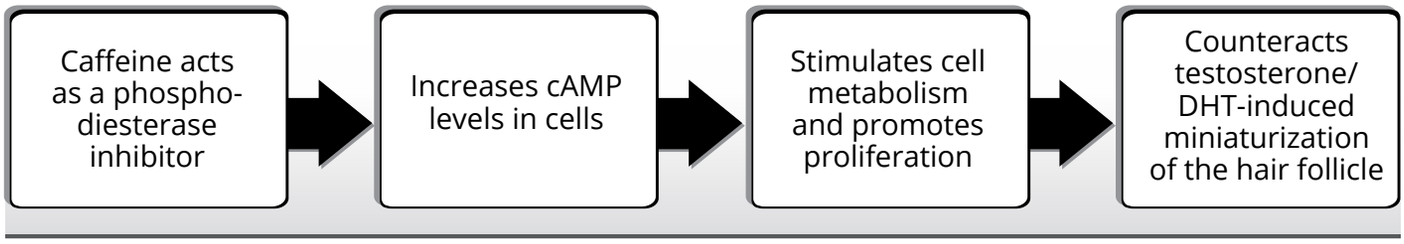
Role of Caffeine in hair growth

Adding caffeine has been shown to counteract the suppressive effect of testosterone on hair growth. Advances have shown that caffeine has beneficial effects in patients suffering from hair loss, both genetic as well as hormonal. The proposed mechanism of caffeine is elaborated in Figure 2.[5]

Benefits of topical caffeine application

- Multiple beneficial effects of topical application of caffeine in AGA are thus attributed to:[5]
  - » Inhibition of phosphodiesterase
  - » Improvement in barrier function
  - » Follicular penetration
  - » Stimulation and promotion of hair growth
- Hence, caffeine may be useful adjuvant in the management of AGA.
- Caffeine has also been shown to prolong anagen duration and stimulated hair matrix and outer root sheath keratinocyte proliferation.
- In vitro study has shown that caffeine helps transforming growth factor-β2/ins-like growth factor-1-mediated regulation of the hair cycle in hair follicles of both men and women, which is its new growth-promoting effect at different levels (molecular, cellular, and organ).[6]

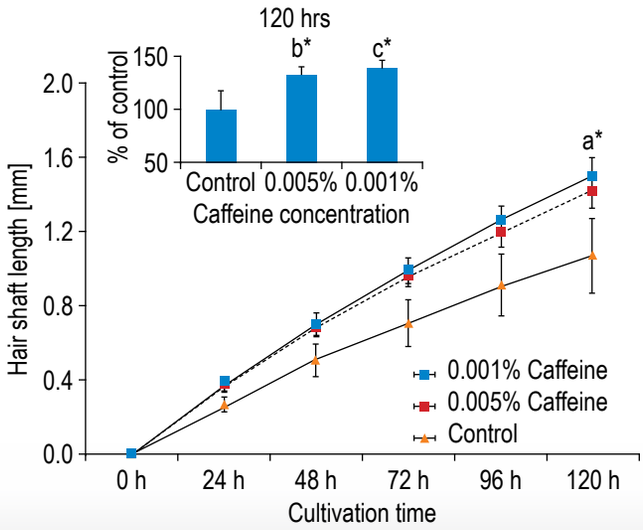
Figure 2. Mechanism of caffeine in promoting hair growth



Topical caffeine for managing androgenetic alopecia and telogen effluvium

Clinical evidence suggests topical caffeine to have beneficial effects in patients suffering from androgenetic alopecia and telogen effluvium with minimal adverse effects.[7] In a study, hair organ culture model was used by Fischer et al to investigate the effects of testosterone and caffeine on hair follicle growth stimulation. This study used male androgenetic alopecia scalp biopsy samples cultivated in different concentrations of testosterone or caffeine for duration of 120–192 hours. Increased hair shaft elongation was also seen after caffeine administration at 120 hours in comparison to the control group, Figure 3. Caffeine led to significantly higher hair shaft elongation at 120 h cultivation time in concentrations of 0.005% and 0.001% compared with caffeine-free control. The effect at this time-point was 33% and 39.4% increase, respectively. Thus, this study demonstrates that caffeine promotes human hair growth in the treatment of androgenetic alopecia.[8]

