WORLD TRICHOLOGY SOCIETY

TOT Times of Trichology™

2020 | Issue 2

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President's Message

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

Welcome to the second 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

Topical caffeine is effective, well-tolerated, and safe in the treatment of AGA

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Introduction

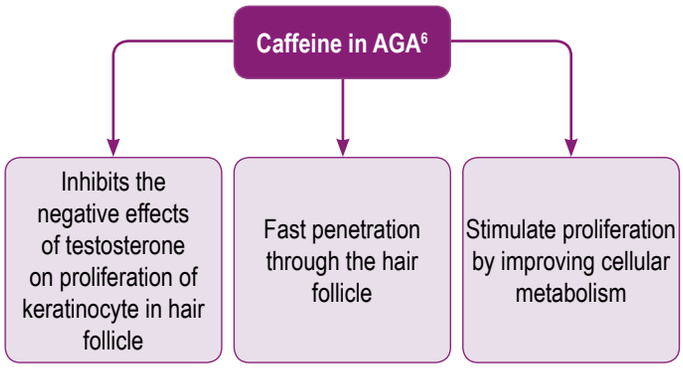
Thicker, fuller hair is the symbol of youthfulness in many men and women, and hair loss can negatively affect the person's self-image and quality of life.[1] The most common causes of hair loss in both men and women are androgenetic alopecia (AGA) and telogen effluvium, which shares similar symptoms but have different pathophysiological mechanisms.[1]

AGA or male-pattern hair loss is a dihydrotestosterone induced hair loss resulting from miniaturization of hair follicles by transforming terminal hair into vellus hair. If the disease is left untreated, patients may further experience progressive hair loss.[2] The incidence of AGA increases with increasing age, with 30%, 50%, and 80% of the men are affected over the ages of 30, 50, and 80 respectively.[2,3] Even though AGA is highly prevalent in men, it can affect women as well.[3] Approximately 57% of women over the age of 80 are affected by AGA.[2] Continuous inflammation and remodeling of the connective tissue of follicles are the leading cause of permanent hair loss in patients with AGA.[3]

Telogen effluvium is another frequent cause of diffuse hair loss, characterized by excessive shedding of hair diffusely from the scalp.[3,4] This usually begins 8–12 weeks after a series of triggering events and resolves within 3–6 months.[4] Typical triggering factors include metabolic stress, hormonal changes, medications, pregnancy, major illness or complicated surgery.[4,5] Self-limiting telogen effluvium once resolved can be diagnosed as acute telogen effluvium and considered to be chronic telogen effluvium (CTE) when shedding persists beyond 6 months. Primary CTE occurs unexpectedly in women aged 30 to 50 years. The bi-temporal recession of the anterior hairline, decrease in the thickness of their ponytail diameter, and trichodynia are the clinical features commonly seen in patients with primary CTE.[4]

Therapeutic options for the treatment of AGA and telogen e uvium: Role of ca eine

Oral finasteride and topical minoxidil 2% and 5% are the approved drugs for the treatment of AGA. Also, considering the potential side effects, expense, or invasiveness, surgical options are unsuitable or impractical for use in managing AGA. Hence, a suitable alternative or adjuvant therapies to systemic drug treatment should be considered for managing AGA.[1] There are several topical drugs and cosmetic substances available, which complement the medical treatment of AGA.



Over the past years, caffeine has been considered for the management of AGA. It is typically applied in the form of shampoo or a lotion, and has good efficacy, tolerability, and skin compatibility in AGA management.[6]

