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Diffuse alopecia in females: An update.

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ABSTRACT

Scalp hairs complete the body self-image and patients with diffuse alopecia suffer from overt disfiguration, leading to psycho-social embarrassment and significant lack of self-esteem. Diffuse alopecia is a non-scarring type of alopecia. Telogen effluvium is the most common cause of diffuse alopecia in females. Abrupt, rapid, generalized shedding of club hairs, 2-3 months after a triggering event like parturition, high fever, major surgery etc. indicates telogen effluvium, while gradual diffuse hair loss with thinning or widening of central parting line indicates female pattern hair loss, another common cause of diffuse alopecia. Excessive alarming diffuse hair loss, from a normal looking ahead without an obvious cause, is the hallmark of chronic telogen effluvium, which is a distinct entity different from telogen effluvium and female pattern hair loss. Hence an early diagnosis and an aggressive treatment in the case of active hair loss are crucial in the management of diffuse alopecia.

Keywords: Diffuse alopecia, females, non-scarring, pattern, Telogen Effluvium.

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INTRODUCTION

Diffuse alopecia or hair loss is a very common problem observed in the outpatient department of dermatology. However a rational or comprehensive classification system of various causes is still lacking. This article reviews the various causes of diffuse alopecia in females, because women tend to find, the hair- shedding more troublesome than men do; thus more women seek medical advice for the condition. There are three patterns of diffuse alopecia observed in females [1] (Table-1).

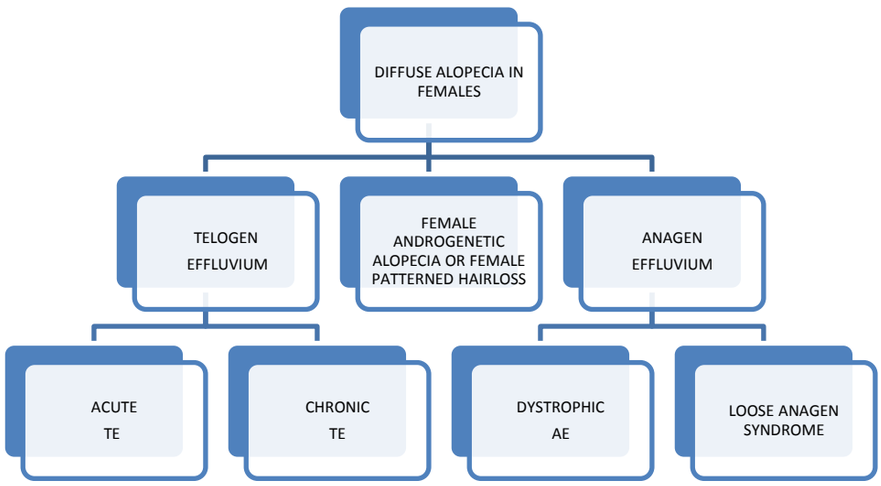

Table-1: - Patterns of diffuse alopecia observed in females.

Throughout life, the hair follicles undergo period of cyclic growth. At any given time, 85-90 % of follicles are in the anagen phase and during this phase; mitotically active matrix cells in the hair bulb differentiate and divide, resulting in hair growth [2]. Approximately 10% of follicles are in telogen (dormant) phase, during which all mitotic activity is arrested and about 1% is in catagen (involution) phase. The final step of hair cycle is exogen, when the hair is released from the follicle [2]. A telogen hair remains in its follicle for upto 4-6 weeks after the onset of anagen. This cycle results in the replacement of every hair on the scalp every 3-5 years, with individual follicles undergoing 10-30 such cycles in a lifetime [3, 4]. The hair cycle is not synchronous throughout the scalp and the length of each phase as well as the length of the entire cycle varies with the site and the age of the patient.

Diffuse alopecia in females shows three patterns: - (A) AE (anagen effluvium), (B) FPHL (female pattern hair loss), and (C) TE (telogen effluvium) [1].

