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Acute Versus Chronic Telogen Hair Loss: A Review of the Knowledge and Recent Facts

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ABSTRACT

Telogen effluvium (TE) is defined as non-scarring diffuse scalp hair loss, varying from 150 to more than 400 hair falls per day. TE is a very important cosmetic query that cannot be ignored, especially for women. Different kinds of search literature were reviewed, regarding the mechanism of hair shedding, causes, clinical types, the role of diagnostic tools, and potential treatment modalities. The deviations in the follicular cycle period led to telogen hair loss. The disease exists in an acute or chronic state, sometimes associated with trichodynia. Diagnostic evaluations include the hair pull test, the clip test, trichoscopy, the modified wash test, and less frequent histopathological studies. Almost all cases of acute disorders can be cured by eliminating the identified causative factors. As a result, for most of them, no exact therapy is necessary. The chronic form is considered a challenging treatment goal for physicians, and many modes of treatment for telogen effluvium have been used with different benefits. Newly approved causes include post-hair transplantation, vitamin D deficiency, and coronavirus disease-19. Until now, limited treatment choices with variable benefits have been offered. This narrative review article evaluates the acute and chronic presentation, available investigations, and treatment options for telogen hair loss.

Keywords: Hair loss; Telogen effluvium; Alopecia; Follicular cycle

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INTRODUCTION

Telogen effluvium (TE) was initially described by Kligman in 1961[effluvium= outflow, rapid]. It is characterized by excessive loss of telogen club hair, which could occur after a disruption of the hair cycle [1]. Normally, hair follicles of the scalp of adult humans go through cyclical phases organized in a haphazard mosaic pattern, including the anagen (a growing phase), which comprises about 90-95% of hair follicles that continue between 2–8 years; the catagen (an involuting phase) includes only 1% and lasts from 3 to 6 weeks; and the telogen (a resting phase) involves 5–10%, between 2–3 months until the formation of club hair (Figure 1) [1, 2]. Each single hair replacement on the scalp occurs every 3-5 years, with individual hair follicles experiencing 10-30 such cycles in a lifetime [3, 4].

Furthermore, numerous reports have introduced the concept of teloptosis (exogen phase), characterized by a loss of binding between club hair cells and surrounding epithelial cells. This loss leads to the termination of the hair cycle and the removal of depigmented club hair from its follicle [5]. The exogen phase probably occurs due to the effect of circulating stress hormones or cytokines that result from an alteration of the metabolic pathway [6]. The follicular mitosis and growth were arrested prematurely by different suggested mechanisms, including nutritional insufficiencies, cytostatic effects of the drug, and/or the probable lymphocytotoxin effect [7].

Hair loss disorders are heterogeneous, comprising diseases with separate pathophysiology and clinical characteristics [8–10]. TE is defined as non-scarring diffuse scalp hair loss, that varies from 150 to more than 400 hair falls per day [4]. Several hypotheses have proposed regarding the pathophysiology of TE. However, there are five various functional kinds of TE built on fluctuations in certain stages of the follicular cycle [11]. Acute TE is characterized by hair shedding that continues for less than six months and spontaneously resolves with completely new growth of hair [11]. Chronic TE is an idiopathic illness that persists for more than six months [12]. A variety of insults have been proposed as a source of the

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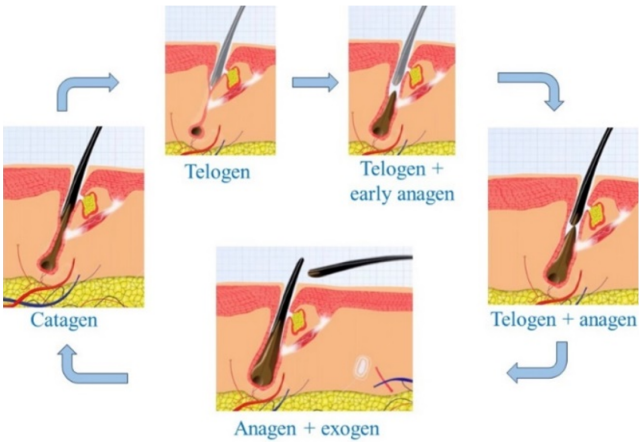

Figure 1. Showing the normal cyclical phases of the hair follicle of an adult human's scalp.

disease, which can be mental, physical, or chemical [4, 13]. Recent studies elucidate diagnostic measures, including trichoscopy and hair pull tests, with evidence-based data [14–16]. Additionally, the other non-invasive diagnostic modalities were novel and in progress [17, 18].