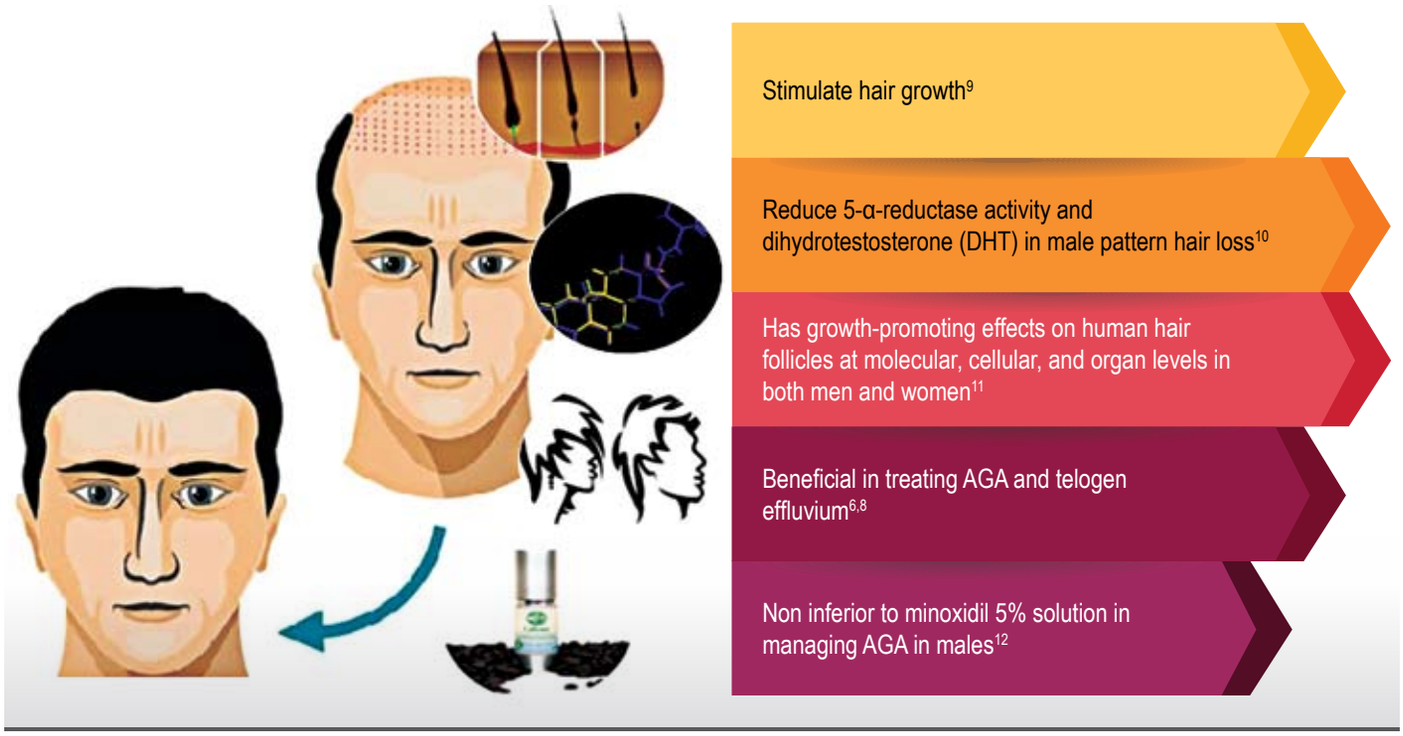
Minoxidil is a vasodilator that has a positive impact on the anagen phase of the hair cycle. It plays a major role in the management of CTE. According to expert consensus in India, minoxidil is prescribed only for patients with CTE, but not with active TE (ATE). Minoxidil 2% and a peptide combination therapy provide better results than each drug alone in telogen effluvium management. Patient counseling is necessary as the treatment with minoxidil leads to hair loss in the early phases when the telogen hairs sheds and new hair growth occurs. Serum-based peptides, caffeine-based preparations, and topical botanicals have been proven to be beneficial in the treatment of telogen effluvium.[7] Topical use of caffeine provides better efficacy and skin compatibility in the treatment of female pattern hair loss or telogen effluvium [8]

Ca eine is safe and e ective in men with AGA[12]

A study was conducted to determine the non-inferiority of 0.2% caffeine-based topical liquid solution (n=82) compared to 5% minoxidil solution (n=79) in males with AGA (aged 18–55 years). The frontal and occipital trichogram was used to assess the percentage change in the proportion of anagen hairs from day 1 (baseline) to day 180 (6 months).

- At 6 months, mean improvement in anagen ratio of the trichogram of 11.68±12.44% was reported in subjects using 5% minoxidil

Efficacy of caffeine in hair growth



solution, while 10.59 ± 12.02% in subjects using 0.2% caffeine solution (p = 0.574), Table 1.

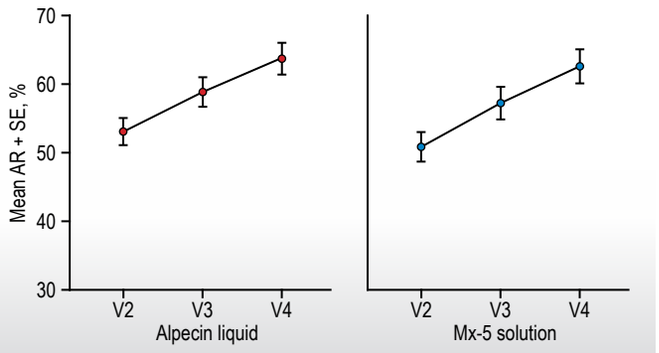
Table 1. Change in percentage of anagen hairs from baseline to 3 and 6 months in the per-protocol population using frontal and occipital trichograms