Anagen Effluvium

It is the abrupt loss of hairs that are in their growing phase (anagen) due to an event that impairs the mitotic activity of hair –follicle [5].Anagen effluvium is categorized into two types**:** the common dystrophic anagen effluvium and the loose anagen syndrome [6]. Although anagen effluvium is commonly associated with chemotherapy and radiation to the head and neck[7,8], other causes of anagen effluvium are severe protein energy malnutrition, pemphigus vulgaris[9], Systemic Lupus Erythematosis [7], alopecia areata[10] and exposure to thallium[11], mercury[12] etc. Other medications that can rarely cause anagen effluvium are Bismuth, L-dopa, Colchicine, and Cyclosporine [3, 12].Any insult or event that causes abrupt cessation of mitotic activity leads to weak and breakage of partially keratinized hair shaft within the hair canal and even complete failure of hair formation [5, 6, and 7]. Hair shedding usually begins 1-3 weeks after this incident. Due to its long anagen phase, the scalp is the most common location for hair loss. Normally upto 90% of scalp hairs are in the anagen phase, and hair loss is copious and results in alopecia that is quite obvious.

In anagen effluvium, only the proliferating cells in the bulb are affected, the quiescent stem cells of the bulge are spared, so hair loss is usually completely reversible. The hair follicle resumes normal cycling within a few weeks of cessation of insult and regrowth is apparent within 1-3 months [5, 7, and 13]. The new hair may have different characteristics like graying, curling or straightening effect, which is likely due to different effects of CT(chemotherapy) or RT(radio therapy) on hair follicle melanocytes and inner root sheath epithelium[13,14,15]. Permanent alopecia occurs with more than 30Gy of deep X-ray or 50Gy of soft X-rays [5].

Female Androgenetic Alopecia or Patterned Hair Loss

Alopecia induced by androgens in genetically predisposed individuals is termed as androgeneticalopecia [16]. For women affected with androgenetic the main factors contributing to psychological distress were inability to style their hair, dissatisfaction with their appearance, concern about the continuing hair loss and concern about others noticing their hair loss [17]. In Caucasian female patients [18], the prevalence was 19% and Chinese female [19] patients the prevalence was only 6%.

The incidence of androgenetic in women also tends to increase with age (postmenopausal) with a possible hormonal influence [18, 20]. A better term for female androgenetic would be female pattern hair loss (FPHL). FPHL is gradual onset, slowly progressive, non-scaring alopecia and is characterized by a reduction in hair density over the crown with retention of the frontal hair line. The clear difference in the clinical pattern of male and female pattern hair loss suggest that these are two separate entities. This is also based on studies which have shown no clear relation between excess testosterone levels and females pattern loss [18].

In androgenetic the duration of anagen phase gradually decreases and that of telogen phase increases causing stepwise miniaturization of the hair follicle, leading to vellus transformation of terminal hair follicle giving a bald look [21, 22].

In the **Ludwig's classification**, three patterns representing stages or progression of FPHL are recognized [23].

- **Stage 1** is characterized by a perceptible thinning of hair from the anterior part of the crown with minimal widening of midline part. Biochemical levels are normal [24].
- **Stage 2** is seen with advancing age when the rarefaction on the crown becomes more pronounced, and the number of thinner and shorter hairs increases. An excess of androstenedione, and androstanediol glucuronide may be detectable [24].
- **Stage 3** is usually not encountered before menopause, and the crown may become literally bald and a fringe of hair along the frontal hair line persists. Very high levels of androstenedione, (DHEA-S) Dehydro Epiandroestene Sulfates, sometimes Prolactin are the usual findings at this stage [24].

