INTRODUCTION

Alopecia, a frequently observed problem, severely impacts the quality of life of patients and is often associated with loss of confidence and low self-esteem.[1] Several conditions are associated with hair loss, namely telogen effluvium (TE), anagen effluvium, diffuse type of alopecia areata, female pattern hair loss, hair shaft abnormalities, loose anagen hair syndrome, and congenital atrichia or hypotrichosis.[2] An Indian study that aimed at determining causes of diffuse hair loss in women reported that diffuse hair loss is multifactorial in origin and majority of such patients suffered from TE.[3]
NEED FOR STANDARDIZATION OF MANAGEMENT

Although hair loss is a common complaint, an etiopathogenesis-based, logical, and comprehensive classification has not been developed. Currently, alopecia is classified on the basis of abnormalities of hair growth cycle or follicular damage.\(^4\)

The actual prevalence rate of TE is not reported since most cases are subclinical in nature. Further, since women get more alarmed by hair fall and promptly seek treatment, they tend to be over-represented. However, both genders can suffer from this condition if triggering factors are present. The clinical research has enabled a better understanding, especially in terms of control of hair follicle cycling.\(^4\) This consensus paper has been developed taking opinions of all the participating panelists in response to the need for standardization of diagnostic and therapeutic approaches from the Indian perspective.

A group of 120 dermatologists, indulged in focused group discussions across 11 Indian cities – brainstorming on etiopathogenesis, clinical features, diagnostics, and therapeutics of TE. Every discussion was recorded and transcribed. The compiled manuscript was critically reviewed by the moderators. This paper is a synopsis of the discussion covering all aspects of the management of TE.

DEFINING THE PROBLEM

TE can be defined as 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; however, it is self-limiting and is characterized by premature termination of the anagen phase of the hair follicular cycle and predominance of the telogen phase. It is difficult to identify triggering factors in nearly one-third of the cases.\(^5\) Table 1 can act as a ready reckoner for diagnosis.

RECALLING THE PATHOLOGY

Telogen hair shedding has been attributed to five functional alterations in the hair cycle, namely immediate anagen release, delayed anagen release, short anagen syndrome, immediate telogen release, and delayed telogen release.\(^6\)

HISTORY

Understanding a detailed history is essential for evaluating patients with TE. The subtype of TE and probable triggering events before the start of hair loss must be identified. Specific queries must cover the patient’s chief complaint, associated complaints, personal and family history, systemic symptoms, nutritional status, medications, psychosocial state, and usage of cosmetics/hair-care products. Symptoms such as acne, hirsutism, or irregular menstrual cycles indicate excess androgen, whereas a strict vegetarian diet or heavy menses are suggestive of iron deficiency anemia.\(^7\)

The panel emphasized that understanding the duration of hair fall can help classify the condition as acute (hair loss <6 months) or chronic (hair loss >6 months). Any change of residence must be confirmed since environmental factors, and quality of water may be a triggering factor. A recent history of diseases or any surgery could also help identify the causation. For female patients, menorrhagia and puerperal disorders should be considered. Current medications or those that have been taken in the recent past must be noted, especially watching out for isotretinoin, steroids, beta blockers, phenytoin, contraceptives, and anticancer drugs. Details about prior treatment with minoxidil and its initiation and withdrawal should be noted. Stressors of all kinds – physical, psychological, nutritional, or medicinal – must be noted. Since emotional trauma has been found to be an important trigger, inquiring about the personal lives of patients to some extent may be significant. Sleep disturbances should be looked for. Nutritional deficiencies need to be ruled out.

| Table 1: Causes of telogen effluvium\(^5\) |
|---|
| Causes |
| Physiological causes | Initial stage of AGA, physiological effluvium in the newborn, telogen gravidarum |
| Physical/emotional stress | Severe febrile illnesses or infections, malnutrition/malabsorption, micronutrient deficiencies, thyroid disorders, acrodermatitis enteropathica, severe trauma (major surgeries or accidents), chronic illnesses (autoimmune disorders, malignancies, organ failure), overwhelming emotional stress, unknown cause (idiopathic) |
| Drugs | Anticoagulants, anticonvulsants, antithyroid agents, beta blockers, high-dose contraceptive pills, heavy metals, hormone replacement therapy, hypolipidemic drugs, oral retinoids |