	Caffeine-based topical 0.2% liquid (n = 82)	Minoxidil 5% solution (n = 79)
Baseline	53.01 (17.13)/59.37 (0.7–76.6)	50.81 (19.33)/56.86 (0.8–77.3)
3 months	58.76 (19.75)/67.42 (3.8–83.1)	57.15 (20.94)/65.45 (2.4–80.0)
6 months	63.60 (21.43)/74.75 (5.7–82.1)	62.49 (22.47)/72.86 (4.8–83.2)

Results are expressed as means with standard deviations in parentheses, followed by medians with ranges in parentheses

- The difference in the mean increase in anagen ratio was 1.09% between both groups
- The upper limit of the 95% CI has not reached below the non-inferiority range of 5%, therefore, 0.2% caffeine-based topical liquid was found to be non-inferior to 5% minoxidil solution, Figure 1.

Figure 1. Mean (±standard error, SE) of the rate of anagen hair (AR; %) by treatment and visit (baseline, after 3 months, and after 6 months)



- The intensity of hair loss, the number of hairs falling out while combing, and hair thickness significantly better at 6 months than baseline (p < 0.01) in both groups. But, there was no significant differences found between groups at 6 months.
- In subjects using the caffeine-based topical 0.2% liquid, a significant improvement was reported in scalp itchiness at 6 months (p = 0.003), while no such improvement was reported in subjects using minoxidil 5% solution (p = 0.211).
- A significant improvement in scalp tension/ dryness (p < 0.05) in both groups were reported from baseline to 6 months, with no significant difference between groups.
- A significant improvement in terms of hair strength, balding progression, and extent of hair loss (all p < 0.01) were observed from baseline in both treatment groups at 6 months, with no significant differences between groups at 6 months.
- A significant improvement in scalp redness and scaling/dandruff from baseline in both treatment arms at 6 months (all p < 0.05) was observed, with no significant differences between groups.

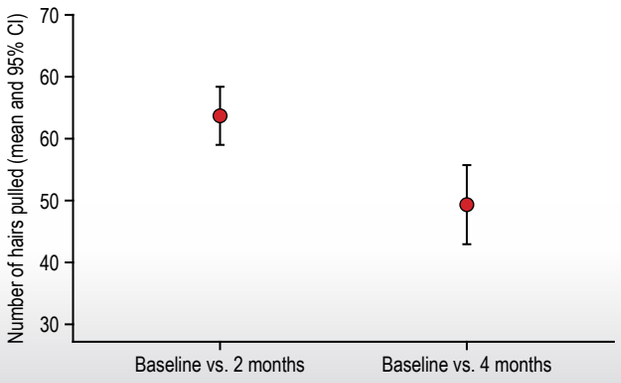
In conclusion, a caffeine-based topical liquid is as effective as minoxidil 5% solution in the management of AGA in males.

Safety and tolerability of ca eine in the management of AGA[6]

A study was conducted in 40 men (age range: 19–55years) to test skin compatibility, efficacy, and cosmetic quality of caffeine-containing lotion in patients with AGA. Measurement of primary efficacy variable (hair pulled back to pull-test) mean, standard deviation, and 75%-percentiles were done at baseline, 2, and 4 months after application of the caffeine lotion.

- A very good skin compatibility and good cosmetic efficacy were observed in patients with AGA after the application of the caffeine lotion.
- No clinical signs or discomfort were reported with the use of caffeine lotion.
- The use of caffeine lotion reduced the number of hair by 8.14% after 2 months and 15.33% after 4 months of caffeine treatment in hair pull test, indicating an improvement in hair tensile strength and a decrease in hair loss.
- The percentage of 'positive' volunteers (increase in tensile strength of hair and decrease in hair loss) was 75% after 2 months and 83% after 4 months of caffeine treatment, Figure 2.

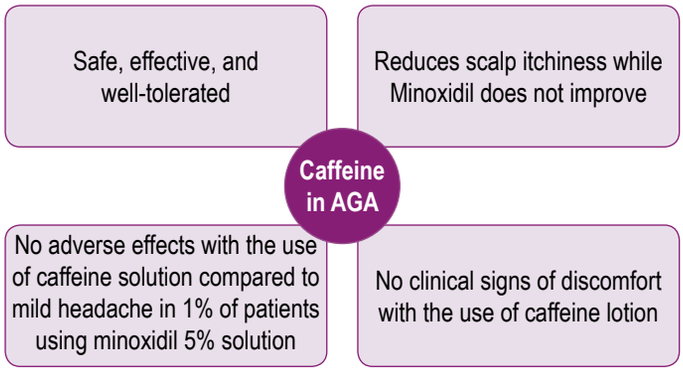
Figure 2. Decreased hair pulled in the pull-test after 2 and 4 months of caffeine lotion application, compared to baseline



- The dermatological control confirmed the good efficacy of the product, with a 43% reduction in premature hair loss, and a 53% improvement in hair texture (force, tensile strength)
- The use of caffeine lotion also improves scalp conditions in most of the volunteers who showed abnormalities at enrolment.
- Around 80% of patients were satisfied with the test product after 4 months of treatment.

