**CAUSES AND MANAGEMENTS OF HAIR LOSS IN WOMEN**

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**ABSTRACT**

Alopecia or hair loss is a quite disturbing case. It can occur in both women and men. In women, the most familiar reason for hair loss is telogen effluvium (TE) succeed by female pattern hair loss (FPHL) and chronic telogen effluvium (CTE). Telogen effluvium generally occurs at any age, it is caused by prematurely transition of anagen hairs to telogen hairs. It can be induced by several events such as physiologic or emotional stress or certain drugs. Meanwhile, female pattern hair loss is caused by increasing of 5α-reductase enzymes and androgen receptors in effected scalp. Chronic telogen effluvium is the shedding of telogen hairs that occur over 6 month with idiopathic cause. There are also other causes of hair loss in women such as alopecia areata (AA), trichotillomania, and traction alopecia. TE and CTE are treated by removing the triggering factors. Patients need to be informed and educated that it is self limited and usually resolve with time. For FPHL, the treatment includes minoxidil 2% solution, antiandrogen therapies and hair transplantation. Meanwhile, AA is treated by intralesional corticosteroid injection, psoralen and ultraviolet A (PUVA). In the other hand, women with trichotillomania and traction alopecia need counselling and education to change their behaviours.

**Keywords** : Telogen effluvium, Female pattern hair loss, Chronic telogen effluvium, 5α-reductase enzymes, Androgen receptors

**INTRODUCTION**

On average, the scalp contain about 100,000 hairs which grow in cycles. Each hair follicles will undergo about 10 up to 30 cycles in its life span.[1,2] The cycles include three phases which are anagen (active hair growth phase), catagen (involution phase) and telogen (resting phase).[1,2] Disturbances of any phases in this cycles can contribute to the occurring of diffuse hair shedding.[2] Anagen phase may persist for 3 to 7 years, while catagen phase will take place about 4 to 6 weeks until it lastly proceed to telogen phase that lasts about 2 to 3 months. About 10% of scalp hair is in phase of telogen. About 100 up to 150 shedding of telogen hairs per day is acceptable.[1,2]

Hair loss can be categorized into two which are scarring (such as discoid lupus, lichen planopilaris, and folliculitis decalvans) and non scarring. However this report will focus on discussing about non scarring hair loss.[1,5] The most known diffuse hair loss in women is telogen effluvium (TE), then followed by female pattern hair loss (FPHL) which is also named as androgenetic alopecia (AGA), and chronic telogen effluvium (CTE).[4] Meanwhile, other causes of women hair loss that rarely to occur are alopecia areata, anagen effluvium, severe bacterial infections, trichotillomania, abnormal hair-care practices, hypothyroidism, iron-deficiency, tinea capitis, and hyperthyroidism.[1,5]

Nowadays, hair transplantation has been developed to treat the patient with diffuse hair loss instead of only using topical solution. However there are certain criteria that need to be fulfilled and some contraindication that should be looked over before the patients can undergo the hair transplantation. The treatment of diffuse hair loss generally based on the causal.[3,4,5] Therefore, it is important to establish the diagnosis in order to go to the next step in choosing the suitable treatment for hair loss.

**CAUSES OF HAIR LOSS IN WOMEN**

The most familiar causes of hair loss in women is telogen effluvium (TE). Then, followed by female pattern hair loss (FPHL) and chronic telogen effluvium (CTE).[4] Still, there are many other reason of hair loss in women that should not be taken lightly such as alopecia areata, trichotillomania and traction alopecia.[1,5]

**Telogen Effluvium (TE)**

Telogen effluvium is described as an abrupt in onset, and rapid, diffuse, self-limited, extreme shedding of normal hair that commonly appear two to three months following the triggering events. It is results from abrupt transformation of hair from anagen to telogen by the ratio of 90:10 to 70:30. Clinical presentation that may be complained by patients is elevation in number of hair shedding which is usually about 150 to 400 hairs in a day.[1,2,4,5] TE can be causes by physiologic conditions such as after delivery (postpartum), physical or emotional stress and drugs like oral retinoids and hormone replacement therapy (HRT). The frequent triggers are severe febrile illness (such as malaria), telogen gravidarum, emotional distress, chronic systemic illness, accidental trauma, severe haemorrhage and major surgery,.[4]

Telogen effluvium can be diagnosed by several means. First based on it features which is the hair shedding is abrupt in commencement and diffuse rapidly. Normally the triggering events occur 2 or 3 month before the shedding. Secondly, base on hair pull test where a group of 25 to 50 hairs are gently pull from their base to the tips. In shedding conditions, 10 to 15 hairs can be dislodged compared to only 1 or 2 hairs in normal conditions.[2] TE have strongly positive hair pull test compare to FPHL which are usually show negative result in hair pull test. Occasionally, biopsy is done to rule out the differential diagnosis of FPHL and alopecia areata (AA).[1,4,5]

