



Telogen Effluvium

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Abstract

Background: Hair loss is a common clinical presentation in any medical clinic. Telogen effluvium is considered among the most prevalent causes of hair loss particularly in female patients. Telogen effluvium may associate with significant psychosocial comorbidities and the medical treatment may be challenging. In this article we will review the recent literatures about epidemiology, etiopathogenesis, clinical presentation and management of telogen effluvium.

Method: An electronic literature search was performed using the PubMed and Google Scholar to identify relevant articles published between 1993 and 2017. Search keywords included "telogen effluvium" and "hair loss". We included studies published in English. Editorials, brief notes, conference proceedings, and letters to editors were excluded.

Introduction

Scalp hair plays a pivotal psychosocial role in recognition and social interactions [1]. Hair characteristics, including length and style, are an integral feature of an individual's identity [1]. Therefore, hair loss, or alopecia, can result in significant psychosocial problems in both sexes [1]. Hair loss is very common, with more than one-third of women experiencing clinically important hair loss during their lifetime [2]. Hair loss is classified into scarring and non-scarring forms, which include telogen effluvium (TE). The aim of this article is to review the current literature on TE, which is a common cause of hair loss.

Hair Biology

The normal hair cycle is composed of three characteristic phases: anagen, catagen, and

telogen. The anagen phase represents the period of hair growth, where follicular stem cells in the bulge of the outer root sheath start regeneration[3]. In normal circumstances, the hair fiber growth rate is 10 mm/month [2]. The average normal scalp has 100,000 hairs, with approximately 86% being in anagen, 1% in catagen, and 13% in telogen phases [4]. Normally, scalp hair follicles remain in the anagen phase for 2 to 6 years [5]. Hair follicle regression occurs in the catagen phase, which persists for around 3 weeks. In the telogen phase, hair follicles are quiescent. The telogen phase usually lasts around 3 months. Changes to this normal hair cycle precipitate hair disorders [5].

Hair Follicles in Telogen Phase

After the catagen phase, hair enters the telogen phase, with hair follicles in the telogen

phase located in the upper part of the dermal layer. They appear as small fingers of resting epithelial cells above a cluster of papilla fibroblasts [6]. The transition from the telogen to anagen phase takes place when basal stem cells reside in the bulge of the outer root sheath of the hair follicle in the telogen phase are stimulated to regenerate a new hair fiber [3]. The duration of the telogen phase is associated with hair disorders, with longer durations linked to a higher risk of clubbed hairs shedding, leading to alopecia [5]. When hair follicles fail to start a new anagen phase, old shed fibers are not replaced. The aforementioned is found in cases of both androgenetic alopecia and TE and is the most common mechanism underlying alopecia affecting the scalp [5]. As hair in the telogen phase is located in the upper part of the dermis, it can be pulled out easily. A healthy scalp sheds about 100 telogen-phase hairs daily [2].

Definition

TE was first described by Kligman in 1961 as nonscarring, diffuse, hair loss from the scalp that occurs around 3 months after a triggering event and is usually self-limiting, lasting for about 6 months [7]. This hair disorder results from an acute transition of large numbers of scalp hairs in the anagen phase to hairs in the telogen phase. As a result, the normal ratio of hairs in the anagen to telogen phase is altered [2].

Epidemiology

The prevalence of TE is largely unknown [4]. TE can affect either sex and start at any age, but it affects women more often than men because of hormonal changes [4]. In addition, women usually seek medical advice more often than men for hair-related problems [4]. Chronic TE has been reported mainly in women [8]. Fatani et al. reported an incidence of TE of 1.74% among females in Saudi Arabia [9]. Nnoruka et al. reported an incidence of 9.7% among children in South-East Nigeria [10].

Etiopathogenesis

In a healthy scalp, the ratio of hair follicles in the anagen to telogen phase is 90:10. In someone with temporary hair loss (TE), this ratio shifts to 70% in the anagen phase and 30% in the telogen phase, with daily shedding of up to 300 hairs [2]. In cases of TE, there is

no scar formation at the level of hair follicles [7]. Regarding the mechanism of hair shedding, Headington proposed five separate functional types of TE: [1] immediate anagen release, [2] delayed anagen release, [3] short anagen phase, [4] immediate telogen release, and [5] delayed telogen release [11]. Three of these types are related to events taking place in the anagen phase, and the other two types are related to the telogen phase.

Headington's Classification of Functional types of TE

Immediate Anagen Release

In this common form of TE, hair shedding occurs rapidly, usually over a period of 3 to 5 weeks. After being stimulated by a potential trigger, hair follicles in the anagen phase shift prematurely into the telogen phase. According to Headington, the premature shift from the anagen phase to the telogen phase is probably induced by exposure to drugs or physiological stress, such as a high fever [11].

