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Management of Telogen Effluvium: An Overview

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Background:Telogen effluvium (TE) is a diffuse, non-scarring hair loss characterized by shortening of the anagen phase and precipitation of the telogen phase that leads to consequent decreasing of the total hair volume. Identifying and correcting the underlying cause is the most important component in management. Minoxidil is mainly used for the treatment of male and female androgenetic alopecia but it has a place in TE management. The goal of mesotherapy in management of some types of hair disorder is to restore and increase local microcirculation, provide nutritional input, slow down the programmed process of follicular involution.
Keywords:Telogen Effluvium (TE), Minoxidil.

Telogen effluvium (TE) is a diffuse, non-scarring hair loss characterized by shortening of the anagen phase and precipitation of the telogen phase, that leads to consequent decreasing of the total hair volume. Its epidemiology is unclear and data about it is limited. It is one of the most common forms of diffuse, non-scarring hair loss for which patients present for clinical evaluation. Telogen effluvium doesn't appear to have a predilection for particular racial or ethnic groups (1).

Telogen effluvium is divided into acute and chronic variants. Acute telogen effluvium may occur at both sex and at any age, including infants and children. It is more common in female than male (**2**). Chronic telogen effluvium is less common than the acute variant and can further be subdivided into chronic and chronic-repetitive presentations that affects mainly female between the ages of 30 and 60 years (**3**).

Telogen effluvium may be primary that occur without obvious causes or secondary to many trigger factors. Causes of secondary TE can be listed in the following table (**4**).

Table (1):Causes of Telogen Effluvium(4).

Infection	Malaria, typhoid, HIV, syphilis and tuberculosis.
Physiological conditions	Telogen of newborn and post partum.

Stress	Psychological, difficult labour.
Severe illness	Prolonged febrile illness, major injury and post operative.
Drugs	Anticonvulsant, antithyroid, oral contraceptive, captopril, oral retinoids, amphetamins, hypolipidemic drugs, betablockers, , and heavy metals
Endocrinal diseases	Hypo and hyperthyroidism.
Organ failure	Renal and hepatic failure.
Local application	Hair dye application
Nutrition	Iron deficiency anemia, Acrodermatitis enteropatheca, Zinc deficiency, Starvation, Crash diet, Malnutrition (Marathmus and Koashiorokor)
Miscellaneous	Systemic lupus erythematosis, short anagen syndrome and heamorrhage.

Diagnosis:

Clinically, patients with TE suffer from diffuse hair loss that mainly occurs 2-3 months after the triggering event**.** The diffuse loss may produce thinning of hair all over the scalp and frequently manifest with bitemporal recession. In most cases hair loss doesn't exceed half of the scalp hair **(3).**

A- Patient history: It is the most important key in the diagnosis of telogen effluvium. It can confirm a course of hair loss that consistent with telogen effluvium and reveal the underlying causes of telogen effluvium. It is also useful for ruling out other causes of hair loss. In older adult patients or other patients with multiple medical disorders, the cause of telogen effluvium may be multifactorial **(5)**.

B- Examination: The second line in diagnosis is dermatological examination of skin, hair and nail. The detection of scales, inflammation, pustules, and scarring in scalp are not features of isolated telogen effluvium. Such findings suggests the presence of a another scalp disorder. Abnormalities of the hair shaft also are not features of telogen effluvium **(6).**

C- Dermoscopy can be used in diagnosis of telogen effluvium. Acute telogen effluvium may demonstrate empty follicles and many short regrowing hairs of normal thickness (>0.03 mm). In chronic TE, there is increased proportion of single-hair follicular units, no significant difference between frontal and occipital areas and no brown perifolliculer discoloration. Dermoscopy may help to distinguish chronic telogen effluvium from female androgenetic alopecia in which there is greater hair diameter variability **(7).**

D- Diagnostic tests: many tests can be performed as part of the physical examination in patients who present with clinical findings suggestive of telogen effluvium.