Olsen emphasized on the frontal accentuation (or the "Christmas tree" pattern) of hair loss in FPHL [25]

.**Table-2: Differences between Female Pattern hair loss and chronic telogen effluvium.**

Particulars	Female Pattern hair loss	Chronic telogen effluvium
Distribution	central portion of scalp and preserved frontal hairline	Generalized
Onset	Gradual	Abrupt with a trigger
Appearance	Hair thinning with wide midline part	Diffuse thinning
Hair shedding	Minimal	Prominent
Hair pull test	usually negative	Positive
Other history	family history positive	History of previous illness
Scalp biopsy	T: V reduced (less than 4:1)	T: V normal (8:1)
Miniaturized hair	Present	Absent
Trichogram	A:T normal	A:T reduced
Dermoscopy	Marked variation in shaft diameter miniaturized follicle (hallmark)	No significant variation, No miniaturization
Course	Gradually progressive	Prolonged and fluctuating course

T: V (Terminal: Vellus) Ratio, A: T (Anagen: telogen) ratio.

Telogen Effluvium

Telogen effluvium is one of the most common causes of diffuse non- scarring hair loss. The term telogen effluvium was first coined by flagman to describe increased shedding of normal club hairs, with the hypothesis that irrespective of the cause, the follicle tends to behave in a similar fashion by undergoing a premature termination of anagen, precipitating telogen [26, 27].

The degree of effluvium depends on the severity and duration of exposure. TE is caused by any disruption of hair cycle resulting in increased synchronized telogen shedding [28]. As most cases are subclinical, its true incidence and prevalence is largely unknown.

Heading ton described five functional types of TE [29].

1) **Immediate anagen release** occurs when follicles are stimulated to leave anagen and enter telogen prematurely, resulting in increased telogen effluvium 2-3 months later. This can be seen with physiological stress, severe illness, or drug induced hair loss. The reversal of stress is associated with resumption of normal cycle.
2) **Delayed anagen release** is due to prolongation of anagen resulting in delayed but synchronous onset of heavy telogen shedding. E.g. is telogen gravidarum and after discontinuation of contraceptive pills.
3) **Immediate telogen release** generally occurs with drug induced shortening of telogen leading to follicles reentering anagen prematurely. Hence, a massive release of club hairs occurs as is seen when starting therapy with minoxidil.
4) **Short anagen syndrome** is characterized by an idiopathic shortening of anagen duration, leading to persistent telogen hair shedding. This mechanism is considered responsible for majority of cases with CTE with mild persistent hair loss and inability to grow the hair long.
5) **Delayed telogen release** is characterized by a prolonged telogen and delayed transition to anagen. It occurs in animals with synchronous hair cycles but may be responsible for seasonal hair loss in humans.

Acute telogen effluvium

ATE was first described as an acute onset scalp hair loss, occurring 2-3 months after a triggering event, which could be unidentifiable in 33% cases [3, 29]. Common causes are high fever, major surgery [26], hospitalization, hemorrhage [30], emotional stress [31], crash diets [32], postpartum [29], Actinic [33, 34] (Sunlight and UV light induced) effluvium, and seasonal variation (July to Oct) [35].

The functional mechanism of shedding in majority of the casesis immediate anagen release [29]. In cases of childbirth, there is acute telogen effluvium after 3-5 months of the event because of the delayed anagen release. The mechanism of hair loss in seasonal and actinic effluvium is not scientifically proved. The authors proposed that it could be possibly ultra violet light induced, manifesting itself in autumn in predisposed individuals[35].The production of porphyrins by propianibactin the pilosebaceous duct leading to oxidative tissue injury and follicular micro inflammation, keratinocyte response to physiochemical stress from ultra violet radiation ,irritant and pollutant induced radical oxygen species and nitric oxide; or release of pro inflammatory cytokines damaging follicular stem cells could be responsible[33]. Acute telogen effluvium usually remits within few months in 95% of cases.

