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Review Article

Dermatology Section

Telogen Effluvium: A Review

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ABSTRACT

Telogen effluvium was first described by Kligman in 1961. It is a most common cause of diffuse hair loss. Women with telogen effluvium more frequently present to dermatologist. A wide variety of potential triggers have been implicated in the pathogenesis of telogen effluvium. Diffuse shedding of telogen hair are seen after 3-4 months of triggering event. The observation of increased telogen hair shedding does not infer a cause. Establishing aetiology of telogen effluvium requires elicitation of relevant history and appropriate laboratory investigations to exclude endocrine, nutritional and autoimmune disorders.

Keywords: Diffuse hair loss, Iron deficiency, Telogen hair

INTRODUCTION

Hair is an ectodermal structure with great cosmetic importance. It helps an individual to maintain self-image and carry on healthy and fruitful social interactions [1]. Hair is essential in identity of many women. Femininity, sexuality, attractiveness and personality are symbolically linked to woman's hair rather than in men. Women are more likely to have lowered quality of life and restricted social contacts as compared to men as a result of hair loss [2]. Loss of hair becomes a matter of concern in all individuals irrespective of age and sex [2].

Normal hair cycle results in replacement of every hair on the scalp by 3-5 years [3]. Telogen effluvium (TE) is the most common cause of diffuse hair loss. There are other causes of diffuse hair loss, which include, female pattern hair loss, chronic TE, anagen effluvium, loose anagen hair syndrome, diffuse type of alopecia areata, congenital atrichia, congenital hypotrichosis and hair shaft abnormalities (hair breakage, unruly hair) [4].

AETIOLOGY AND PATHOGENESIS

By definition TE is a 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 month. In TE hair loss is usually less than 50% of the scalp hair [5]. This condition was first described by Kligman in 1961, as a disease state of hair follicle, where diffuse shedding of telogen hair are seen [4]. Kligman hypothesized that whatever is the cause of hair loss, the follicle tends to be in the form of premature termination of anagen. Later the follicle precipitates into catagen and transforms into resting stage mimicking telogen [6]. The observation of increased telogen hair shedding does not infer a cause. Establishing aetiology of telogen effluvium requires elicitation of relevant history and appropriate laboratory investigations to exclude endocrine, nutritional and autoimmune disorders [6]. A wide variety of potential triggers have been implicated in the pathogenesis of TE [7]. True incidence of TE is not well determined due to lack of data, especially of subclinical cases [8].

Hair cycle implies sequential phases of growth and rest that each follicle goes through which includes anagen (active hair growth), catagen (involution) and telogen phase (resting). The anagen phase may last for about 2 to 8 years, the catagen phase lasts for 4 to 6 weeks and the telogen phase lasts for 2 to 3 months. The exogen phase of hair follicle (the release of telogen hair) coincides with the end of telogen phase [3,5,6]. In the normal scalp, 90–95% of the hair follicles are in the anagen phase and the remainder (5–10%) in the telogen phase with about 100-150 hair being shed daily. Only a few follicles will be in the transitional or catagen phase. The biological clock that determines the end of the anagen phase and the beginning of the catagen/telogen phase is a complex phenomenon whose molecular basis is being unveiled. Various metabolic alterations such as pregnancy, malnutrition and other stressful conditions are capable of influencing the biological clock within hair follicles and it is possible for abnormally large number of hair follicles to enter the telogen phase simultaneously.

TE occurs if a significant number of anagen hair are triggered to stop growing prematurely by any stimulus and subsequently enter catagen phase, followed by telogen phase. After about 2-3 months of initial insult there is excessive hair shedding. The causes of TE have been presented in [Table/Fig-1].

The physiological daily shedding of 100-150 telogen club hair from the scalp is a natural consequence of the hair cycle. Follicles normally retain telogen hair until they have re-entered anagen phase. Eventually the old telogen hair is pushed out by new anagen hair. This shedding does not produce visible alopecia and does not alter the trichogram [7]. A temporary alopecia develops as the long telogen hair are replaced by the shorter new anagen hair, provided the insult is not repetitive. Alopecia resolves as the new anagen hair grow in 3-6 months [7]. There is no genetic cause for telogen effluvium [9].

In post-partum TE, follicles remain in prolonged anagen phase rather than cycling into telogen phase. When finally released from anagen, the clinical sign of increased shedding of telogen hair will be found [9]. There are five functional types of telogen effluvium which are as follows [9]:

1. Immediate anagen release: It is a common form of TE which follows physiological stress including high episodes of fever. During fever, the cytokines initiate apoptosis of hair follicle keratinocytes starting with catagen then followed by telogen [5].

2. Delayed anagen release: This type of TE typically occurs in post-partum hair loss. It is also termed as telogen gravidarum. It occurs due to the high level of circulating placental estrogen which prolongs anagen phase and leads to a full head of hair during pregnancy. The withdrawal of these trophic hormones at delivery causes all the overdue anagen hair to enter into catagen phase simultaneously. This leads to increased shedding of telogen hair, seen after few months of delivery [7].