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Hair pull test: The test aids in the recognition of active hair shedding. Grasp 50 to 60 hair fibers close to the skin surface and tug the hairs from the proximal to distal ends. Repeat the pull test in several areas of the scalp. The extraction of more than ten hair fibers is abnormal and False-negative hair pull test can occur, if the patient has shampooed or vigorously groomed the hair on the day of examination and a false-positive hair pull test may be noted if the patient has not shampooed or combed hair for several days (8).

-**Trichogram (hair pluck test):** It is a method of hair examination in which the patient should not wash or cosmetically treat hair for 4–5 days and then 50–100 hairs are usually plucked using a tightly closing epilation forceps. The epilated hair roots are then placed on a glass slide and a mounting medium should be added before arranging the hair roots side-by-side and then covering the hair mount with a cover slide. light microscopy is usually used to examine the hair mount and the individual hair root types are specified in percentage values and proportion of anagen and telogen hair should be calculated. Hair pluck sample in TE is abnormal as it shows that anagen-to-telogen ratio is reversed, and greater than 25% of hair is in telogen phase (9).

-**Wash test** also used in TE examination. It is probably considered the best method to adopt. In wash test, the patient is instructed to wash hair after 5 days of last shampoo, in a sink covered its drain by gauze. The hair entrapped in the gauze is then counted **(10).**

E- Laboratory tests should be performed to detect the proper cause of TE, which includes complete blood count, urine analysis, serum ferritin, serum zinc level and T3, T4, thyroid stimulating hormone (TSH) (11).

F- Scalp biopsy is usually not needed. Biopsy of TE is normal except for an increase in telogen follicles (normal telogen counts 6-13%). Biopsy in chronic TE resembles normal scalp, but shows an anagen : telogen ratio of 8:1 compared with the ratio of 14:1 on normal scalp biopsies. Proportion of telogen follicles more than 15% suggests TE but more than 25% is definitive feature. It can be used to differentiate TE and other causes of hair loss as alopecia areata and female pattern hair loss **(11).**

G- Photographs are less helpful in TE than in other causes of non scarring alopecia. However, standardized photographs of midline part, central, and occipital scalp at the time of diagnosis provide a baseline assessment and a frame of reference for future visits. Since hair grows 0.5 to 1 cm a month, photographic follow-up will also allow for assessment of regrowth **(12).**

Differential diagnosis:

A variety of non-scarring hair and scalp disorders share clinical features with TE.

●**Diffuse alopecia areata**: Diffuse alopecia areata is an uncommon form of alopecia areata that is characterized by the diffuse loss of scalp hair, resulting in the

appearance of generalized hair thinning Similar to anagen effluvium, exclamation point hair may be present and performance of the hair pull test may reveal dystrophic anagen hair. A biopsy here revealing an inflammatory infiltrate that isn't present in TE **(13).**

●**Androgenetic alopecia**: It is the commonest type of progressive hair loss. Clinical examination is needed to detect differences between androgenetic alopecia and TE. in AGA, hair loss is mainly from temporal region, there is peripiler halo and great variation in hair diameter (14).

●**Anagen effluvium**: Anagen effluvium is an acute loss of anagen hair fibers secondary to chemotherapy or toxin exposure and represents acute loss of more than 80% of the scalp hair. Exclamation point hairs (short, 1 to 3mm hairs with a tapered base) that result from dystrophic hair growth are a common finding(15).

●**Loose anagen syndrome**: Loose anagen syndrome is a rare non-scarring hair loss disorder that affect young children particularly blond hair females, manifests with easily extracted anagen hairs from the scalp. Characteristically, examination of shed hairs reveals anagen hairs with ruffled cuticles (16).

●**Structural hair disorders**: A variety of structural hair disorders cause weakening of the hair shaft lead to easily fractured hair. Unlike telogen effluvium in which hair is shed from the follicle, these conditions result in increased breakage of hair. (17).