Figure 3. Hair shaft elongation with different caffeine concentrations



Values represent means ± SEM from two representative experiments; a* = caffeine vs. control, p = 0.038; b* = caffeine 0.005%, p = 0.044; and c* = caffeine 0.001%, p = 0.0275.

Table 1. Clinical studies using caffeine solution for the treatment of androgenetic alopecia and telogen effluvium[7]

Authors	Patient demographics	Treatment regimen	Results
Bussoletti et al (2011)[9]	40 male patients with AGA (age range: 19–55 years)	Topical caffeine lotion, once daily for 4 months	• Hair pull-tests results showed an improvement in hair tensile strength and a decrease in hair loss in 75% of patients by 8.14% after 2 months and in 83% of patients by 15.33% after 4 months • Improved scalp symptoms (redness, scaling/dandruff, tension/dryness), hair strength, and thickness • Reduced hair loss and progression of balding • After 4 months, 80% of patients satisfied with the test product.
Golpur et al. (2013)[10]	60 male patients with AGA (age range: 20–50 years)	1mL topical caffeine + minoxidil 2.5% solution twice daily for 150 days	• At day 150, increase in hair count from baseline hairs was observed (4.93 ± 0.18 to 5.39 ± 0.29) • Around 58.33% of patients 'very satisfied', 29.17% moderately satisfied' and 12.5% 'hardly satisfied' with treatment • Caffeine + minoxidil was more effective than minoxidil alone
Bussoletti et al. (2018)[11]	140 female patients with AGA (age range: 46–65 years	Group A: 7mL topical Phyto-caffeine daily for 6 months Group B. control group	• After 6 months, significant improvement in hair pull test was observed in group A (-3.1 vs. -0.5, p < 0.001) • Significant improvement in hair strength, thickness, and scalp erythema were reported in group A after 6 months • Improved hair loss intensity at 6 months • Around 40% of patients satisfied with the treatment after 6 months in group A compared to 10% in group B
Sisto et al. (2013)[12]	30 female patients with stress-induced TE (age range: 18–40 years)	Topical caffeine, once daily for 6 months	• Significantly decreased hair loss in 57% of patients after 6 months • Improved hair loss intensity and hair thickness • Decreased scalp dryness or tension • Around 67% of patients were satisfied with the treatment

AGA: Androgenetic alopecia, TE: Telogen effluvium.

Advantages of liposomal caffeine

Specific benefits for local action are offered by topical drug delivery administration over other routes. But major obstacle for drug delivery is presented by the barrier nature of the skin. Various strategies such as penetration enhancers and use of more advanced colloidal systems (microemulsions, solid lipid nanoparticles, and nanostructured lipid carriers) can reduce the effect of the skin barrier.[13]

Liposomes comprises of the phospholipid and an aqueous phase, which can hold the hydrophilic drugs in the aqueous compartment, while lipophilic drugs can be sequestered in the phospholipid. It has been reported that hydrophilic drugs such as caffeine delivery into hair follicle is enhanced by the liposomes.[13]

Efficacy of liposomal caffeine in enhancing follicular penetration[13]

A study was conducted to compare the ability of five different vesicle systems to deliver a model hydrophilic drug, caffeine, into and through excised human skin.[13]

- Significant enhancement of caffeine penetration into hair follicles was found for transferosomes and those liposomes containing oleic acid.
- As a percentage of the total applied drug dose, total caffeine delivery ranged from 6.6 and 7.4% for the aqueous solution and the conventional liposomes up to 14.9 and 16.0% for the transferosomes and niosomes, respectively.