3. Immediate telogen release: Hair follicles are normally programmed for release of the club hair after an usual interval of 100 days. This results from a shortening of the normal telogen cycle.

This type of hair shedding usually occurs 2-8 weeks after initiation of therapy with topical minoxidil [5,10]. This paradoxical phenomenon occurs because, with the anagen phase being stimulated, there is release of the exogen hair which were resting [10].

4. Delayed telogen release: In this type, hair follicles remain in prolonged telogen rather than being shed and recycling into anagen. Clinical sign of increased shedding of club hair is observed when finally teloptosis (termination of telogen phase with hair shedding) sets in. This process underlies mottling in mammals and probably also seasonal shedding of hair in human or mild telogen effluvia which occurs following travel from low-daylight to high-daylight environment [5].

5. Short anagen phase: It is characterized by the inability to grow long hair because of an idiopathic short anagen phase. The condition is not associated with hair shaft fragility or hair unruliness. This results in resistant TE. It occurs in hereditary hypotrichosis, ectodermal dysplasia and as an isolated disorder in otherwise healthy children [5].

Ribonuclease reductase requires iron as an essential cofactor, which is involved in DNA synthesis. It has been proposed that iron deficiency reduces the proliferation of matrix cells. The arrest of matrix proliferation results in TE. Iron deficiency without anaemia is seen in 20% of cases and manifests solely with a serum ferritin below 20 mg/l [7].

Decreased thyroid hormone level in blood inhibits cell division both in the epidermis and in the cutaneous appendages. In some patients this inhibition of mitosis induces catagen and delays re-entry of telogen hair into anagen. The pathogenesis of hair loss in hyperthyroidism is unknown [7].

CLINICAL FEATURES

The period of dramatic hair loss occurs diffusely from the scalp approximately 2-3 months after the triggering event [7]. The diffuse loss may produce thinning of hair all over the scalp, but frequently manifest with bitemporal recession. Loss is normally not more than 50% of the scalp hair [5]. Usually patients do not relate these events to their recent illness and are anxious that they may go bald [7]. Scarring and inflammation are absent [3]. Clumps of telogen hair can be extracted with ease from both the vertex and the margins of the scalp [7]. Chronic starvation, in particular marasmus may result in dry, lusterless, fine, straight hair that are sparse and easily pluckable. Kwashiorkor results in periods of interrupted hair growth that either sends the hair into telogen phase, or, if less severe, it affects the hair caliber more than its linear growth, hence producing multiple Pohl Pinkus lines. Hair colour change is a prominent feature in this situation. Dark hair becomes brown or red, while brown hair changes to blond. This colour change along with periodic constrictions produces the so called “flag sign” of Kwashiorkor. Essential fatty acid deficiency produces lightening of hair colour and also marked telogen hair loss [7].

Chronic telogen effluvium (CTE) is a diffuse hair loss of scalp that persist for more than six months [10]. It is characterized by abrupt, excessive, alarming, diffuse shedding of hair that runs a fluctuating course over several years [5,6]. This condition predominantly affects healthy women in the fourth to fifth decade of life [10]. It may be primary or secondary to various causes [7]. In primary CTE no specific triggering agent is evident. CTE may be triggered by an acute TE [5]. Examination shows normal or high hair density, although there may be some rarefaction with short hair along the frontotemporal region [11].

The hair pull test is strongly positive in TE. It is done by grasping 40-60 closely grouped scalp hair with thumb and index finger and gentle traction is applied as the hair are pulled firmly and slowly from the scalp. Normally only 2-3 hair are pulled out by this method. In excessive shedding, more than 10% hair are easily pulled out from any part of the scalp provided that the patient has not shampooed for more than 24 hours [4]. The trichogram (hair pluck test) from a hair pluck sample is abnormal and shows greater than 25% telogen hair [7]. The hair collected during pull test and pluck test on light microscopic examination shows club hair [12]. Hair pull test has been found to be a poorly sensitive method as telogen percentage in trichogram does not correlate with severity of hair loss. While daily hair count is a cumbersome method, it has been proposed that the wash test is probably the best method to adopt. In wash test, the patient is instructed to wash hair after 5 days of last shampoo, in a sink with its drain covered by gauze. The hair entrapped in the gauze is then counted [5].