Delayed Anagen Release

In this form of TE, some follicles remain in the anagen phase for a longer than normal duration. When these follicles are released from the anagen phase, increased shedding of scalp hair occurs [11]. Most cases of post-partum hair loss are due to delayed anagen release [11]. In these cases, withdrawal of circulating placental estrogen prolongs the anagen phase during pregnancy, leading all hair in the anagen phase to enter the catagen phase at the same time [7].

Short Anagen Phase

Idiopathic shortening of the anagen phase, known as short anagen syndrome, may lead to resistant TE, without hair shaft abnormalities [11]. It occurs in the presence of various disorders, such as hereditary hypotrichosis and ectodermal dysplasia, as well as an isolated disorder in otherwise healthy children [7].

Immediate Telogen Release

This form of TE is characterized by shortening of the telogen phase and early commencement of the anagen phase in response to extrinsic signals. Drugs, such as topical minoxidil, can induce immediate induction of the telogen phase. This paradoxical pheno-

menon occurs because stimulation of the anagen phase results in shedding of resting hair [7,11].

Delayed Telogen Release

Delayed telogen release underlies mottling in mammals and probably also seasonal shedding of hair in humans or cases of mild TE that occur following travel from a low-daylight to high-daylight environment hair [7,11].

Rebora's Classification of TE

Rebora proposed a novel classification of TE to simplify Headington's classification system [12]. Rebora identified three pathological types of TE: [1] premature teloptosis, [2] collective teloptosis, and [3] premature entry into the telogen phase [12].

Role of Iron Deficiency and Thyroid Hormone in TE

Iron is a significant cofactor in cells' DNA synthesis, and iron deficiency decreases the capacity of cell proliferation in the hair matrix, predisposing individuals to TE [7]. Hypothyroidism can inhibit cell proliferation, induce the catagen phase, and delay the start of a new anagen phase. The pathogenesis of hair loss in hyperthyroidism is unknown [7].

The Role of Vitamin D Levels in TE

There is no solid evidence about the role of vitamin D in TE. Karadağ et al. reported that patients with TE had higher serum levels of 25-hydroxyvitamin D compared with those of control subjects, whereas serum levels of 1,25-dihydroxyvitamin D were comparable with those of the control group[13]. In contrast, Rasheed et al. compared serum 25-hydroxyvitamin D levels in female patients with chronic TE and healthy controls and found that the patients had significantly lower serum 25-hydroxyvitamin D levels in comparison with those of the control group [14]. Nayak et al. presented similar results [14]. In the presence of these contradictory results, further studies are needed to examine the role of vitamin D levels in both acute and chronic TE.

Clinical Presentations of TE

Acute TE

Acute TE refers to abrupt onset of scalp hair shedding, which takes place 2–3 months after exposure to a potential trigger [15]. In about

33% of acute TE cases, there is no identifiable trigger [15]. Immediate anagen release is the most common underlying mechanism of hair loss[4]. Affected patients commonly complain of increased hair loss during hair washing or combing. Patients with acute TE may show different degrees of anxiety and disquiet at the prospect of total baldness [4]. On clinical examination of the scalp, there is a diffuse scalp hair loss; few patients may show bitemporal hair recession [4]. Hair loss usually does not exceed more than 50% of the scalp hair [7]. The scalp skin appears normal, with no signs of inflammation or evidence of miniaturization [4]. A trichogram may show an increased percentage of hairs in the telogen phase(more than25%) [15]. A hair pull test in acute TE is usually positive for hairs in the telogen phase at sites of the vertex and hair margins. However, a negative hair pull test does not exclude this disorder [15]. In 95% of cases of acute TE, the patient shows some improvement within a few months, but persistent and episodic hair shedding may continue in some patients [4]. It important to consider that cases of prolonged acute TE may signify early androgenetic alopecia or diffuse alopecia areata [4]. Potential underlying triggers that may predispose patients to acute TE include: high fever, surgery, hospitalization, hemorrhage, emotional stress changes in medication, crash-diets, postpartum (telogen gravidarum), contact allergic dermatitis, idiopathic (up to 33% cases) [4].