In the other hands, telogen effluvium can also evolve to FPHL or reveal the presence of FPHL that maybe underdiagnosed for years.[1]

**Female Pattern Hair Loss**

Female pattern hair loss (FPHL) is also known as androgenetic alopecia (AGA).[1,6] It is induced by androgens in genetically susceptible men. However, the androgens role in women with AGA is less certain and still under investigation. The onset of FPHL is after puberty.[1,4,6]

Peripheral conversion of testosterone by 5α-reductase will produced dihydrostestosterone (DHT). In susceptible follicles, DHT will bind to the androgen receptor and form hormone-receptor complex that activates certain genes. These genes will cause the gradual transformation of large, terminal follicles to the small, miniaturized follicles. Furthermore, the duration of anagen is shortened and the matrix size is decrease in FPHL. As a result, smaller follicles will be formed. These smaller follicles then will produces shorter, finer, miniaturized hairs that various in length and diameters. These miniaturized hairs become the hallmark of FPHL.[6]

The level of 5α-reductase and androgen receptors are both elevated in women and men with AGA. However, the increasing level of the hormones in women is about half of the increasing level in men which explained why the extent of hair loss in women is commonly less than in men. In the meantime, there are much higher levels of cytochrome p-450 aromatase in frontal and occipital follicle in young women compared to men. Aromatase is capable in converting the testosterone to estradiol which means less 5α-reductased products like DHT will be produced in the presence of aromatase. As a result, hair loss in women with FPHL is less severe than in men.[4,6]

There are three types of FPHL pattern which are diffuse central thinning (Ludwig type), frontal accentuation (Olsen type) and frontotemporal recession/vertex loss (male pattern or Hamilton type). The first two types which are Ludwig and Olsen types are commonly occur.[4]

Female pattern hair loss is diagnosed by several steps. The onset is gradual, the diffuse hair loss advanced slowly and usually affects frontoparietal part conducting to central thinning. The hallmark of FPHL are the sight of miniaturized or vellus hairs (thin short hairs <3cm and diameter of shaft is ≤0.3mm) at the frontoparietal region. Bald scalp is more common in men and not a feature of FPHL.[4] Hair shedding may or may not be present which can become one key to differentiate FPHL with TE. Histopathological examination once again can reveal the hallmark of FPHL which are the miniaturization of hair follicles.[4,6]

**Chronic Telogen Effluvium**

Chronic telogen effluvium is featured with extreme diffuse hair shedding in 30 to 60 years old female with prolong duration, fluctuating in course, almost-normal histology and lasts for more than 6 months.[1,2,4] The cause of CTE is usually unknown or idiopathic.[1,4]

Women with CTE usually havea record of abrupt, extreme, alarming, diffuse, and generalized hair shedding from a look like normal head. Pull test is positive at all sites of scalp during the active phase. It can be differentiate with FPHL by the lack of widening central parting and hair miniaturization. CTE is diagnosed by excluding all other fundamental reason of chronic diffuse hair loss. Histological pictures show normal results apart from slightly raised of telogen hair follicle.[4]

Table 1. Features of telogen effluvium, female pattern hair loss, and chronic telogen effluvium[4]

|  |  |  |  |
| --- | --- | --- | --- |
| Features | Telogen effluvium | Female pattern hair loss | Chronic telogen effluvium |
| Cause | Underlying trigger | Multifactorial, hereditary, hormones, age | Idiopathic |
| Onset | Abrupt | Gradual | Abrupt |
| Shedding | Excessive, diffuse, and generalized | Minimal | Excessive, alarming (hallmark) |
| Scalp appearance | Diffuse hair loss | Normal or with sparse hairs at central scalp area | Diffuse hair loss |
| Thinning | Diffuse thinning | Central thinning with or without widened central parting line | Absent, if present, it is all over |
| Bitemporal recession | Absent | Mild to moderate and only in male type FPHL, which is uncommon | Moderate to severe and common |
| Miniaturized hairs | Absent | Present (key feature) | Absent |
| Hair pull test | Strongly throughout the scalp | Usually absent, if present, only at central scalp | Present throughout in active phase |
| Trichogram | Significantly reduced anagen:telogen ratio | A:T ratio is normal or slightly reduced | Reduced A:T ratio in active phase |
| Dermascopy | No variation in shaft diameter | Marked variation in shaft diameter  Miniaturized follicles (hallmark) | No significant variation  No miniaturization |
| Biopsy | Increase in percentage of telogen hairs (11-30%), terminal: vellus (T:V) ratio normal, no miniaturization | T:V reduced (<4:1 is diagnostic) | T:V ratio normal (8:1) |
| Course | Self limited, event specific | Gradually progressive | Prolonged and fluctuating |