In conclusion, caffeine is effective, safe, and well-tolerated in the treatment of AGA.

Safety of caffeine in AGA[12]



Summary

- Hair loss can negatively affect the patient's self-image and quality of life. The most common conditions of hair loss in both men and women are AGA and telogen effluvium which share similar symptoms but have different pathophysiological mechanisms.
- AGA or male-pattern hair loss is a dihydrotestosterone mediated hair loss disorder, which induces miniaturization of hair follicles by conversion of terminal hair into vellus hair.
- Telogen effluvium is another frequent cause of hair loss, characterized by excessive shedding of hair diffusely from the scalp.
- Oral finasteride and topical minoxidil 2% and 5% are the only drugs approved for the management of AGA. However, topical drugs and cosmetics should be considered as a safe alternative to systemic drug treatment in AGA patients.
- According to expert consensus in India, minoxidil is prescribed only for patients with CTE, but not with ATE. In addition, serum-based peptides, caffeine-based preparations, and topical botanicals have been proven to be beneficial in the treatment of telogen effluvium.
- The caffeine-based topical liquid is as effective as minoxidil 5% solution in managing AGA in males.
- The caffeine-based topical liquid is well-tolerated and safe for the treatment of alopecia
- Hence, caffeine is considered interesting and promising for the cosmetic treatment of AGA.

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Effective management of Telogen Effluvium: Key expert opinions

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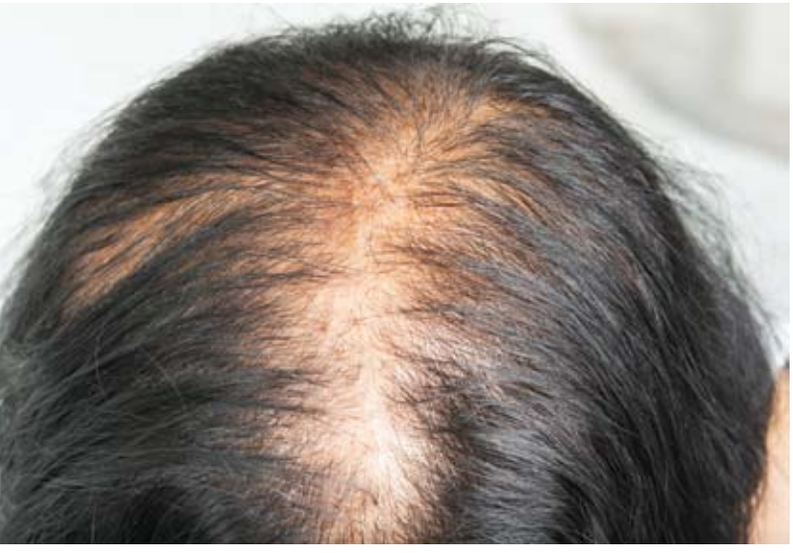

Introduction

Alopecia is a common condition that affects the quality of life and is associated with low self-esteem and loss of confidence. It is classified based on the abnormalities of hair growth cycle or follicular damage.[1] The prevalence of telogen effluvium is well recorded due to its subclinical nature and it affects both genders, but often women are over-represented due as they are more alarmed by the hair fall and seek treatment.

According to a study in India aimed to determine the causes of diffuse hair loss in women, diffuse hair loss is multifactorial in origin and most of these individuals suffered from telogen effluvium.[2]

Recently, a consensus paper has been developed taking opinions of 120 dermatologists to standardize the diagnostic and therapeutic approaches from the Indian perspective.[3] The opinions were recorded after brainstorming on the etiopathogenesis, clinical features, diagnostics, and management of telogen effluvium.

Definition

Telogen effluvium: Excessive shedding of hair after 2–3 months of a triggering event. The condition is diffusive in nature, abrupt in onset, and rapid in progression.

Clinical characteristic

Telogen effluvium is self–limiting condition that is characterized by premature termination of

the anagen phase of the hair follicular cycle and predominance of the telogen phase.