Physiological causes	Postpartum effluvium (telogen gravidarum)
	Physiological effluvium of newborn
Febrile states	Typhoid
	Malaria
	Tuberculosis
	HIV infection
Stress	Severe febrile illness
	Emotional stress
	Serious injuries
	Major surgery
	Difficult labor
	Haemorrhage
	Starvation
	Crash diet
Drugs	Oral retinoids (etretinate and acitretin)
	Oral contraceptives
	Antithyroid drugs
	Anticonvulsants
	Hypolipidemic drugs
	Heavy metals
	Beta blockers [7]
	Captopril [7]
	Amphetamines [7]
Endocrine	Hyperthyroidism
	Hypothyroidism
Organ dysfunction	Renal failure
	Hepatic failure
Disorder of hair cycle	Short anagen syndrome
Nutritional	Iron deficiency anemia
	Acrodermatitis enteropathica
	Acquired zinc deficiency
	Malnutrition
Local cause	Hair dye application
Others	Syphilis
	Systemic lupus erythematosus

[Table/Fig-1]: Causes of telogen effluvium

INVESTIGATIONS

Detailed history and clinical examination helps to detect the cause of TE. If not, a minimum battery of laboratory tests should be performed, which includes complete blood count, urine analysis, serum ferritin and T3, T4, thyroid stimulating hormone (TSH) [4]. Antinuclear antibody titre and serum zinc level should be done if there are other features on history or on examination to suggest these conditions [7]. Two common conditions associated with TE are iron deficiency anaemia and thyroid disorders and many a times there are no apparent clinical features to suggest these conditions.

Hence, these are included in the minimum battery of tests in cases with TE/diffuse alopecia with no apparent cause [4].

Jain et al., recorded the following probable aetiological causes of diffuse hair loss in their study (n=100); fever (33%), psychological stress (30%) and systemic illness (23%) [8]. Scalp biopsy is usually not needed but is the most definitive way to diagnose TE. Usually biopsy of TE is normal except for an increase in telogen follicles (normal telogen counts 6-13%). Proportion of telogen follicles more than 15% suggests TE but more than 25% is definitive feature. Scalp biopsy may not be necessary for the diagnosis, but it helps to rule out FPHL and alopecia areata [4].

TE must be differentiated by psychogenic pseudoeffluvium in which the patient seeking advice for hair loss is not necessarily balding. Normal dense scalp hair and absence of any clinically convincing evidence of hair loss is regarded as the feature of imaginary hair loss or psychogenic pseudoeffluvium. In these cases patients might need psychological consultation [5].

TREATMENT

The most important aspect in the management of TE is counseling the patient about the natural history of the condition. Normal hair cycle and relationship between triggers and timing of hair loss should be explained [13]. Attempts should be made at identifying the specific cause and once identified, they have to be corrected. Hair shedding takes 3-6 months to cease, after which regrowth can be noted 3-6 months on removal of trigger, but cosmetically significant regrowth can take 12-18 months [14,15].

Stress is one of the major contributing factors for telogen effluvium. There is no specific therapeutic intervention which could prevent stress induced premature onset of catagen [16]. Psychological counseling being the least invasive and easy to address the psychosocial impact, is considered as the best and safest treatment [14].

The patient needs a brief discussion on the diagnosis and treatment options. Potential therapeutic options include the following depending on the pathogenesis of TE [11].

1. Inhibition of catagen.
2. Induction of anagen in telogen follicles.
3. Inhibition of exogen.

Presently there are no FDA approved highly efficient catagen inhibitors or anagen inducers [11]. Butcatagen-inducing drugs (beta-blockers, retinoids, anticoagulants, or antithyroid drugs) need to be avoided and catagen-inducing endocrine disorders (thyroid dysfunction, hyperandrogenism, or hyperprolactinaemia) should be treated. Substitution therapy for catagen promoting deficiencies (like iron, zinc, estradiol, proteins) can also be initiated [14,17].

The association of low serum ferritin level and hair loss has been debated over the years. There are ongoing controversies for low serum ferritin levels to be designated as nutritional deficiency triggering hair loss [18]. In order to reverse severe hair loss some authors suggest serum ferritin level should be maintained above 40ng/dl [17] or 70ng/dl [19]. Adequate dietary intake and oral administration of ferrous sulfate 300mg three to four times a day is an effective, initial therapy [20]. Iron supplementation is given for 3-6 months till stores are replenished [20,21].

Balanced diet is an utmost important for hair loss. Hair loss due to measurable deficiencies may be supported by replacement therapy but supplements in the form of vitamins are not proved for hair loss [22]. For instance there is no effect of biotin supplement over hair loss [22]. Efficacy of the replacement of iron or thyroxine on the outcome of TE are also lacking, although some benefits have been achieved in few controlled studies [23].

Topical minoxidil has also been tried and it is a reasonable candidate drug which is known to prolong anagen [24].

CONCLUSION

Telogen effluvium is the most common cause of diffuse non-scarring alopecia. It is characterized by an abrupt onset of diffuse hair loss usually seen 2-3 months after a triggering event. It is usually self-limiting lasting for 6 months whereas in chronic telogen effluvium it persists beyond 6 months. No specific treatment exists for telogen effluvium. The most important aspect in the management of telogen effluvium is educating the patient about natural history of the condition.

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