Researchers suggested that polar drugs delivered from vehicles which are poorly miscible with sebum will have little chance to reach deeper parts of the hair follicles. On the other hand, lipophilic vehicles may assist the transport of drugs dissolved in the oil phase and into the pilosebaceous units. Therefore, the enhancement of follicular penetration would rely on the solubility of the drug in the formulation and the compatibility of the formulation with the lipid environment of the sebum.[13]

Summary

- Recent advances have shown that addition of caffeine has beneficial effect on hair loss in both genders.
- Caffeine enhances hair shaft elongation, prolongs anagen duration and stimulated hair matrix and outer root sheath keratinocyte proliferation.
- Various evidences have shown that caffeine administration improves the tensile strength of hair shaft and thickness and decreases hair loss. Improved scalp symptoms, and increase in hair count were also observed.
- A liposomal formulation of caffeine enhances the delivery of caffeine into hair follicles.

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DPCP in alopecia areata

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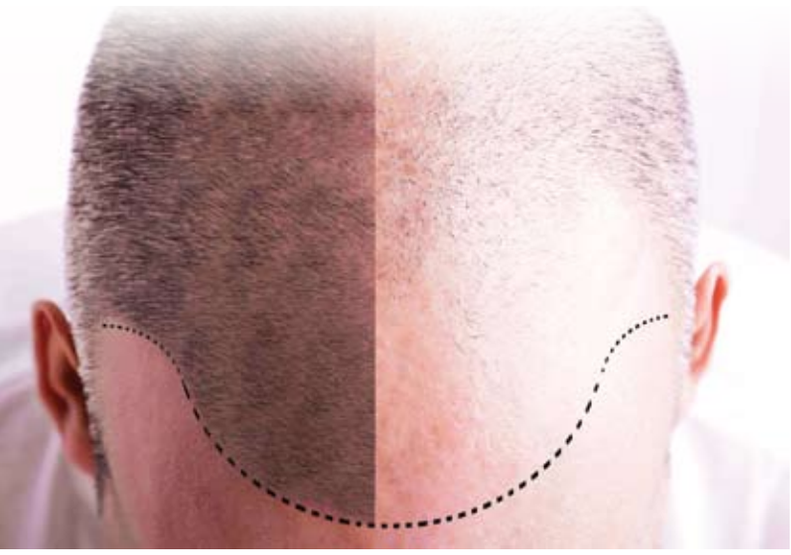
What is DPCP?

Diphenylcyclopropenone (DPCP) is a potent contact-allergy inducing hapten used in topical immunotherapy for various dermatological conditions such as alopecia areata and recalcitrant warts. It is available as 1 and 5 gram powder in a glass bottle. DPCP is the first choice for the treatment of extensive alopecia areata, especially in children.

How does it work?

Diphenylcyclopropenone triggers an immune response that is thought to oppose the action of autoreactive cells that would otherwise cause hair loss. One hypothesis is that in response to DPCP treatment, the body will attempt to downregulate inflammation through a variety of pathways, resulting in downregulation of the autoimmune response at the hair follicle. This autoinflammatory reaction would otherwise destroy body's hair follicles.

In alopecia areata, the earliest theory suggested was that immunotherapy may attract a new population of T cells into the treated area of the scalp, which could eliminate the antigenic stimulus present in alopecia areata. Another theory later proposed the concept of antigenic competition in which recruited suppressor CD8 T cells presumably exert a non-specific inhibitory effect on the immune response against hair follicles, thereby permitting hair growth.



How DPCP solution is prepared?

DPCP is white powder and various concentrations (2%, 1%, 0.5%, 0.2%, 0.1%, 0.01%, and 0.001) are made mixing it with acetone.

Weight	Volume of Acetone	Percentile
2 gm	100 ml	2%
1 gm	100 ml	1%
500 mg	100 ml	0.5%
200 mg	100 ml	0.2%
100 mg	100 ml	0.1%
10 mg	100 ml	0.01%
1 mg	100 ml	0.001%

It is stored in amber coloured bottles. Usually, while opening the bottle each time, acetone evaporates and the solution gets concentrated. Therefore, we suggest the use of bottles with a rubber stopper to store the solution and each time the desired amount is withdrawn, avoiding repeated opening of the lead.