AGA – Androgenetic alopecia
Consensus key point 1

“All patients with diffuse hair loss must be subjected to a detailed history-taking exercise which involves asking direct questions about the 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

The clinical examination of the scalp indicates degree and pattern of hair loss and provides evidence of erythema, inflammation, or scaling, if present. This should be followed by assessment of hair shafts in terms of parameters such as breakage, diameter, and length. The hair-pull test must be performed on all patients with hair loss. Counting shed hair daily may be useful, where the patient is asked to collect shed hair in dated envelopes at a specific time every day at home. Collections of more than 100 hairs per day indicate TE. Further, microscopy can help identify them as anagen or telogen hairs and may indicate specific nutritional deficiencies. Unnecessary skin biopsies can be avoided by using trichoscopy. While the panelists were in agreement about these examination modalities, they were more specific about their methodologies.

As per their recommendations, hair should not be washed and preferably not combed for at least 3–4 days before the hair pull test. After examining occipital, frontal, and parietal areas of the scalp, gentle traction must be applied to a bunch of about 30–40 hairs, and the number of shed hairs from different areas should be counted. A positive test for all areas of the scalp indicates TE. Phototrichograms should be obtained after selecting a unit of scalp area in square centimeters, and proportion of anagen and telogen hairs should be calculated. In TE, the anagen-to-telogen ratio is reversed. Hair combing test was also recommended where patients should be asked to use shampoo and after five consecutive days of shampooing, hair must be combed to assess hair fall.

Consensus key point 2

“Clinical examination of scalp, hair-pull test, trichoscopy, and hair combing test are four essential tools that can enable a quick diagnosis in patients with hair loss.”

Investigations

The underlying causes of diffuse telogen hair loss can be identified by laboratory tests; therefore, their role in diagnosis cannot be underplayed. The panel categorized investigations as basic and advanced. The following basic investigation parameters can be ordered for every patient with diffuse hair loss:
- Complete blood count
- Routine urine examination
- Serum vitamin D
- Thyroid function tests.

Examples of advanced diagnostic tests and their implications are given below:
- Serum calcium to diagnose hypocalcemia
- Vitamin B<sub>12</sub> (common in vegetarians and individuals with pernicious anemia, atrophic gastritis, malabsorption syndromes, and/or autoimmune disorders)
- Serum proteins to identify protein energy malnutrition
- Serum zinc to detect deficiency
- Hormonal assays to distinguish between TE and androgenetic alopecia (AGA)
- Iron profile (total iron binding capacity and serum ferritin level) to diagnose iron deficiency
- Serum anti-mullerian hormone and testosterone levels for mixed alopecia
- Anti-dsDNA antibodies when autoimmune diseases are suspected.

Consensus key point 3

“While basic investigations can be ordered for all patients with diffuse hair loss, advanced tests must be reserved for patients whose history and examination findings are suggestive of a specific disease or deficiency.”

Investigation

The panel mentioned that an overall decrease in density and volume of hair helps in its diagnosis. In TE, diffuse hair loss from 4 to 5 areas is usually observed and global rather than patterned hair loss is seen. Patients who complain of hair fall should be asked to bring a collection of hair shed during the whole week, since most patients do not know that hair fall count of 100/day is normal. The panelists advocated use of modified Sinclair scale for quantitative estimation of hair loss, especially in females, and emphasized on checking every region of the scalp. The expert panel came up with important differentiating features as shown in Table 2.
MEDICAL MANAGEMENT OF TELOGEN EFFLUVIUM

Patient education

Patient education is an integral part of TE management. In acute TE, patients should be informed that identification and removal of the trigger will resolve the problem. Hair shedding will be short-lived and controlled without medications. Patients should be explained that hair fall can continue for up to 6 months, albeit to a lesser extent. Although re-growth can be observed within a few months of trigger removal, significant growth can take more than a year. Nutritional requirements must be considered, and any drug suspected of being a contributing factor must be discontinued or changed for at least 3 months. The complexity of chronic TE due to several sequential or repetitive triggers must also be explained. Underlying issues must be treated appropriately.