Diagnostic feature of acute telogeneffluvium

- Abrupt onset, rapid diffuse generalized shedding of hairs, usually seen 2-3 months after a triggering event.
- Strongly positive hair pull test. No shampooing for 24 hours, approximately 60 hairs are grasped between the thumb and the index and middle finger and gently pulled. A negative test (less than 6hairs) indicates normal shedding, whereas a positive test (more than 6 hairs) indicates active hair shedding.
- A Trichogram (forcible complete hair pluck of 40-60 hairs) showing significant reduction in anagen: telogen ratio usually more than 25% of the plucked hairs are telogen hairs in acute phase.
- Photo-trichogram- all hairs within 2sq cm area are trimmed 1mm from the skin surface and photographed on day one, three and day seven to assess the rate of hair growth, hair density and rate of shedding. As only anagen hairs elongate, this determines the ratio of anagen: telogen hairs.
- Trichoscan is fully computerized photo-trichogram.
- Photo-trichogram and trichoscan are non-invasive, simple and sensitive technique than classical Trichogram.
- Video-dermoscopy will show large number of short tip pointed regrowing hairs in the absence of hair diameter variability.

Chronic Telogen Effluvium

First described by David A Whiting [36] in 1996, chronic telogen effluvium is a primary idiopathic condition affecting middle aged women which needs to be differentiated from chronic diffuse telogen hair loss secondary to organic causes and androgenetic. It presents as telogen effluvium lasting more than 6 months, without any widening of central part or follicular miniaturization [37]. The disorder appears to be distinct from acute telogen effluvium because of it's prolonged, fluctuating course. The telogen hair shedding seen in chronic telogen effluvium could be result of almost any of the functional types described earlier. However, a shortening of anagen is the most common mechanism involved in chronic telogen effluvium [29].Clinical features include an insidious onset and fluctuating course lasting for several years. It typically affects middle aged women. On examination the hair appears normal in thickness, with short regrowing hairs in the frontal and temporal areas. The condition could spontaneously resolve after a decade or so [38, 39].

Chronic diffuse telogen hair loss (CDTHL) refers to telogen hair shedding longer than 6 months secondary to a variety of organic causes. To be a true cause of CDTHL, the relationship between the trigger and telogen effluvium must be reversible and reproducible, the requirement of proof includes exclusion of other known causes of shedding, and reversal of effluvium following correction of causative factor and relapse on re-challenge [3].

Common causes are thyroid disorders, Iron deficiency anemia, acrodermatitis-enteropathica, and malnutrition [30].

Diet has an important role to play in telogen effluvium [40]. Rigorous dietary restriction or crash dieting (less than 0.8 gm/kg protein and 1200kcal/day)can lead to inadequate supply to the hair matrix and precipitate telogen hair loss[32,41].

Investigation

Lab test

- complete blood count
- Serum ferritin levels
- TIBC (Total Iron Binding Capacity)
- Serum T3 T4 TSH levels
- Serum testosterone or dihydro testosterone levels
- Sex hormone binding globulin
- Serum prolactin
- ANA, DS-DNA, CRP,ESR, VDRL

Methods to evaluate hair loss

Category	Method
• Non – Invasive: -	Questionnaire, daily shed hair count standardized and modified wash test, Global photographs, Dermoscopy, Phototrichogram, Trichoscan, Polarizing, and surface electron Microscopy.
• Semi – Invasive: -	Trichogram and unit area Trichogram
• Invasive: -	Scalp Biopsy

Treatment of Diffuse Alopecia

General measures

- Identification and treatment of underlying cause: high fever, severe infection or any other disease causing telogen hair loss should be identified and treated. Similarly, patients of female pattern hair loss need hormonal assessment and treatment.
- Counseling of the patient: The patient should be explained telogen effluvium represent excessive hair shedding rather than actual hair loss, this does not cause baldness. The hair lost would be replaced by regrowth. In case of acute telogen effluvium the hair shedding stops after 3-6 months of removal of triggering event and may take 3-10 years in case of chronictelogen effluvium.
- Assessment and treatment of iron deficiency and thyroid hormone disorder: It is recommended that serum ferritin level should be maintained between 40-70µg/ml. When no apparent cause is found, screening for T3, T4 and TSH is recommended.
- Anagen effluvium is usually reversible and appropriate hair and scalp care along with temporarily wearing of wig may be the most effective coping strategy required in these patients.