Before you begin treatment you need to know that…

1. DPCP is not FDA approved, so, only specially trained doctors will apply it. It is a clear liquid, which is swabbed on to the skin using a large cotton "Q-tip".
2. Women of childbearing age must use a reliable method of contraception, as the effects of DPCP on fetus are not known.
3. This is a weekly treatment.
4. DPCP does not work for everyone. DPCP is successful in approximately 17%–70% of patients, depending on the extent and duration of alopecia areata.

How to start?

A 2% solution is applied on 2 x 2 cm area of scalp. This initial dose allows patient's immune system to prepare for regular exposure to DPCP.

Leave the DPCP on scalp for 48 hours. Keep scalp/hair covered with a hat or scarf for two days, as the solution can break down with exposure to light. After 48 hours, shampoo the scalp to ensure the solution is completely removed.

Care must be taken to ensure that DPCP is not transferred from scalp to other parts of body or to someone else. Try to avoid touching the scalp for 48 hours.

On next visit, post 48 hours, response to 2% DPCP is assessed. There should be mild to moderate sensitization which could be in the form of erythema, itching, redness and few blisters. Severe reaction will be treated by appropriate medications. A week later, according to our protocol, we apply 0.001% of DPCP to alopecia areata patches if initial reaction is mild to moderate and 0.0001% is applied if reaction is severe. The strength of DPCP will be increased on weekly basis, instead of 3 weekly, as mentioned in literature. Our protocol ensures safety to the patient. If scalp becomes too itchy or if there is burning or blistering; wash DPCP early. Remember more is not better in this case. A slight tolerable itch is sufficient to be effective. This concentration is maintained. Once there is complete regrowth, the frequency of application is reduced and treatment is maintained. Treatment can be discontinued for a long time if the condition goes into remission.

What are the Side effects?

Some or all of which you may experience. These include itching, dry flaking skin, severe eczema, blistering, lightening or darkening of the treated skin and enlarged lymph nodes. Side effects will be dealt with on an individual basis as they arise.

Studies related to DPCP in alopecia areata:

1. In a study by Cotellessa et al 48% of patients with extensive alopecia areata treated with DPCP (42 with >90% and 14 with 30%–90% area of scalp) showed regrowth of terminal hair all over the scalp after 6 months of therapy.
2. Wiseman et al reported >75% growth of terminal hair in 100% of patients with 25–49% alopecia areata, 88.1% of cases with 50%–74% alopecia areata and 60.3% of cases with 75%–99% of alopecia areata.

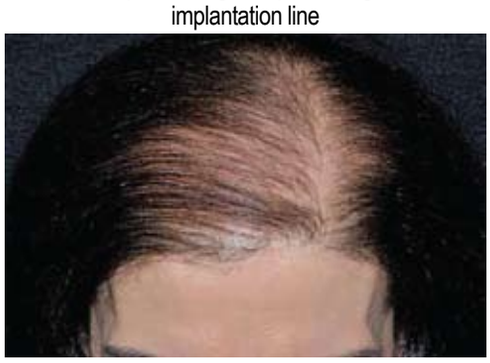
Efficacy and safety of finasteride in women with female pattern hair loss

Dr Dhanashree Bhide
MD (Skin), DNB (Skin)

Female pattern hair loss: An overview

Female pattern hair loss (FPHL) is a condition characterized as a non-scarring diffuse thinning of hair especially in the central, frontal and parietal scalp regions. It evolves with progressive miniaturization of hair follicles and ultimately leads to the reduction of the number of hair (Figure 1).[1]

Figure 1. Female Pattern Hair Loss. Diffuse thinning of the hair in the frontal and parietal regions, preserving the anterior hair implantation line



Nonscarring alopecia (unspecified) is reported to be one of the ten most common diagnoses in female patients.