It is significant that the knowledge of hair loss illnesses is strongly growing and thus requires appropriate updates. This descriptive review tries to comprehensively summarize new advances in the understanding of etiopathogenesis and outline the management of illustrative acute vs. chronic telogen effluvium.

MECHANISMS OF HAIR SHEDDING AND THE POSSIBLE TRIGGERS IN TELOGEN EFFLUVIUM

Various physical, mental, or chemical insults can initiate irregularities in the hair cycle, resulting in TE (Table 1). An alternation in the particular stages of the regular follicular cycle is caused by five (3 are correlated to anagen, and 2 are linked to telogen) mechanisms that were proposed by Headington, including [11]:

1. Immediate anagen release: This occurs when follicles enter telogen prematurely and are stimulated to leave the anagen, subsequently increasing telogen hair loss after 2–3 months. The reversal of the insult is followed by a return to the normal cycle [11]. This can be generalized as getting after a severe illness, nutritional deficiency, drug, emotional stress, infection, or other physiologic trauma. The localized TE seen in inflammatory diseases such as psoriasis and seborrheic dermatitis [4, 19–21], as well as post-hair transplantation [22]. Several articles suggested religious headwear and hijab were associated with secondary TE through aggravating underlining seborrheic dermatitis of the scalp [23, 24]. Others recommend that vitamin D deficiency may significantly impact the development of TE [25]. On the other hand, a previous study showed that trans-epidermal ultraviolet light significantly had harmful effects on human hair follicles [26]. Coronavirus disease-19 (COVID-19) was suggested as a new and common cause through immediate anagen release mechanism [27–32].
2. Delayed anagen release: This is owing to the extension of the anagen phase followed by the late, synchronous, and sudden onset of heavy TE [11]. The prolonged anagen phase occurs commonly during pregnancy due to its hormonal effect. Later on, after 3–4 months postpartum, the hormonal drive is removed, and resulting in the shifting of many hair follicles to the telogen phase, also, called telogen gravidarum. Similarly, this occurs after the withdrawal of the contraceptive pill [33, 37]. In addition, many neonates experience that hair follicles in the occipital area are synchronized in utero and remain in the anagen phase until delivery. The onset of the telogen phase is thus delayed until it reaches the term, and hair loss results within 2 to 3 months after labor and is classically present with a major occipital alopecia [34]. Pressure alopecia, band hypotrichosis nevus, and halo scalp ring are important differential diagnosis to be considered.
3. Immediate telogen release: This occurs because of the shortening of the telogen phase, as happens with early treatments such as topical minoxidil treatment, which encourages the shortening of the telogen hair phase and leads to prematurely reentering the anagen phase. Then, activation of the exogen phase occurs, resulting in massive loss of club hairs [11].
4. Short anagen syndrome: It is marked by an idiopathic shortening of the anagen phase, resulting in persistent telogen hair loss [11]. This mechanism is suggested behind most of the cases complaining of chronic TE of unknown etiology, which is considered a failure to grow the hair long, besides mild persistent hair shedding [35].
5. Delayed telogen release: This is due to a prolonged telogen follicular stage and a delayed conversion to the anagen phase. It is believed to cause seasonal telogen hair loss. Delayed telogen is common in animals with synchronous hair cycles and hair loss [11]. Authorities showed an increased rate of hair shedding between July and October, they named as actinic effluvium and suggested that it was brought on by sunlight, showing TE in the autumn season [36]. They proposed that ultraviolet light leads to variations in the cellular parts and harm to the follicular cuticle and cortex.