Consensus key point 4

“Patient education plays an important role in the management of patients with TE and can go a long way in allaying their anxieties, especially with respect to going bald. The normal hair cycle should be explained so that they do not have unreasonable expectations.”

The panelists seconded the role of management by addressing that medical therapy is largely supportive for the treatment of TE and no treatment is needed if the underlying cause is addressed. Serum levels of vitamin B<sub>12</sub>, D<sub>3</sub>, and iron must be determined before deciding the treatment protocol. The importance of healthy food habits should be explained and if required, amino acids, vitamin B complex, zinc, and proteins may be prescribed. Topical peptides, found to be beneficial in TE, may be prescribed. Nutrients may be given in a cyclical manner, although evidence to support the same is lacking.

Consensus key point 5

“Amino acids play an important role in the treatment of TE. Topical peptides may also be prescribed which are found to be beneficial in the management of this condition.”

TREATMENT

Minoxidil

Minoxidil, a piperidino-pyrimidine derivative (2,4-diamino-6 piperidino-pyrimidine-3-oxide), is believed to open potassium channels, leading to cell membrane hyperpolarization. It widens blood vessels, thereby allowing larger amounts of blood, nutrients, and oxygen to reach the hair follicles. Topical minoxidil is believed to shorten the telogen phase of the hair cycle, thereby compelling resting hair follicles to prematurely enter the anagen phase. It possibly extends the anagen phase and also increases the size of hair follicles. It has variable effects on growth by delaying keratinocytic senescence and stimulating/inhibiting proliferation of epithelial and fibroblastic cells. It inhibits collagen and prostacyclin production, while stimulating synthesis of prostaglandin E<sub>2</sub> and vascular endothelial growth factor.

Consensus key point 6

“Minoxidil does have a role to play in the management of chronic TE. Its use in acute TE is not recommended. A combination therapy of minoxidil and a peptide could produce better results in comparison to either of them being used as a monotherapy.”

According to the panelists, minoxidil has a place in TE management, since it is a vasodilator that positively influences anagen phase of the hair cycle. However, it is never recommended for patients with active TE, but can be prescribed for chronic TE. While 2% minoxidil is used for females, a 5% preparation may be used for males. Patient counseling is important since minoxidil therapy can induce hair loss in the initial stage when the telogen hairs shed off and new hair growth occurs. Combination
therapy using minoxidil along with a peptide could produce better results. The panel opined that serum-based peptides, caffeinivated preparations, and topical botanicals may prove aid in the treatment of TE. For offering symptomatic relief to patients with an inflamed scalp, flakes at the base of the hair, or minoxidil-induced irritation, a short course (for up to 5 days) of topical mild steroids can be prescribed.

**Shampoos**

The panel opined that shampoos do not have a direct role in the treatment. The use of a mild shampoo without sodium lauryl sulfate may be considered. Shampoos with anti-inflammatory agents should be prescribed only if needed in case of dandruff or seborrheic dermatitis.

**Consensus key point 7**

“Shampoos do not have a direct role in treatment, but using the right shampoo is important. When shampooing their hair, patients should be careful about washing it off thoroughly. Further, vigorous application must be avoided as this can damage the hair.”

**Hair oils**

The panel noted that hair oil does not play an important role in the management of TE. Oil act as a good prewash conditioner and protect the cuticles to make hair smooth and shiny. However, too much oil can have adverse effects. The application of hair oil for 30–60 min is sufficient. Coconut oil is absorbed in the cuticle layer and may be used as a conditioning agent.

**Consensus key point 8**

“Hair oil does not play an important role in the management of TE; however, they can be used as they protect the cuticles and act as a good prewash. The application of oil should be restricted to the shafts, and not the roots of hair.”