The prevalence of FPHL increases with age with onset during reproductive years. Among patients aged 25–40 years, greater demand for treatment is observed. A second peak incidence is observed among women between 50 and 60 years of age. However, FPHL demonstrates an inconsistent response to treatment.[1] FPHL is also associated with hyperandrogenic conditions such as PCOS; around 67% of patients with FPHL were diagnosed to have PCOS, compared with 27% in those without PCOS as per a study.[2]

Association of the anagen phase duration to FPHL

The biological cycle of the hair follicles are divided into three phases: anagen (growth phase), catagen (regression), and telogen (resting phase). The original hair falls out (exogenous phase exogen) and is replaced by new hair at the end of the telogen phase. A reduction in the duration of the anagen phase and thinning of the hair leads to FPHL.

Miniaturized hair takes the place of thick pigmented hair.[1]

The multifactorial cause of FPHL

FPHL is caused by a combination of hormonal factors, microinflammatory process, and genetic factors. FPHL can be associated with conditions in which androgen levels are elevated. In addition to this, different patients have shown different response to various treatment strategies suggesting that multiple pathogenetic mechanisms are involved in the development of FPHL.[1, 3]

Hormonal therapies for FPHL: Focus on Finasteride

The only medication approved for FPHL is 2% topical minoxidil with twice daily application and now 5% with once a day usage. There are several other treatment alternatives that are possibly effective, but not approved.[6]

The hormonal therapies used to treat FPHL act by inhibiting the process of androgen conversion and subsequent binding to its target. The currently used therapies include 5-alpha-reductase inhibitors (finasteride and dutasteride), androgen receptor blockers (spironolactone, cyproterone acetate and flutamide), and estrogen and oral contraceptive drugs.[4]

Overall, there is not enough evidence based data to support the routine use of antiandrogens in FPHL. All hormonal treatments are potentially teratogenic when used in women of child bearing age group. Most of these agents amount to off label use and patient counselling regarding the same is mandatory especially for avoiding pregnancy.[7]

Finasteride is a 5-alpha-reductase type II inhibitor, which prevents the conversion of testosterone into dihydrotestosterone (DHT). It is approved for use in male pattern hair loss but has not been approved for FPHL, so its use in women is off-label. Finasteride has a safe side-effect profile in men, but further controlled studies need to be conducted for extending the knowledge on its benefits and safety profile for women. Premenopausal women need to utilize safe contraception methods during treatment as the drug can cause feminization of the male fetus if taken throughout pregnancy. Another possible concern for this treatment is the slight rise of estrogen levels due to aromatase conversion of testosterone to estradiol. For this reason this treatment is not advisable in females with a family or personal history of breast cancer.[6]

Efficacy of oral finasteride 2.5 mg/day in women with FPHL[3]

In a recent retrospective study, researchers evaluated the clinical efficacy and safety of finasteride at a dose of 2.5 mg/day in premenopausal or postmenopausal patients with FPHL. About 112 patients (mean age at initial visit was 54.1±7.5 years) were included.[3]

Global photographs of the patients obtained at the time of their first visit were compared with those obtained at the time of their last follow-up. A 3-point scale was used to grade the change in scalp hair appearance as follows: no response or aggravation (0), slightly improved (1), and significantly improved (2).[3]

Findings of the study revealed that: [3]

Global photographs assessment:

- A better hair growth was associated with an older age of onset, a longer duration of treatment and lower Ludwig grade (Spearman's correlation coefficient = -0.331, $p<0.001$) (Table 1).[3]
- Around 33/112 (29.5%) patients showed slight improvement, 73/112 (65.2%) showed significant improvement, whereas no change was recorded in 6/112 (5.4%) patients.[3]
- A positive association was observed between the age of onset, duration of treatment,