Specific Treatment

- Minoxidil topical solution available in 2%, 5% and 10% strength. It is used in female pattern hair loss.
- Hair prosthesis and hair cosmetics are used for severe FPHL.
- Anti androgens may be beneficial, especially in cases of FPHL with hyper androgenism.
- Finestride is reserved for cases of FPHL with hyper androgenism.

REFERENCES

[1] Jain VK, Kataria U, Dayal S. Study of diffuse alopecia in females. Indian J Dermatol venereol Leprol. 2000 Mar- April 66 (2)

[2] Vogt A, McElvee K, Blume- Peytavi U. Biology of hair follicle. In: Blume- Peytavi U, Tosti A, Whiting D, Trueb R, editors. Textbook on hair – From basic science of clinical application. Berlin Springer Verlag; 2008.p.1-22.

[3] Harrison S, Sinclair R. Telogen effluvium. Clin Exp Dermatol 2002; 27:389-5.

[4] Kligman AM. The human hair cycle. J Invest Dermatol 1959; 33:307- 16.

[5] Trueb RM. Diffuse hair loss. In: Blume – Peytavi U, Tosti A, Whiting DA, Trueb RM, editors. Hair Growth and Disorders. Berlin: Springer; 2008.p.259-72.

[6] Paus R, Oslan EA, Messenger AG. Hair Growth Disorders. In: Wolff K,Goldsmith LA, Katz SI Gilchrest BA, Zller As, Leffell DJ, editiors.Fitzpatrick's Dermatology in General Medicine.7th ed., vol, 2. New York: McGraw – Hill; 2008.p. 753 -77.

[7] Sperling LC. Hair and systemic disease. Dermatol Clin 2001; 19:711-26.

[8] Sinclair R, Grossman KL, Kvedar JC. Anagen hair loss, in Disorders of Hair Growth: Diagnosis and Treatment. In: Olsen EA, editor. McGraw-Hill: New York; 2002.p.275.

[9] Delmonte S, Semino MT, Parodi A, Rebora A.Normal anagen effluvium: A sign of pemphigus vulgaris. Br J Dermatol 2000; 142:1244-5.

[10] Quercetani R, Rebora AE, Fedi MC, Carelli G, Mei S, Chelli A, et al. Patients with profuse hair shedding may reveal anagen hair dystrophy :A diagnostic clue of alopecia areata incognita. J Eur Acad Dermatol Venereol 2011; 25:808-10.