**Other Types of Hair Loss in Women**

Alopecia areata (AA) is another cause of hair loss in women that occur less frequently. Commonly it is present with circular patch of alopecia that may become multifocal and also can confluence into large areas. However, sometimes diffuse generalized alopecia may occur and need biopsy for confirmation.[1] AA is usually reversible, however unfortunately it tends to recur. In about 5% of women, it can progress to alopecia totalis which is total loss of scalp hair and about 1% of women may progress to become total loss of body hair which also known as alopecia universalis. The cause of AA remained uncertain although some journal report it is thought to be autoimmune.[1,5] A positive family record are discovered in 10% to 42% of cases shows that genetic factors also play a role in AA.[5]

Trichotillomania is force to pull out one's own hair. It is presents with parts of incomplete hair loss and short hair.[1,5] In women, it is usually associated with depression or anxiety.[5]

Traction alopecia is diffuse loss of hair resulting from reiterative traction on the hair by twisting or pulling action such as in tight braids, ponytails and roller. It is commonly occur in curly hair women.[5]

Other causes of hair loss include anagen effluvium, severe bacterial infections, and tinea capitis.[1]

**MANAGEMENT OF HAIR LOSS**

Generally, the treatment is based on the cause. For TE and CTE, the treatment is usually by removing the triggering factors. TE is self-limited and usually resolves in 3 to 6 month after the triggering factors are removed. CTE in the other hands may take about 3 to 10 years to resolves. For FPLH, the treatments include topical minoxidil, antiandrogens therapies and hair transplantation.[1,4,5,6]

**Minoxidil Solution**

The first option to be chosen in women with FPHL is minoxidil solution. Furthermore, minoxidil topical solution 2% has been approved by FDA to be used for women with thinning hairs. It is used for mild and up to moderate FPHL. The efficacy of minoxidil 2% have been shown in about 60% of women with FPHL by arresting hair loss and result in mild to moderate growth.[1,4,6] However, the pharmacodynamic of minoxidil in order to elevate duration of anagen and enlarge miniaturized hair follicle is still unbeknown.[4] 1ml of minoxidil solutions must be applied to dry scalp twice daily and takes about one hour to absorb.[4,6] The process need to be repeated if the hairs are wet. It may take 6 to 12 month to see the considerable improvement.[1,4,6]

Adverse effects of minoxidil include irritation, hypertrichosis over forehead and cheeks that usually uncommon and resolves within 4 months after the treatment is stopped.[1,4] Two to eight weeks after treatment, patients may have allergic contact dermatitis that will subsides shortly with continuation of treatment. Minoxidil is contraindication for pregnant and nursing women.[1,4]

Minoxidil 5% solution can be considered to be used in women who did not response with minoxidil 2%. However the usage of 5% minoxidil solution shows higher incidence of side effects such as hypertrichosis and contact dermatitis compared to minoxidil 2%. It also have not been approved by FDA to be used in women.[1,4]

**Antiandrogens Therapies**

Antiandrogens can be used along with minoxidil topical 2% in women with Ludwig stage I and II FPHL with hyperandrogenism.[4] Antiandrogens agents include cyproterone acetate, androgen-receptor blockers spironolactone, flutamide and the 5α-reductase inhibitor finasteride.[1,4] However, none of these agents have been approved by FDA to be used for FPHL. It is commonly used in Europe but not in North America. In United States, finasteride which are competitive inhibitors of type II 5α-reductase has been approved to be used in treatment of hair loss in men but not in women.[1] It is contraindication for women who are or may become pregnant since it can result in external genitalia anomaly of a male unborn. A placebo-controlled study shows non-effectiveness of finesteride when used in postmenopausal women.[4,6]

**Hair Transplantation**

Nowadays, hair transplantation has been increasingly used to treat women with FPHL.[1] Six principles qualities of donor hair are needed to be assessing before undergo hair transplantation which are follicular unit density, interfollicular unit distance, hair shaft diameter, colour, texture, and wave. The average of 2 or 3 hairs per follicular unit is needed to produce satisfactory results. Interfollicular unit of distance and diameter of hair shaft are enough to be evaluated subjectively although it can be measured objectively. The better result will be produced by coarse, wavy hair of large calibre compared to the straightway, thin, and silky hair of donor. Besides, the better scalp coverage is produced by less contrast between the scalp colour and transplanted hair.[3]

The ideal candidates for hair transplantations is moderate condition of FPHL. This type of hair loss has high donor hair up to more than forty follicular unit/cm2 in certain parts and extreme thinning just in frontal or mid frontal scalp. It is not for mild type of FPHL as the result after the transplantation is difficult to appreciate. While for the severe cases, there are no donor hair can be taken as all parts of scalp are susceptible to hair loss.[6]