Table 1. Baseline characteristics and their association with improvement in scalp hair appearance in 112 patients [2]						
Characteristic	**Significant improvement (+2) (n=73)**	**Mild improvement (+ 1) (n=33)**	**No change (0) (n=6)**	**Mean score**	**Spearman's correlation coefficient**	**p value**
Mean age±SD (range)	53.8 ±7.4 (38–73 years)	54.1 ± 8.1 (30–69 years)	57.8 ± 5.8 (51–67 years)	-	-0.092	0.335
Mean age at onset±SD (range)	43.3 ± 9.9 (17–59 years)	37.7 ± 10.8 (20–61 years)	26.8 ± 6.4 (17–35 years)	-	0.348	<0.001
Mean duration of FPHL ± SD (range)	10.5 ± 8.3 (1–33 years)	16.4 ± 9.8 (4–41 years)	31.0 ± 6.4 (21–41 years)	-	-0.452	<0.001
Mean duration of treatment ± SD range)	8.0 ± 3.4 (4–59 months)	16.1 ± 11.8 (3–40 months)	8.0 ± 3.4 (4–12 months)	-	0.192	0.044
Ludwig scale					-0.331	<0.001
I (n = 59)	47	9	3	1.75	-	-
II (n = 47)	25	20	2	1.49	-	-
III (n = 6)	1	4	1	1.00	-	-

and improvement of scalp hair (Spearman's correlation coefficient = 0.348, 0.192, p<0.001, p=0.044) (Table 1). [3]

Safety: No adverse effects were reported by the patients.

Conclusion

- FPHL, a condition defined as a non-scarring diffused thinning of hair especially in the central, frontal and parietal scalp regions, is highly prevalent in old aged women.
- FPHL results due to a reduction in the duration of the anagen phase and thinning of the hair.
- FPHL is caused from an interplay of multiple pathogenetic mechanisms which involves hormonal factors, microinflammatory process and genetic factors.
- Finasteride, a 5-alpha-reductase type II inhibitor, prevents the conversion of testosterone into dihydrotestosterone (DHT), an androgen known to play a role in causing hair follicle miniaturization.
- Finasteride at a dose of 2.5 mg/day demonstrated significant improvement in hair growth in 65.2% of premenopausal/postmenopausal patients.
- Finasteride at a dose of 2.5 mg/day for patients with FPHL also had a better effect on hair growth when patients had a lower Ludwig score and an older age at onset.
- No adverse effects were reported during the study.
- Therefore, finasteride at a dose of 2.5 mg/day in patients with FPHL was found to be safe and efficacious.

Important Note:

The 5-alpha reductase inhibitors are reported to be teratogenic, hence, classified in FDA pregnancy category X, and is contraindicated in women who are or may become pregnant.[5]

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LATEST HAPPENINGS

Vitamin D deficiency: A risk factor associated with the development of alopecia areata

Vitamin D deficiency plays a major role in the pathogenesis and exacerbation of alopecia areata. In a study conducted by Rehman F et al, vitamin D levels and its correlation with severity, pattern, and extent of the disease were evaluated. The study included 135 individuals with alopecia areata who were grouped according to the severity and extent of the disease. It was observed that, vitamin D deficiency/insufficiency was significantly higher in patients with alopecia areata as compared to control group ($p = 0.01$). A highly significant mean difference in the vitamin D levels were observed between alopecia areata and control group ($p = 0.0004$). A negative correlation between vitamin D levels and severity, pattern, and extent of diseases were also observed. Hence, vitamin D deficiency may be considered as one of the factors associated with the development of alopecia areata.