**Nutritional supplements**

Nutrients such as iron, zinc, selenium, niacin, folic acid, biotin, Vitamin A, Vitamin D, Vitamin E, fatty acids, and amino acids play a key role in promoting hair growth and maintenance of hair structure. Studies have reported an association between nutritional deficiency and TE. However, the ideal range of micronutrient levels for prevention/correction of hair loss is unclear. When protein deficiency is identified due to some metabolic or dietary causes (protein consumption is <0.8 g/kg), protein supplementation is required.

The panel agreed that nutritional supplements play an important role in the treatment of TE. Amino acids, zinc, calcium, iron, copper, selenium, and folic acid supplements can be considered. Soya protein, omega-3 fatty acids, and green tea extract may be recommended, but evidence about their efficacy is lacking. Protein-rich diet must be recommended for patients with established protein deficiency. Supplementation by protein powders may be considered in protein-deficient patients. They added that animal sources of proteins are superior to plant sources, and sulfur-containing amino acids are specifically good for hair growth. Doctors should ask more questions during history-taking about protein powder(s) that the patients have been consuming, because some of the products could contain anabolic steroids.

**Consensus key point 9**

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

**Biotin**

Biotin, also known as vitamin B7 or vitamin H, is a co-enzyme for carboxylase enzymes, it plays a role in the metabolism of glucose, branched-chain amino acids, and fatty acids. The prevalence of biotin deficiency in patients with hair loss is not well known and its utility in treating hair loss (not associated with biotin metabolism disturbances or deficiency) has not been established.

The panel opined that biotin was useful at recommended dietary allowance (RDA) limits of 30 μg. Since it is water-soluble, excess intake is usually excreted. No evidence is available supporting higher doses of biotin for treatment of hair loss, unless the patient has biotin deficiency or a history of consuming large quantities of raw eggs.

**Consensus key point 10**

“Biotin supplementation may be useful at RDA limits of 30 μg, but there is no evidence to suggest that higher doses of biotin are beneficial in the treatment of hair loss unless a deficiency is present.”

**Platelet-rich plasma**

Platelet-rich plasma (PRP) is a novel treatment modality for hair loss, where an autologous concentration of platelets contained within a small volume of plasma is
used to promote rejuvenation of hair follicles, owing to the presence of various growth factors and cellular adhesion molecules.[14]

The panelists affirmed that selecting appropriate candidate is crucial since PRP does not have an established role in TE. The unnecessary use of PRP should be avoided. Although treatment outcomes of PRP are better in AGA, results vary depending on procedural methodologies and technical processes involved.

Consensus key point 11

“PRP therapy has no place in the management of acute TE but may be of some use in chronic TE; albeit, the unnecessary use of PRP should be avoided in patients with TE, considering that the results are variable.”

TACKLING COMPLIANCE ISSUES

A major hurdle in treatment of hair loss is noncompliance to treatment. Several barriers have been identified for patient noncompliance which indicate an urgent need to develop better physician-patient interactions.[15] Suggested strategies include recommending the most appropriate therapy, minimizing the number of medications (by choosing combination preparations) and reducing frequency of their dosing (by use of controlled release products); prescribing medicines with better safety and tolerability and teaching patients to cope with side-effects in case there are no alternative options.[15]

The panel agreed on the importance of patient counseling for reducing noncompliance. It is important to educate patients about TE and its course of treatment, and motivate them about the results which may take 3–4 months. Using educational videos or animations is advisable. Capturing photographs at baseline and during follow-ups can help in motivating patients. Demonstrating good results with trichoscopy and hair analysis can also promote patient compliance.

Consensus key point 12

Patient counseling plays an important role in the treatment of TE. It is necessary to educate the patients about the condition and its treatment. Furthermore, assuring patients about the progress of the treatment and its results (which may take at least 3–4 months) would help in reducing the noncompliance issues.

CONCLUSION

This article underlines the evidence-based treatment and management of TE contributed by Indian experts in the field of dermatology.

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