CLINICAL PRESENTATION OF TELOGEN EFFLUVIUM

TE is often characterized by excessive thinning and/or progressive shedding of the scalp hair. The individuals almost develop spaces among the hair and reach the level where they cannot cover their scalp. Furthermore, women sometimes complain about major changes in the proportions of their ponytails. Typically, the diffuse hair thinning will become obvious to the patient if about 50% of the scalp hair is lost. Estimated hair loss in TE range from 150 to more than 400 [38].

The exact incidence of TE is not well known. However, some studies have mentioned the frequency in children to be about 2.7% [3, 39]. TE has no racial predilection, and it involves both sexes, with a greater prevalence rate in women. Though, females consider hair loss disease more significant than men and are in search of medical advice [4].

Trichodynia refers to pain, discomfort, itching, stinging, local tenderness, or paresthesia of the scalp skin, or hair and is considered a common symptom in patients with TE. Its prevalence is around 20%, and it frequently coexists with depression, anxiety, and obsessive personality disease [40]. Observations propose that peri-pilar inflammatory reactions may

Table 1. The mechanisms of hair shedding and the possible triggers in telogen effluvium (TE).

Mechanisms of TE	Causes	Trigger factors
1. Immediate anagen release	Physiologic trauma [4, 19]	Surgical trauma Hemorrhage High fever
	Chronic systemic illness and endocrine disorders	Hypothyroidism or hyperthyroidism Renal failure or liver failure Systemic amyloidosis Inflammatory bowel disease Lymphoproliferative disorders Autoimmune diseases: dermatomyositis and systemic lupus erythematosus
	Nutritional deficiency	Iron deficiency [21] Acquired zinc deficiency [21] Vitamin D deficiency [19, 20, 25] Riboflavin (Vitamin B2) deficiency Severe protein, fatty acid deficiency Biotin deficiency
	Drugs	Androgens, retinoids, beta-blockers, angiotensin-converting enzyme inhibitors, non-steroidal anti-inflammatory drugs (NSAIDs), anticonvulsants, antidepressants, Lithium and anticoagulants.
	Infection	Typhoid, malaria, Mediterranean spotted fever, HIV, tuberculosis, and syphilis.
	Post-COVID-19 acute TE [27–29]	Coronavirus disease-19
	Emotional stress [4]	Depression, bulimia or anorexia nervosa
	Localized inflammatory problems	Psoriasis [24], Seborrheic dermatitis [24], Post-hair transplantation [22].
2. Delayed anagen release	Telogen gravidarum [11]	Acute postpartum TE affects 20% of pregnant women.
	Withdrawal of contraceptive pill [33]	
	TE of the newborn [34]	The occipital hair loss during the first few months.
3. Immediate telogen release	Early treatment of TE	Topical minoxidil [11]
4. Short anagen syndrome	Idiopathic	Chronic TE [35]
5. Delayed telogen release	Actinic effluvium [36]	Sunlight (presented in autumn season)

be a causative agent of trichodynia [41]. TE is classified according to its duration into acute and chronic TE (Table 2).

Acute TE: The hair shedding is lasting for less than six months. Mostly, it happens two to three months after exposure to the triggering factor. The majority of cases reveal trichodynia but are uncorrelated with quantifiable hair loss activity. Subsequently, about 95% of patients spontaneously resolve and get remission, when the cause is short-lived. In cases with healed effluvium there is a presence of shorter, regrowing frontal hair [11, 42].

Chronic TE: is a self-limiting condition characterized by hair loss that lasts for more than six months. It is usually a fluctuating, intermittent course, with irregular periods of spontaneous remissions and exacerbation. The examination reveals normal hair thickness with a shortened anagen phase [4]. Sometimes they show severe frontal hair thinning and temporal recession, which may fluctuate in severity [12]. Patients are particularly fearful of total baldness and are anxious about the continuing hair loss. Repeated reassurance is essential, as chronic TE disorder does not cause complete baldness and represents hair shedding rather than actual complete hair loss. However, it is mandatory to get diagnosed after excluding other causes like abnormal thyroid function, a low protein diet, or iron deficiency. Classically, for unknown reasons, this disease occurs almost exclusively in women, and should not be confused with female pattern androgenetic alopecia (Table 3) [43].