Etiopathology

In telogen effluvium hair fall has been attributed to five functional changes in the hair cycle, such as immediate anagen release, delayed anagen release, short anagen syndrome, immediate telogen release, and delayed telogen release.[4]

History of hair fall

1. Understanding the duration of hair fall can help classify the condition as acute (hair loss 6 months), example, change in residence for environmental factors, quality of water etc.
2. History of diseases, surgery
3. For women, menorrhagia, puerperal disorders
4. Current medication (isotretinoin, steroids, beta blockers, phenytoin, contraceptives and anticancer drugs)
5. Prior treatment with Minoxidil
6. Stressors of all kinds (physical, psychological, nutritional or medicinal)
7. Sleep disturbances
8. Nutritional deficiencies

KEY POINT

In individuals with diffuse hair loss a detailed history must be taken, which should include direct questions on duration of hair fall, any change in their residence, recent diseases or surgeries, gynecological/obstetric issues, stress, nutritional deficiencies, and any hair treatment.

Diagnosis

Examination

1. Clinical examination: Indicates degree and pattern of hair loss and provides evidence of erythema, inflammation, or scaling
2. Assessment of hair shafts: breakage, diameter, and length
3. Hair-pull test: should be performed on all patients with hair loss
4. Counting shed hair daily: >100 hair per day indicate telogen effluvium
5. Hair combing tests

KEY POINT

In clinical examination, the four essential tool are scalp hair-pull test, trichoscopy, and hair combing test, which enables a quick diagnosis in patients with hair loss.

The following basic investigation parameters can be ordered for every patient with diffuse hair loss:

- Complete blood count
- Routine urine examination
- Serum vitamin D and thyroid function tests

KEY POINT

Basic investigations are to be ordered for all patients with diffuse hair loss. The advanced tests must be conducted in patients whose history and examination findings indicate specific disease or deficiency.

Advanced diagnostic tests and their implications:

- Serum calcium to diagnose hypocalcemia
- Vitamin B12 (for pernicious anemia and in vegetarians, malabsorption syndromes, atrophic gastritis, autoimmune disorders)
- Iron profile (total iron binding capacity and serum ferritin level) to detect iron deficiency
- Serum zinc (to detect deficiency)
- Serum proteins (for protein energy malnutrition)
- Hormonal assays to distinguish between eelogen effluvium and androgenetic alopecia (AGA)
- Anti–dsDNA antibodies if autoimmune diseases are suspected
- Serum anti–mullerian hormone and testosterone levels for mixed alopecia

Differential diagnosis

Table 2: Differences between androgenetic alopecia and telogen effluvium

Androgenetic alopecia	Telogen effluvium
Commonest type of progressive hair loss	Non-scarring diffuse hair loss from scalp (usually self-limiting)
Anisotrichosis: Variation will be > 20%	Anisotrichosis absent
Terminal: Vellus hair ratio is < 4:1	Terminal: Vellus hair ratio is normal, > 7:1
Hair pull test: Temporal pattern	Hair pull test: Diffuse pattern
Presence of a peripilar halo around the hair with visible yellow dots	Peripilar halo absent
Miniaturization: More pronounced (60%)	Miniaturization: Less pronounced (up to 40%)

KEY POINT

Patient education is important in the management of patients with telogen effluvium and can enable handling anxieties, especially in case of balding. The normal hair cycle should be explained so that patients do not have unreasonable expectations.

Consensus on medical management of telogen effluvium

Patient education

- Inform that identification and removal of triggers may resolve problem (hair fall can continue for up to 6 months or less).
- Hair shedding can be short-lives and controlled without medication.
- Nutritional requirement must be considered and suspected drug should be discontinued or changed.
- Medical therapy is mostly supportive for the treatment of telogen effluvium and the treatment is not required if the underlying cause is removed.
- Before deciding the treatment of protocol, serum levels of vitamin B12, D3, and iron should be determined.
- Healthy food habits should be recommended, and if needed amino acids, vitamin B complex, zinc, and proteins can be added.

KEY POINT

Amino acids and topical peptides may be beneficial in in the treatment of telogen effluvium.

TREATMENT

The use of Minoxidil

- Shortens telogen phase of the hair cycle, thus encouraging hair follicles to prematurely enter the anagen phase
- Extends anagen phase and increases the size of hair follicles
- Delays keratinocyte senescence and stimulates/inhibits proliferation of epithelial factors and fibroblastic cells
- Inhibits collagen and prostaglanding E2 and vascular endothelial growth

KEY POINT

Minoxidil is not recommended in the management of chronic telogen effluvium. Minoxidil and a peptide combination therapy may be better as compared to either of the monotherapy.

Biotin

Biotin (vitamin B7 or vitamin H) is a co-enzyme for carboxylase enzymes. It aids in the metabolism of glucose, branched-chain amino acids, and fatty acids.