Chronic TE

Chronic TE is an idiopathic, self-limiting condition, which is characterized by increased shedding of hairs in the telogen phase for at least 6 months but with no widening of the central part line of scalp hair and no miniaturization of hair follicles on a scalp biopsy [15].Patients usually present with excessive and diffuse shedding of scalp hair in a fluctuating course over several years [15].Chronic TE may be a consequence of any of the aforementioned functional types of TE, with shortening of the anagen phase the most commonly involved mechanism [4]. Although there is no identifiable triggering agent in the majority of cases, chronic TE may be induced by acute TE [15]. Chronic TE affects middle-aged women, who usually present with a diffuse decrease of scalp hair length and volume [4]. In the clinical examination, the hair ap-

pears normal in thickness, with short hairs in the frontal and bitemporal areas [4]. Some patients may show marked bitemporal recession, and a hair pull test is usually positive [4]. A negative hair pull test does not exclude a diagnosis of chronic TE [15]. Before reaching a diagnosis of chronic TE, a thorough history taking and clinical examination should be undertaken to rule out other causes of hair loss, such as androgenetic alopecia [15]. Routine work-up should include a complete blood count, serum ferritin and thyroid function tests [4]. Syphilis serology, antinuclear antibody titer, serum zinc levels, and other investigations may be performed, if indicated by findings of the medical history taking and clinical examination [4].

Chronic Diffuse Telogen Hair Loss

Chronic diffuse telogen hair loss refers to telogen hair shedding of longer than 6 months in response to organic causes [4]. To be a true cause of chronic diffuse telogen hair loss, the relationship between the trigger and TE should be reversible and reproducible [4,15]. The diagnosis of chronic diffuse telogen hair loss requires exclusion of other possible diagnoses, such as androgenetic alopecia, reversal of TE following correction of causative factor, and relapse on rechallenge [4]. Causes of chronic diffuse telogen hair loss include thyroid disorders, iron deficiency anemia, acrodermatitis enteropathica, malnutrition, advanced malignancy, hodgkin's disease, senility, systemic lupus erythematosus, dermatomyositis, secondary syphilis and medications including cytotoxic drug, antithyroid agents, anticonvulsants, anticoagulants, antihypertensives [4,15].

Psychosocial Impact

Hadshiew et al. suggested that there was a mutual connection between psycho-emotional stress and hair loss [16]. They proposed that acute or chronic stress may be the primary inducer of TE, whereas stress may be a secondary problem in response to existing hair loss. They suggested that such stress could aggravate the problem of hair loss and induce a self-perpetuating vicious circle.

Diagnostic Approach

Although patients with TE may have symptoms that point to underlying diseases, they are usually asymptomatic. Patients should

be asked to recall any potential trigger that occurred 2–3months before the onset of hair loss. To identify potential triggers, it is crucial to question patients about any medical history of systemic diseases and drug use. A clinical examination of the scalp typically shows uniform hair thinning on normal skin. The presence of scaling, signs of inflammation, altered or uneven hair distribution, or changes in hair shaft's characters may suggest other diagnoses [4]. Laboratory investigations are indicated if the history and clinical examination are suggestive of underlying disorders, such as iron deficiency anemia, zinc deficiency, renal disease, liver disease, or thyroid disease [15]. Trichodynbia, which refers to pain, discomfort, and/or paresthesia in the skin of the scalp or the hair, has been reported to be associated exclusively with the presence of active TE [4].

Treatment

TE is usually self-limited, and treatment includes removing the underlying trigger and reassuring the patient that the condition will usually resolve within 2–6 months [17]. Cosmetically significant hair regrowth can take 12–18 months from the time of the removal of the trigger[7]. In patients with TE, psychological counseling is important to address underlying anxiety or depression [15]. Presently, there is no Food and Drug Administration (FDA) approved treatment for TE. There is no consensus on the level of serum ferritin that can be considered a nutritional deficiency and hair loss trigger [7]. Some experts suggest that the serum ferritin level should be maintained above 40ng/dl, whereas others propose a level of 70ng/dl [4,7]. Hair loss secondary to detectable deficiencies, such as a low zinc level, may be corrected by replacement therapy [7]. Supplements in the form of multivitamins are not approved for the treatment of TE. Topical minoxidil may be helpful because of its effect on prolonging the anagen phase [7]. There is no credible evidence to support a role of antioxidants or other supplements, such as biotin, in the treatment of TE [4]. Although research suggested that green tea containing polyphenolic compounds improved patchy hair loss in mice, no controlled studies of its effects are available in humans [4]. A retrospective study investigated the treatment of chronic telogen effluvium with oral minoxidil sugges-

ted that once daily oral minoxidil therapy appears to reduce hair shedding in CTE [18]. well controlled studies on oral minoxidil are lacking.

Conclusion

TE is a common cause of diffuse hair loss. A diagnosis of TE can be made based on the patient's medical history and a physical examination. As TE is usually a self-limiting event, the approach is observational until spontaneous resolution. When the problem of hair shedding becomes prolonged, further investigations may be required. In such cases, differential diagnoses, such as androgenetic alopecia, should be kept in mind.

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