INVESTIGATIONS AND ANALYTICAL CONSIDERATIONS

Recently, the frequency of TE has increased in contrast to other kinds of hair loss [27]. During the COVID-19 pandemic, approximately 25% of individuals experienced TE within 2-12 weeks [44]. Measuring the natural everyday hair loss is

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Table 2. The difference between acute and chronic telogen effluvium (TE).

Variable	Acute TE	Chronic TE
Duration of hair shedding	Less than six months	Six months or more
Outlook	Acute and short-lived	Spontaneous remissions and relapses
Sex	Women more than men	Almost women
Age	Younge age group	Between 30–60 years old
The triggers	Acute and short-lived	Constant, or not reversed
The trichodynia	The majority of cases	Less common
The underlying mechanism	Immediate anagen release. Delayed anagen release. Immediate telogen release.	Delayed telogen release. Shortened anagen phase
Healed telogen effluvium	Shorter, re-growing hairs	Normal thickness of re-growing hair
The site	Frontal hair	Frontal and bitemporal areas
Treatment	Eliminating the underlying causative factors	Difficult and challenging

Table 3. The differences between the telogen effluvium and female patterned hair loss.

Disease	History	Total appearance	Hair loss	Pull test	Trichoscopy/3 zones
Female patterned hair shedding	-Adolescence or older	-Follicular thinning ± bald patches	-Minimal, but Progressive	-Often negative	-Vertex sparseness of hair.
	-Onset: slow	-Spreading: Broader midline, part of the crown.		-Sometime it is positive: frontal (telogen hair)	-Villus hairs in affected areas
	-Possible family history	-Mild bitemporal recession			-Terminal: Vellus hair ratio is $< 4:1$
Telogen effluvium	-Almost adult	-Follicular thinning No bald patches	-Prominent, and Fluctuating ± trichodynia	-Positive: telogen hair	-Equally sparseness of hair.
	-Onset: sudden	-Spreading is generalized			-Upright growing hairs in recovering Disease
	-May be associated with iron and vitamin deficiency, thyroid imbalance, general anesthesia, postpartum, and drugs	-Bitemporal often severe recession			-Terminal: Vellus hair ratio is normal, $> 7:1$

hard. Typically, TE commonly affects ladies, and they complain of hair shedding. Analyses the follicular cycle phase of scalp hair, and calculation of the anagen/telogen ratio can prove the diagnosis of TE, as well as add evidence of the response to treatment. Additionally, the following techniques and laboratory tests may aid in the diagnosis:

- General laboratory work-up: Includes a full blood count, serum levels of iron, ferritin, vitamin D3, zinc, thyroid function tests, a syphilis serology study, an antinuclear antibody titer, and additional tests based on the clinical indication.
- Hair pulls test: Avoid shampooing for 24 hours. Around 40 hairs firmly grasp between the thumb and fingers with a slow comfortable pull (Figure 2A). It performs in 4 different regions of the scalp. A count of more than 4 to 6 club hairs is abnormal [38, 45].
- The clip test (Phototrichogram and TrichoScan): About 25–30 hairs are cut just above the scalp $2cm^2$ surface area. Thereafter, a photo of the same part is taken on different days and assesses the anagen- telogen hair ratio. As well as measure hair growth, hair density, and rate of shedding. The TrichoScan is a noninvasive, and fully computerized phototrichogram [46].
- Trichoscopy (Dermatoscopy): This can be achieved either with or without an interface solution such as alcohol or water. It shows empty follicles and many short, regrowing normal-thickness hair as a result of acute TE (Figure 2B). Chronic TE reveals less than 20% variability in hair diameter, unlike androgenic patterned hair loss, where there are numerous villus follicles [47, 48]. In addition, the trichoscopy needs examination of multiple parts of the scalp, like the frontal, occipital, and parietal areas. Noticing an equal sparseness of hair in all 3 zones gives a hint of chronic TE.
- Modified wash test plus counting hair loss: This is a noninvasive procedure that can be done in the clinic. The patient requested not to shampoo for 5 days; thereafter, they were asked to bathe their hair in a sink enclosed by gauze, gather the hair, and put it in a packet. Afterwards, we calculate the number of lost hairs and the percentage