KEY POINT

Biotin supplementation at a dose of 30 µg may be useful but evidence are lacking for benefits of higher doses, unless a deficiency is present.

Other Adjuvant treatment options

- Shampoos do not have any role in the treatment of telogen effluvium.
- Hair oils are good pre-shampoo conditioners and protect the cuticles.
- Nutritional supplement: Iron, zinc, folic acid, biotine, niacin, vitamins A, D, and R, fatty acids and amino acids may aid in promoting hair growth and its structure.
- Nutrition deficiency is associated with telogen effluvium

KEY POINT

Nutritional supplements play an important role in the treatment of telogen effluvium. Supplements containing amino acids, zinc, calcium, iron, copper, selenium, and folic acid can be considered

Platelet rich plasma

Platelet-rich plasma (PRP) is a novel treatment option for hair loss. The autologous concentration of platelets contained in a small volume of plasma is used to promote rejuvenation of hair follicles. This is because of the presence of various growth factors and cellular adhesion molecules.[5]

KEY POINT

PRP treatment is not used in the management of acute telogen effluvium. However, it is sometimes used in chronic telogen effluvium.
Unnecessary use of PRP should be avoided in patients with telogen effluvium as the results are variable.

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LATEST HAPPENINGS

Alopecia areata incognita and di use alopecia areata: Are they similar?

Alopecia areata (AA) is a non-scarring hair loss that causes round patches of baldness on the scalp. Alopecia areata incognita (AAI) and diffuse alopecia areata (DAA) are the two most common subtypes of non-patchy AA that can lead to a diffuse and acute pattern of hair loss. Alessandrini A et al analyzed the clinical, trichoscopic, histological, and therapeutic features of AAI and DAA from hair disease outpatient consultation department data (April 2012 to April 2017) of 107 patients (female = 105 [98.13%]; mean average age = 40.55 years). The results of the analysis demonstrated that DAA was greatly involved in the parietal and anterior-temporal regions whereas AAI was mainly observed in the occipital-parietal regions in patients with AA. In DAA, dystrophic hair and black dots were more recurring. The most recurring hair loss was yellow dots, yellow dots with vellus hairs, and smaller hair in regrowth; whereas the presence of pigtail hair was found most extensively in patients with AAI. Accordingly, the most recurrent trichoscopic sign in both DAA and AAI was the existence of yellow dots without vellus hairs, which were majorly seen with an increased percentage in patients with DAA. Both diseases exhibit a benign course and response to topical steroidal treatment. The authors concluded that trichoscopy is very beneficial in the differential diagnosis of DAA and AAI, and the selection of the best site for biopsy. The authors also suggested considering these entities in the presence of diffuse hair thinning.

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Erosive pustular dermatosis of the scalp

Introduction

Erosive pustular dermatosis of the scalp (EPDS) is a chronic inflammatory disorder of the scalp. This condition is predominantly observed in the elderly population. It is characterized by detectable small recurrent pustules, inflamed erosions and more or less thickened grey, yellow or yellow-brown crusts, which develop slowly scarring alopecia that spreads slowly.[1]

The disease may become chronic and respond poorly to the treatment, requiring a long-term management. Often, EPDS seem to affect elderly with androgenetic alopecia, sun-damaged skin, or a history of scalp trauma. However, occasionally younger people including children who may have Klippel-Feil syndrome, Caput succedaneum, surgical trauma, and prolonged labor, may also get EPDS.[2]

It not only affect the scalp but can localize other body areas such as the face and extremities.

EPDS may look like a fungal or a bacterial infection, but the microorganisms upon culturing represent secondary colonization, rather than a primary infection.[3]

Triggers and predisposing factors

- Herpes zoster
- Trauma and androgenetic alopecia
- Contact dermatitis[1]
- Actinic damaged scalp (due to impaired mechanism of wound healing)[4]

Pathogenesis of EPDS

The pathogenesis of EPDS is not completely understood. One of the hypothesis include:

Pathogenesis could be an autoimmune response toward the hair follicles induced by trauma with subsequent chronic inflammation and scarring.[5] This hypothesis is made based on the association of EPDS with other autoimmune disorders and its responsiveness to anti-inflammatory drugs.[6]

Immunosenescence is a new concept that describes the decline in efficiency of immune system with aging, leading to increased susceptibility to pathological conditions related to inflammation or autoreactivity.[7]

Diagnosis of EPDS

The diagnosis of EPDS requires a high clinical suspicion with in the clinical context. Condition that mimics EPDS should be ruled out. These mimickers include:

- Skin tumors (NMSC)
- Fungal/bacterial infections
- Bullous dermatosis
- Folliculitis decalvans