[11] Bank WJ, Pleasure DE, Suzuki K,Nigro M,Katz Thallium poisoning. Arch Neurol 1972; 26:456-64.
[12] Elhassani SB. The many faces of methyl mercury poisoning. J Toxicol Clin Toxicol 1982; 19:875-906.
[13] Vowels M, Chan LL,Giri N, Russell S,Lam-Po-Tang R. Factors affecting hair regrowth after bone marrow transplantation. Bone Marrow Transplant 1993; 12:347-50.
[14] Tosti A,Pazzaglia M.Drug reactions affecting hair: Diagnosis. Dermatol Clin 2007; 25:223-31.
[15] Trueb R. Chemotherapy – induced Hair Loss. Skin Therapy Lett 2010;15:5-7
[16] Sehgal VN, Kak R, Aggarwal A, Srivastava G, Rajput P.Male pattern androgenetic alopecia in an Indian context: A perspective study. J Eur Acad Dermatol Venereol 2007; 21:473-9.
[17] Girman CJ, Hartmaier S, Roberts J, Bergfeld W, Waldstreicher J. Patient –perceived importance of negative effects of androgenetic alopecia in women. J Womens Gend Based Med 1999; 8:1091-5.
[18] Norwood OT. Incidence of female androgenetic alopecia (female pattern alopecia). Dermatol Surg 2001; 27:53-4.
[19] Wang TL Zhou C, Shen YW,Wang XY, Ding XL, Tian S, et al. Prevalence of androgenetic alopecia in China: A community-based study in six cities. Br J Dermatol 2010; 162:843-7.
[20] Gan DC, Sinclair RD. Prevalence of male and female pattern hair loss in Maryborough. J Invest Dermatol Symp Proc 2005; 10:184-9.
[21] Paus R, Cotsarelis G. The biology of hair follicles. N Engl J Med 1999; 341:491-7.
[22] Pierard – Franchimont C, Pierard GE. Teloptosis, a turning point in hair shedding biorhythms. Dermatology 2001; 203:115-7.
[23] Ludwing E. Classification of the types of androgenetic alopecia (common baldness) occurring in the female sex. Br J Dermatol 1977; 97:247-54.
[24] Camacho – Martinez FM. Hair loss in women. Semin Cutan Med Surg 2009; 28:19-32.
[25] Olsen EA. Female pattern hair loss. J Am Acad Dermatol 2001; 45:S70-80.
[26] Kligman AM. Pathologic dynamics of human hair loss. Telogen effluvium. Arch Dermatol 1961; 83:175-98.
[27] Gilmore S, Sinclair R. Chronic telogen effluvium is due to a reduction in the variance of anagen duration. Australas J Dermatol2010; 51:163-7.
[28] De Berker D. Clinical relevance of hair microscopy in alopecia. Clin Exp Dermatol 2002; 27:366-72.
[29] Headington JT Telogen effluvium. New concepts and review. Arch Dermatol 1993; 129:356-63.
[30] Dawber RPR, Simpson NB, Barth JH. Diffuse alopecia: Endocrine, metabolic and chemical influences on the follicular cycle. In: Dawer RP, editor. Diseases of the hair and scalp. Oxford: Blackwell Science; 1997.p.123-50.
[31] Hadshiew IM, Foitzik K, Arck PC, Paus R. Burden of hair loss. Stress and the underestimated psychosocial impact of telogen effluvium and androgeneticalopecia J Invest Dermatol 2004; 123:455-7.
[32] Goette DK, Odom RB. Alopecia in crash dieters. JAMA 1976; 235:2622-3.
[33] Trueb RM. Is androgenetic alopecia a photo aggravated dermatosis. Dermatology 2003; 207:343-8.
[34] Braida D, Dubief C, Lang G. Photo ageing of hair fiber and photo protection. Skin Pharmacol 1994; 7:73-7.
[35] Pierard –Franchimont C, Peerard GE. L'effluvium telogene actinique: Une facette de la chronobiologie humaine. Int J Cosmet Sci 1999; 21:15-21.
[36] Whiting DA, Chronic telogen effluvium: increased scalp hair shedding in middle aged women. J Am Acad Dermatol 1996; 35:899-906.
[37] Messanger AG, de Berker DA, Sinclair RD. Disorders of Hair. In: Burns T, Breathnach S, Cox N, Griffits C, editiors. Rooks textbook of Dermatology. 8th ed. UK: Blackwell Publishing Ltd; 2010.p.66.27-66.
[38] Sinclair R. Diffuse hair loss. Int J Dermatol 1999; 38 (suppl 1):8-18.
[39] Rushton DH, Ramsey ID, James KC, Norris MJ, Gilkes JJ. Biochemical and trichological characterization of diffuse alopecia in women. Br J Dermatol 1990; 123:187-97.
[40] Kaimal S, Thappa DM. Diet in dermatology: Revisited. Indian J Dermatol Venereol Leprol 2010; 76:103-15.
[41] Rushton DH. Nutritional factors and hair loss. Clin Exp Dermatol 2002; 27:396-404.
[42] Dawber RP,Connor BL. Pregnancy, hair loss, and the pill. Br Med J 1971; 4:234.