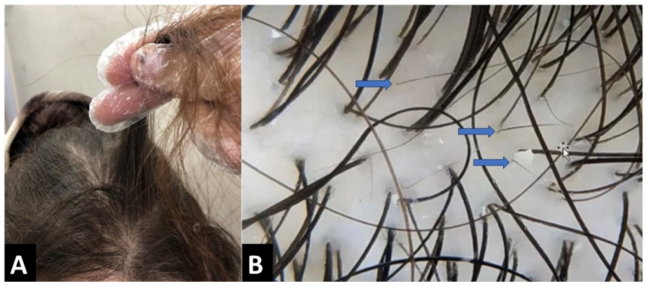

Figure 2. A 31-year-old woman complains of acute telogen effluvium (during recovery). Showing the hair pull test, where around 40-50 hairs firmly grasp between thumb and fingers (A). A Trichoscopy of her scalp displays upright, growing hairs (blue arrows) (B).

of vellus hairs. The possible diagnosis will be TE when the results are exceed 100 shed hairs and less than 10% vellus [49].

- Hair fall count 60-second: It has 2 types:
  1) Home-Based Hair Count: At home, before washing and shampooing the hair, the patient was asked to comb the scalp hair from the back to the front for 60 seconds, put the fallen hair or on the comb onto a white pad, then countdown. The patient repeated the procedure once daily for three days. Finally, calculate the average hair fall count over 3 days. A woman with TE showed that the mean telogen hair loss was 18.18 ± 8.56.
  2) Clinic-Based Modified Count: The patients were evaluated in the clinic. The advice was to refrain from using shampoo. The hair was combed for 15 seconds in each of the four separated regions using the same comb. Meanwhile, counting the hair that had fallen onto the comb or on a pillow, the mean telogen hair loss was 15.81 ± 7.16 [50].
- Trichogram/ hair pluck test/ hair root analysis: A rarely done, semi-invasive test. After 5 days of shampooing, using a clamp or needle holder will pull off 50–100 hairs Thereafter, the determination of hair bulbs. In the case of TE, greater than 25% of telogen hair is revealed to be in the telogen phase [51].
- Histopathology: Sometimes the biopsy is suggested only in patients with long-term, over 6 months of telogen loss. A deep biopsy of 4 mm was taken from the scalp vertex. Acute TE results in a normal anagen/telogen ratio, while chronic TE shows more telogen hair with an anagen to telogen ratio of 8:1, in comparison with 14:1 on a normal looking scalp [52].

MANAGEMENT

Particularly for women, hair shedding has a chief cosmetic concern and hurts self-esteem [53]. Resolving the recognized underlying triggering factor of the self-limited acute TE as soon as possible is crucial for achieving remission [54]. The determinate deficiencies must be corrected, in addition to ensuring a nutritious diet and keeping well-adjusted vitamins, and body weight [55]. Acute TE with an identified triggering factor becomes self-limited and mandates reassurance. The longer period of telogen hair shedding, as well as the more difficult search for causes, may need recurrent evaluation. Generally, all patients are advised to gentle handling of the scalp hair, especially in shampooing, combing, and brushing. On the other hand, there are different modes of treatment of chronic telogen effluvium, including (Table 4):

Minoxidil: It has been advised orally as once daily in chronic TE and appears to reduce hair loss, but it is neither an efficient anagen inducer nor a catagen inhibitor [36, 56]. However, topical minoxidil can be advised in chronic TE. It is believed to increase nourishing hair follicles by causing local vasodilation, but sometimes it results in the exacerbation of disease [57].

Corticosteroids: Topical corticosteroids have been reported in the treatment of TE, especially in decreasing trichodynia, and are considered a sign of effective therapy. Its long period of usage is best avoided [41]. Systemic corticosteroids can be prescribed, particularly in chronic TE, as a manifestation of underlying autoimmune systemic disorders like systemic lupus erythematosus. Actually, the patient must be informed to continue for at least three months [58].