The gold standard for the diagnosis of EPDS is the skin biopsy. Histologically, different features of EPDS are distinguishable according to the stage at diagnosis, Figure 1.[1]

Clinical features

EPDS is a chronic inflammatory dermatosis that result in cicatricial alopecia, if left untreated. The lesions are generally not painful and pruritus is rare. Despite topical anti-inflammatory treatment the affected area undergoes a continuous cycle of healing and recurrence. The skin becomes atrophic and new pustules and erosions arise at the periphery perpetuating the disease. The clinical features of erosive pustular dermatosis of the scalp are provided in Figure 2. A clinic-pathological correlation is a must due to the non-specific histologic features.[8]

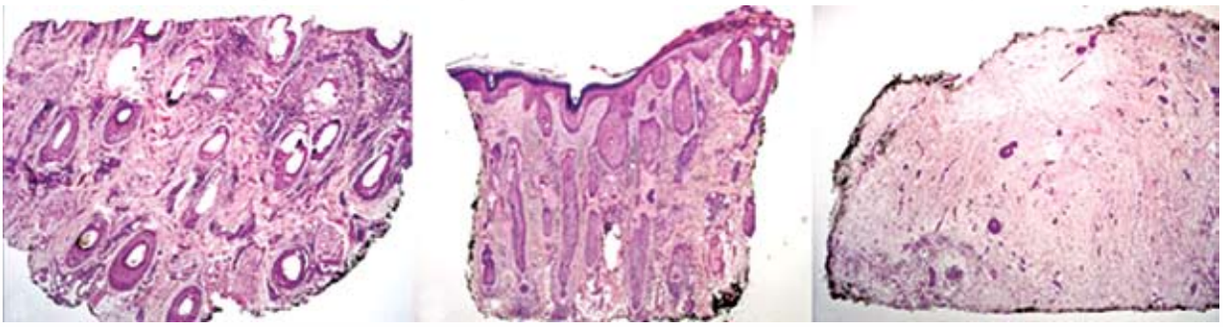
Trichoscopy of EPDS

Only a few studies have reported that trichoscopy of EPDS is reported only in few papers, where the most important finding is skin atrophy with the visualization of the hair bulbs. Other factors observed in trichoscopy are:[8]

- Follicular ostia
- Enlarged dermal vessels
- Perifollicular serous or brown-gray hyperpigmentation
- Pilli torti and broken hair shaft
- Tufted hairs or black crusts or yellow thick exudate

Trichoscopy is very useful in the diagnosis of EPDS in assisting the clinicians in differentiating

Figure 1. Histopathologic features of erosive pustular dermatosis of the scalp



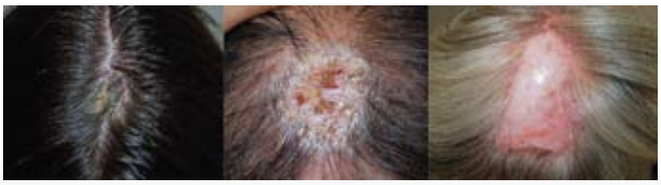
Early-onset EPDS: Psoriasiform hyperplasia of the epidermis, mixed inflammatory infiltrate consisting of neutrophils, lymphocytes, and plasmocytes, and mild fibrosis in the papillary dermis. An increased number of catagen follicles

Intermediate-onset EPDS: extensive fibrosis, absence of sebaceous glands, reduced number of terminal, miniaturized anagen follicles, moderate mixed inflammatory infiltrate, fibroplasia around the follicles at the isthmus level, and tufted folliculitis in the papillary mid

Late-EPDS: Absence of hair follicles and sebaceous glands with a slight mix of inflammatory infiltrate and a diffuse and severe fibrosis in the dermis associated with sclerotic streamers replacing the hair follicles

EPDS from other scalp disorders. The trichoscopic features of EPDS at different stages is given in Figure 3.[8]

Figure 2. Clinical features of erosive pustular dermatosis of the scalp



Erythematous scalp with serous or hemorrhagic crusts is present; in long-standing stage

Diffusely thinned and atrophic scalp (only detectable features) in intermediate stage

Erosions and scales at the center or in an active area and atrophy becomes evident

Figure 3. Trichoscopic features of EPDS at different stages

Perifollicular serous and perifollicular hyperkeratosis, erythematous scalp with enlarged dermal vessels, patchy brown-gray skin hyperpigmentation

Yellow scales in the affected areas and dilated vessels.

Absence of follicular ostia and visualization of hair bulb through the atrophic skin.