Management:

A- First-line interventions: Telogen effluvium is usually a reactive and reversible disorder. Therefore, identifying and correcting the underlying cause is the most important component in management. Potential therapeutic options include inhibition of catagen, inhibition of exogen and inducion of anagen. Presently there are no FDA approved highly efficient catagen inhibitors or anagen inducers **(18).**

Treatment of an associated illness or dietary deficiency can be implemented. If drug-induced TE is suspected, the suspect drug must be discontinued for at least three months to determine whether hair loss will improve without therapy **(19).**

B- Psychologic support: Hair loss can have a profound psychosocial impact, which may seem out of proportion to the degree of hair loss. Techniques for camouflaging hair loss and psychologic support are useful measures for managing the psychosocial effects of hair loss. In addition, education about the hair growth cycle and the expected course of TE may help to reassure patients. Follow-up evaluations can be helpful for reassuring the patient with persistent TE **(20).**

C- Topical minoxidil: Minoxidil is mainly used for the treatment of male and female androgenetic alopecia but it has a place in TE management, since it is a vasodilator that widens blood vessels, thereby allowing larger amounts of blood, nutrients, and oxygen to reach the hair follicles that positively influences anagen phase of the hair cycle.It is recommended for patients with chronic TE not for active TE. Its

concentration either 2% minoxidil that is used for females and 5% preparation can be used for males. Patient counseling is important since minoxidil therapy must be continued to maintain benefit. Occasionally, it increases hair shedding within the first two to eight weeks of treatment, This is thought to result from the release of hair from telogen follicles as they are stimulated by minoxidil to transition from telogen to anagen (21,22).

D- Supplements: patients in whom iron deficiency anemia is detected should receive iron supplementation for treatment of TE. Supplementing iron intake to reach ferritin levels greater than 30, 40, or 70 mcg/L in patients with hair loss has been recommended by some experts based upon a belief that treatment for hair loss is optimized at greater serum ferritin levels. It was believed that adding the diet with vitamin C and vit.D may improve TE (22).

E- Mesotherapy: It is a common treatment line for TE. It is non surgical medical treatment, that employs multiple injections of pharmaceutical medications, plant extracts, multivitamins and other ingradients in to intradermal layer(23).It represents a variety of minimally invasive techniques in which medications are directly injected into the scalp in order to improve alopecia and hair growth. This therapy is effective at some certain forms of alopecia such as TE and androgenetic alopecias, but not effective at scarring alopecias. In hair mesotherapy, there is lack of mixture and application scheme whose effectiveness has been proved scientifically **(24)**.

The goal of mesotherapy in management of some types of hair disorder is to restore and increase local microcirculation, provide nutritional input, slow down the programmed process of follicular involution, stimulate the hair's environment through needling and complement other treatments **(25)**.

F- Platelet-rich plasma (PRP): Platelet-rich plasmais 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. PRP therapy has no place in the management of acute TE but may be of some use in chronic TE. Treatment outcomes of PRP are better in AGA, results vary depending on procedural methodologies and technical processes involved **(26)**.

G- Laser: Low level laser can be used in treatment of TE, there is anecdotal evidence to suggest that light in the wavelength of 650–900 nm, but atsignificantly reduced powers of 5 mw can enhancehair growth (**27**) .

H- Botulinum toxin(A):Botulinum toxin (A) is a drug made from a toxin produced by the bacterium Clostridium botulinum, It has been used in management of some hair falling disorders. The proposed mechanism of action of botulinum toxin (A) is the relaxation of muscles, which reduces pressure on the musculocutaneous and perforating vasculature, thereby potentially increasing the blood supply and

transcutaneous pO_2. This increased blood flow can also lead to washing out of accumulated dihydrotestosterone(DHT), thereby reducing the signal for minituarization of hair follicle **(28)**.

I- Cosmetic measures: Consultation with a cosmetologist can help patients to identify measures that minimize the cosmetic effects of hair loss. Hair styling techniques, hair coloring, scalp coloring, and hair prostheses can be useful for camouflaging hair loss. Hair transplantation is not indicated for patients with TE **(28).**

Prognosis: The prognosis of TE is dependent on the ability to remove or treat causative factors. If the underlying cause spontaneously resolves or can be eliminated, the course of TE will be self-limited. In the other hand, if the underlying causes cannot be identified or removed, shedding may persist for years **(28)**.

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