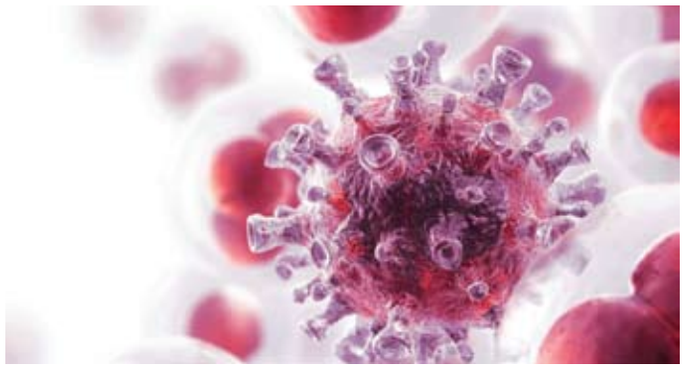
Reference

Rehman F, Dogra N, Wani MA. Serum vitamin D levels and alopecia areata- A hospital based case-control study from north-India. Int J Trichology. 2019; 11(2): 49–57.

Role of cytokines in the progression of alopecia areata

In a recent study, researchers evaluated the serum levels of cytokines secreted by Th1 (IL-2, IFN-γ), Th2 (IL-4), Th17 (IL-23, IL-17A), and Treg (IL-10) pathways in patients with active alopecia areata and correlated their levels with the severity of the disease. A total of 40 patients with untreated active alopecia areata of the scalp were included and the serum levels of cytokines were measured using enzyme-linked immunosorbent assay. The findings of the study revealed that:

- Compared to the control group, a significantly raised serum levels of cytokines IL-2, IFN-γ, IL-17A, and IL-10 and non-significantly raised levels of IL-23 were reported in alopecia areata patients
- A statistically significant reduction in the levels of IL-4 were reported in patients with alopecia areata as compared to control group ($p< 0.05$)



- A significant positive correlation was reported between the increase in SALT Score and serum levels of IL-2, IL-17A, and IL-23. ($p < 0.05$)

Therefore, researchers suggest a role of cytokines as a biomarker of disease activity in patients with alopecia areata.

Reference

Gautam RK, Singh Y, Gupta A, et al. The profile of cytokines (IL-2, IFN-γ, IL-4, IL-10, IL-17A, and IL-23) in active alopecia areata. J Cosmet Dermatol. 2019: 10.1111/jocd.12970.

WORLD TRICHOLOGY SOCIETY CONFERENCE ORLANDO 2020

Orlando is one of the world's most visited family destinations, and while its illustrious themed attractions may steal the limelight, with a vibrant and well-kept city center and a climate that averages around 75° Fahrenheit (25° Celsius), there is a surprising amount of things to experience here. Welcoming tens-of-millions of visitors per year, both Disney World and Universal Orlando pack in an entire trip's worth of fun on their own. But it would be a mistake to overlook Orlando's other activities because the rest of 'The City Beautiful' is full of similar family-friendly attractions like waterparks, giant aquariums and zoos, exciting shows, airboat tours around the swamps and plenty of golfing opportunities.

Dear Friends and Colleagues,

I am delighted to announce that the next World Trichology Society Conference on the United States mainland will take place Sunday-Monday March 1-2, 2020 at one of the Disney Resort Hotels, Orlando, Florida.

This World Trichology Society (WTS) conference will again be a comprehensive, international trichological meeting for the advancement of hair-loss and scalp knowledge. The conference is ideal for all certified trichologists, student trichologists, hair-loss specialists, cosmetologists; and dermatologists, other physicians, physician assistants, and nurse practitioners with an interest in hair and scalp problems.

The World Trichology Society conference will include presentations from some of the best-known names in trichology and hair-loss research. It will be ideal for networking, learning about new research, experience-sharing, and discussions of ways to educate the public on trichology as the go-to specialism for information about hair loss and scalp problems.

Also, you will receive discounts to Disney's theme parks, so bring your family!

More information soon!

We look forward to welcoming you all to Orlando!