Treatment options for EPDS

The presently available treatment options include:

- Topical high-potency corticosteroids and tacrolimus: Are the first-line of the EPDS treatment. It is found to be effective and safe, despite relapses after discontinuation.[2]
- Oral steroids and dapsone are the second-line treatments of EPDS.[9]
- Photodynamic therapy (PTD) with methylaminolevulate cream: Another possible option which has been found to be effective in combination with previously applied topical corticosteroids. It increases the responsiveness to anti-inflammatory drugs. However, it use is controversial due to its ability to be a trigger other diseases.[8]
- Fractional ablative laser (erbium yttrium aluminum garnet [YAG]) therapy is a newer option but its use is unclear.

Conclusion

EPDS is considered to be a rare condition and its pathogenesis has remained unclear. Although researches have suggested that trauma to the skin may cause autoimmune-reaction acts against skin structures resulting in a secondary inflammatory reaction. The treatment of EPDS is difficult as it may be misdiagnosed and treated inappropriately.

It is important to care for the scalp that is prone to the disease condition with avoidance precipitating factors such as trauma. If there is development of actinic damage the treatment may be for long-term treatment. In the late stages of disease, there is no treatment at present able to reverse the cicatricle alopecia because of the inflammatory process underlying this condition. Even surgery is not recommended to reduce the alopecic area.

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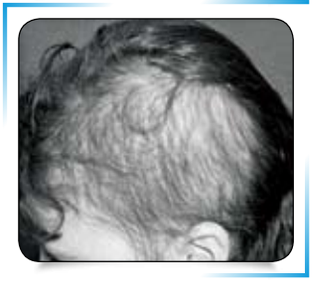
14th International Conference on Cosmetology and Trichology

March 12–13, 2020; Miami, United States

The International Conference on Cosmetology and Trichology brings together the leading academic scientists, researchers, and research scholars under one big roof to exchange and share their challenges, research results, and pondering points of clinical practice in all the aspects of Cosmetology and Trichology. This conference provides a premier interdisciplinary platform for researchers, practitioners, and, educators to present and discuss the most recent innovations, concerns, and trends as well as challenges encountered in their practice and the respective solutions adopted in the fields of Cosmetology and Trichology.

This conference is held in Miami, USA. The Miami metropolitan area (the Greater Miami Area or South Florida) is the 7th largest metropolitan area in the United States and 72nd largest metropolitan area in the world. Miami is famous for its parades and festivals such as the Carnival Miami, Miami Beach pride, winter fest boat parade, winter party festival, white party, and many more other similar festivals. Miami is also famous for its glamorous nightlife. There are few other places which are a must-see such as zoos and aquariums, safari adventure, and the Miami sea aquarium.

Photo Quiz



1. **A 23-year-old woman working as a marketing executive presented with rapidly progressive hair loss that became apparent over a period of two weeks. She noticed her hair fall after a successful and stressful business event. What is the most likely diagnosis?**

   A. Alopecia totalis B. Alopecia universalis

   C. Diffuse alopecia areata D. Androgenetic alopecia

2. **A 32-year-old male engineer working as a construction head presented with excessive hair loss especially from the top of the head and a gradually developing bald patch in the middle of the scalp. What is the most likely diagnosis?**

   A. Telogen effluvium B. Androgenic alopecia

   C. Trichotillomania D. Scarring alopecia

3. **A 4-year old child presented with poor hair quality, patchy hair thinning, and localized bald patches in his scalp for 3 months. What is the most likely diagnosis?**

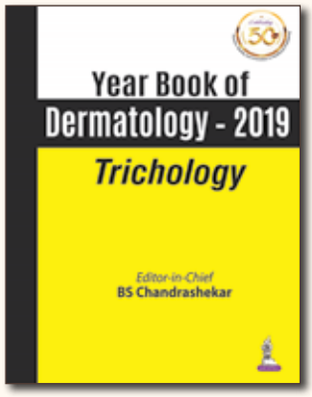
   A. Traction alopecia B. Alopecia mucinosa

   C. Tinea capitis D. Trichotillomania

Answers: 1: C, 2: B, 3: D

Year Book of Dermatology 2019 Trichology

BOOK REVIEW



Author:
BS Chandrashekar

Publisher:
Jaypee Brothers, Medical Publishers Pvt. Limited, September 2019.

Number of pages: 212

This book is well-organized with articles on different sections that focus on the basics, clinical, therapeutic, diagnostic, and surgical aspects in the field of trichology. The selection of the articles is structured based on the excellence and advantage of the researcher and the practicing clinical dermatologist. After a thorough peer review of journals published from more than 250 publications (2018 and 2019) in the recent 12-months period, 118 articles have been meticulously segregated and included in the book focusing the recent advances in the field of trichology. Each article in the book includes key messages showing the summary of the article and the clinically relevant information or experience by the reviewers.

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