However, until now there are no complete data on long-term outcomes and rates of graft hair failure after hair transplantation. The cost of hair transplantations may vary from $4,000 to $15,000 per session.[1]

**Management of Other Types Hair Loss**

The treatment of alopecia areata includes intralesional corticosteroid injections (first-line therapy for adults with scalp involvement <50%), minoxidil, anthralin (commonly used in children), psoralen, ultraviolet A (PUVA) and topical immunotherapy.[1,5]

Women with trichotillomania usually require psychological evaluation and counselling. They commonly need to take psychopharmacologic medications like fluoxetine to control the behaviour of compulsive hair-pulling.[1,5]

Hair loss caused by tinea need to be treated with systemic antifungal agents.[1] In the other hands, extreme and repetitive stress on the scalp by tight braids, ponytails and rollers should be discontinued in patients with traction alopecia.[5]

**CONCLUSION**

Hair loss is a significant problem especially in women. The care providers have to take note that hair loss can severely affect self-esteem. TE is the most common causes of hair loss in women, followed by FPHL and CTE. While other causes include alopecia areata, trichotillomania and traction alopecia. It is important to establish the real diagnosis as it will be reflected on the next step on choosing the appropriate medication.

Prescribing drugs for women need more special attention especially in women who are pregnant or nursing compared to men. Certain hair loss problems such as telogen effluvium may not need any medication intervention as it will resolve gradually after the triggering events have been ceased. Meanwhile, minoxidil topical 2% are the first option treatments and the only drugs that have been approved by FDA to be used for women with FPHL. However, the antiandrogens therapy in the other hand is contraindicated in pregnant women as it can affect the formation of external genitalia of male fetus. Nowadays, the development and improvement of technology in medical science becomes a promising key for women with hair loss. Hair transplantation has been developed in order to resolves moderate hair loss problem.

**REFERENCES**

1. Shapiro J. Hair loss in women. The New England Journal of Medicine. 2007; 357: 1620-30.
2. Harrison S, Bergfeld W. Diffuse hair loss: its trigger and management. Cleveland Clinic Journal of Medicine. 2009; 76 (6): 361-7.
3. Vogel JE. Hair transplantation in women: a practical new classification system and review of technique. Aesthetic Surgery Journal. 2002; 22: 247-59.
4. Shrivastava SB. Diffuse hair loss in an adult female: approach to diagnosis and management. Indian J Dermatol Venereol Leprol. 2009; 75 (1): 20-28.
5. Brenner FM, Bergfeld WF. Hair loss: diagnosis and management. Cleveland Clinic Journal of Medicine. 2003; 70 (8) : 705-12.
6. Price VH. Androgenetic alopecia in women*.* JID Symposium Proceedings; 2003 1 June; California, USA.
7. Shapiro J. Hair loss in women. The New England Journal of Medicine. 2007; 357: 1620-30.
8. Harrison S, Bergfeld W. Diffuse hair loss: its trigger and management. Cleveland Clinic Journal of Medicine. 2009; 76 (6): 361-7.
9. Vogel JE. Hair transplantation in women: a practical new classification system and review of technique. Aesthetic Surgery Journal. 2002; 22: 247-59.
10. Shrivastava SB. Diffuse hair loss in an adult female: approach to diagnosis and management. Indian J Dermatol Venereol Leprol. 2009; 75 (1): 20-28.
11. Brenner FM, Bergfeld WF. Hair loss: diagnosis and management. Cleveland Clinic Journal of Medicine. 2003; 70 (8) : 705-12.
12. Price VH. Androgenetic alopecia in women*.* JID Symposium Proceedings; 2003 1 June; California, USA.
13. Shapiro J. Hair loss in women. The New England Journal of Medicine. 2007; 357: 1620-30.
14. Harrison S, Bergfeld W. Diffuse hair loss: its trigger and management. Cleveland Clinic Journal of Medicine. 2009; 76 (6): 361-7.
15. Vogel JE. Hair transplantation in women: a practical new classification system and review of technique. Aesthetic Surgery Journal. 2002; 22: 247-59.
16. Shrivastava SB. Diffuse hair loss in an adult female: approach to diagnosis and management. Indian J Dermatol Venereol Leprol. 2009; 75 (1): 20-28.
17. Brenner FM, Bergfeld WF. Hair loss: diagnosis and management. Cleveland Clinic Journal of Medicine. 2003; 70 (8) : 705-12.
18. Price VH. Androgenetic alopecia in women*.* JID Symposium Proceedings; 2003 1 June; California, USA.