Best regards,
Dr. David Kingsley, PhD, WTS
WTS President and Conference Co-Chair

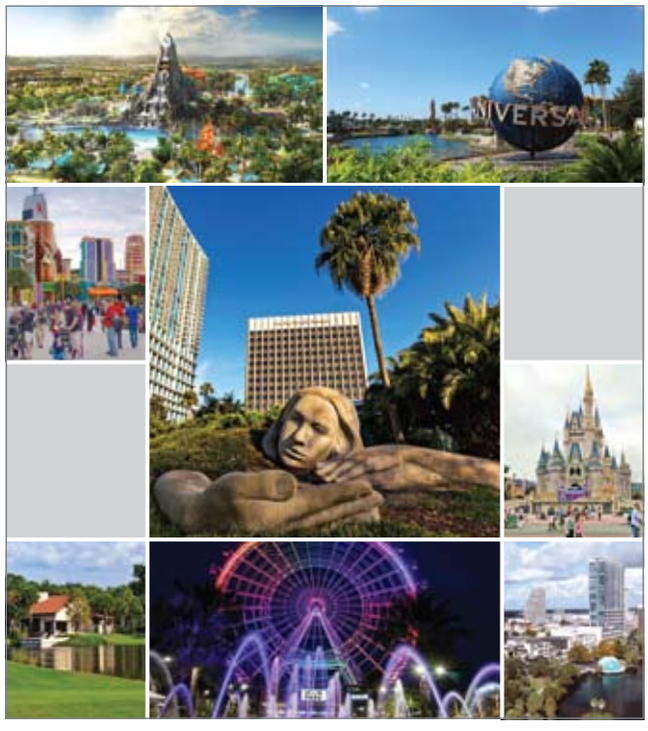


Photo Quiz

1. **A 22-year-old active-duty service member presented with progressive patchy hair loss on the scalp and body with minor sparing of the axillae and pubic region. What is the most likely diagnosis?**

   A. Alopecia totalis
   B. Alopecia universalis
   C. Androgenetic alopecia
   D. Cicatricial alopecia

2. **A 32-year-old man presented with persistent, painful, draining scalp lesions that had been present for the past five years. What is the most likely diagnosis?**

   A. Dissecting cellulitis of scalp
   B. Kerion
   C. Folliculitis
   D. Discoid lupus erythematosus

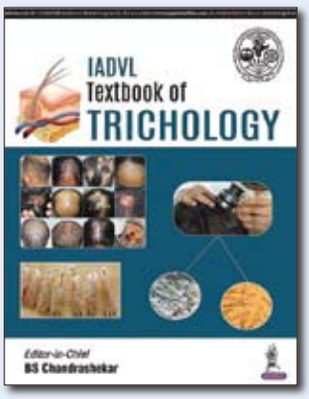
3. **A female infant was noted to have a midline lesion on the posterior aspect of her scalp. The lesion was hairless. What is the most likely diagnosis?**

   A. Langerhans cell histiocytosis
   B. Sebaceous nevus
   C. Neonatal herpes infection
   D. Aplasia cutis congenital

Answers: 1: B; 2: A; 3: D

BOOK REVIEW

IADVL Textbook of Trichology



Author:
BS Chandrashekar

Publisher:
Jaypee Brothers, Medical Publishers Pvt. Limited, 30-Jun-2018

Number of pages: 534

The 'IADVL Textbook of Trichology' is a comprehensive guide to the diagnosis and treatment of diseases and disorders of the hair and scalp.

The book is divided into 6 sections.

- The first section begins with an overview of hair and the normal scalp, factors controlling hair growth and changes that occur during the aging process.
- The next section details diagnostic trichology methods including microscopy and trichoscopy.
- The book also covers diagnosis and treatment of numerous hair disorders, both clinical and surgical procedures, for common and more complex conditions.
- A chapter on recent advances in surgical management is included.
- The book ends with a final section of interdisciplinary issues in the management of hair disorders such as pediatric, psychiatric, gynecologic and endocrinologic liaison therapy.

A 'key messages' box is included at the start of every chapter and concludes with a summary of the topic. The book is highly illustrated with clinical photographs, diagrams and tables.

For the use of a registered medical practitioner or a hospital or a laboratory only.

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