CNPDA: Davis et al, introduced a combination of caffeine, niacinamide, panthenol, dimethicone, and an acrylate polymer (CNPDA), which is considered a new therapy for hair thinning, by increasing the diameter of individual terminal scalp hair follicles, leading to an approximately 10% increase in their cross-sectional area (2–5 μm increase in diameter) and altering the distinctive mechanical properties of thicker hair fibers. The mechanism of action was observed by cryoscanning electron microscopy, showing the deposition of CNPDA, on and between the cuticle layer of hair to expand the baseline diameter [59]. The preceding study established a reduction in hair shedding, and consequently an increase in apparent hair thickness after the use of CNPDA which acts as a functional antioxidant [60]. However, its effectiveness in the treatment of TE remains to be more established.

Adipocyte-derived Stem cells-conditioned media (ADSC-CM): It was effective as a new therapeutic option for patients who complained of TE. The possible principle suggested for hair follicle improvements is based on a combination of growth factors, cytokines, and further proteins formed by adipose-derived mesenchymal cells that proved to encourage hair follicle cell growth. Between each of the two successive sessions, a one-month interval rests for five sittings. The 3 mL of the ADSC-CM solution was applied with a microneedling device at 1.0-mm depth all over the scalp. It is providing positive effects in the clinical sign of efficacy in TE, as well as encouraging significant diffuse regeneration in all trichoscopy parameters [61].

Platelet-rich Plasma (PRP): Monthly sessions of Platelet-rich Plasma (PRP) proved to be an excellent and promising treatment for chronic TE after 1-4 months. It displayed improvement in the visual analogue scale, pull test of hair, and hair density and thickness by trichoscopic examination. There were no significant differences in the hair improvement parameters between the laboratory low-cost tubes and the expensive PRP kit tubes [62, 63]. PRP leads to a decrease in inflammation and improves angiogenesis through several mechanisms by releasing numerous growth factors, chemokines, and cytokines that might trigger the protein kinase B pathway [64]. Subsequently, this results in the improvement and proliferation of the dermal papilla cells and hair follicles [65] Furthermore, PRP could stop hair follicle apoptosis by reducing levels of the anti-apoptotic protein Bcl-2 [66].

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Table 4. The Modes of treatment of chronic telogen effluvium and their outcome.

Treatment	Outcome
Minoxidil (5% Topical)	Improvement in 55.2% of male cases [67].
Corticosteroids	Decreasing trichodynia [41].
Caffeine, niacinamide, panthenol, dimethicone, and an acrylate polymer	Around 10% thickening effect when applied to thousands of fibers [59].
Adipocyte-derived Stem cells-conditioned media	Satisfying results in 70%–92% [61].
Platelet-rich Plasma	The pulled hair decreased by 47–68% after 1 month and by 76% after 3 months [65].

CONCLUSION

Telogen hair fall could present as an acute or chronic form. It has a very important cosmetic query, especially for women. The diagnosis of TE requires a systematic history, clinical examination, and investigations. post-hair transplantation, vitamin D deficiency, and coronavirus disease-19 are considered updating triggers in TE. No exact therapy is essential for most cases. Virtually all patients with acute TE can be cured by eliminating the identified impacted factors. Otherwise, the management of chronic illness is considered a challenging goal for physicians, apart from a new promising therapy by local injection of platelet-rich plasma, or ADSC-CM. Up until now, limited treatment choices with variable benefits have been offered. Therefore, this mandates the authorities to establish or correct the gap in a standard parameter of treatment and discover potential therapeutic options.

ETHICAL DECLARATIONS

Acknoweldgements

None.

Ethics Approval and Consent to Participate

It is not required for a review article. Informed consent had been obtained from the patient in the Figure 2. The author certifies that the patient gave her consent for the image publication and that no personal data are to be included in the article.

Consent for Publication

Not applicable (no individual personal data included).

Availability of Data and Material

None.

Competing Interests

The author declares that there is no conflict of interest.

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Authors' Contributions

Kubaisi TA was responsible for the literature review and writing the manuscript. The author read and approved the final version of the manuscript.

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