REVIEW

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Shedding: How to manage a common cause of hair loss

ABSTRACT

Although telogen effluvium, or shedding—the most common type of diffuse hair loss in both women and men—is usually self-limiting, the condition may become chronic if the trigger is not identified and corrected. The authors discuss the physiologic and emotional triggers, clinical presentation, diagnosis, and management strategies, including the importance of patient education and reassurance.

KEY POINTS

Shedding is not a disease; it is just a clinical sign.

Shedding is often traceable to a trigger, such as physiologic stress (eg, illness, surgery, nutrient deficiency), emotional stress, or drug therapy. It can also represent the early stages of hereditary (androgenetic) alopecia.

SHEDDING (TELOGEN EFFLUVIUM), the most common cause of diffuse hair loss in both women and men, is the premature conversion of hair follicles from the growing (anagen) phase to the resting (telogen) phase (FIGURE 1). It is usually triggered by a specific physiologic or emotional event (TABLE 1). High fever, childbirth, chronic systemic diseases, extreme emotional stress,[1] drug therapy,[1–3] metabolic disorders,[2,3] nutritional disorders,[4] surgical procedures, and anesthesia[5] are potential triggers. Shedding may also occur concomitantly with the early stages of androgenetic alopecia in women and men.[6,7]

Shedding is usually self-limiting, with management consisting of identifying the trigger and reassuring and educating the patient. In chronic shedding, further medical evaluation and therapy may be necessary.

LIFE CYCLE OF HAIR

Normal cycle

Each hair grows from a hair root at the base of a hair follicle. In young healthy people, hair grows at a rate of approximately 0.35 mm/day,[8] or about 1 inch every 2 months.

Each hair follicle has its own individual three-phase life cycle, consisting of a growing phase, an involuting phase, and a resting phase (FIGURE 1). The growing phase for scalp hair can last 2 to 5 years. More than 85% of scalp hair is in this phase at any time. The involution phase is brief, lasting 3 to 6 weeks. The resting phase lasts 3 to 5 months. Up to 15% of follicles are in the resting phase at any time.[8–10] At the end of the resting phase, the hair sheds as a new cycle begins.

A certain amount of hair loss is normal.

About 100 follicles enter the resting phase every day, and these hairs eventually fall out[9]; the same number enters active growth every day.

Since at all times some hairs are growing and some are resting (and shedding), the total number of hairs and their density are constant. The duration of the growing phase determines both hair length (in the absence of haircutting) and the number of hairs in the resting phase.

Triggers that disturb the hair's normal cycle

A disturbance in this cycle may produce premature conversion of growing hairs to resting hairs. In the case of shedding, this may mean a 20% to 30% increase in the amount of hair shed daily.[1,2,8]

As shown in TABLE 1, a wide variety of events and conditions can trigger disturbances in the hair's life cycle.

Drugs. Any drug can potentially cause hair loss, but some drugs are more often associated with shedding (TABLE 2).[2,3]

Nutrient deficiencies that commonly trigger and perpetuate shedding episodes include iron, zinc, magnesium, protein, essential fatty acids, and vitamins.[11,12] Vitamin D and B deficiencies are associated with diffuse hair loss. On the other hand, shedding and dry skin are observed in hypervitaminosis A; in adults, as little as 25,000 IU daily may lead to toxicity and hair loss, especially in persons with hepatic compromise.[11]

Hormonal changes that occur after pregnancy often trigger acute episodes of shedding. Endocrine disturbances in patients with thyroid disease, in menopause, or in the early stages of androgenetic alopecia can also trigger shedding.[2,8]

DIAGNOSIS: IDENTIFY THE TRIGGER

Shedding is more commonly identified in women than in men because women more often report early signs of hair loss. However, shedding also occurs in men, particularly in those with early androgenetic alopecia.[2]

In most cases, the patient observes more hair on the shower drain, on clothes, or on bed pillows. Usually, patients begin to notice a decrease in hair density or volume only when

TABLE 1

Causes and triggers of shedding (telogen effluvium)

- Hormonal changes after pregnancy
- Early stages of androgenetic alopecia
- Fever
- Drug therapy
- Infection
- Chronic illness
- Emotional stress
- Major surgery
- Metabolic and endocrine disorders
- Autoimmune diseases
- Nutrient deficiencies

TABLE 2

Drugs and drug classes often associated with shedding (telogen effluvium)

- 5-Fluorouracil
- Albendazole
- Aminosalicylic acid
- Amiodarone
- Amphetamine
- Antidepressants (SSRIs, tricyclics)
- Beta-blockers
- Bleomycin
- Bromocriptine
- Captopril
- Carbamazepine
- Carmustine
- Cimetidine
- Colchicine
- Coumadin
- Cyclophosphamide
- Cytarabine
- Dactinomycin
- Danazol
- Doxorubicin
- Enalapril
- Etoposide
- Gentamicin
- Gold salts
- Heparin
- Hydroxyurea
- Ibuprofen
- Interferons
- Isoniazid
- Lipid-lowering agents
- Lithium
- Methotrexate
- Methyldopa
- Methysergide
- Minoxidil
- Nicotinic acid
- Nitrofurantoin
- Nitrosourea
- Oral contraceptives
- Proguanil
- Pyridostigmine
- Retinoids
- Sulfasalazine
- Thiouracil
- Trimethadione
- Valproic acid
- Verapamil
- Vinblastine
- Vincristine
- Vindesine

MODIFIED FROM HEADINGTON JT. TELOGEN EFFLUVIUM. ARCH DERMATOL 1993; 129:356–363.

Loss of 100 hairs a day is normal

The hair growth cycle and shedding

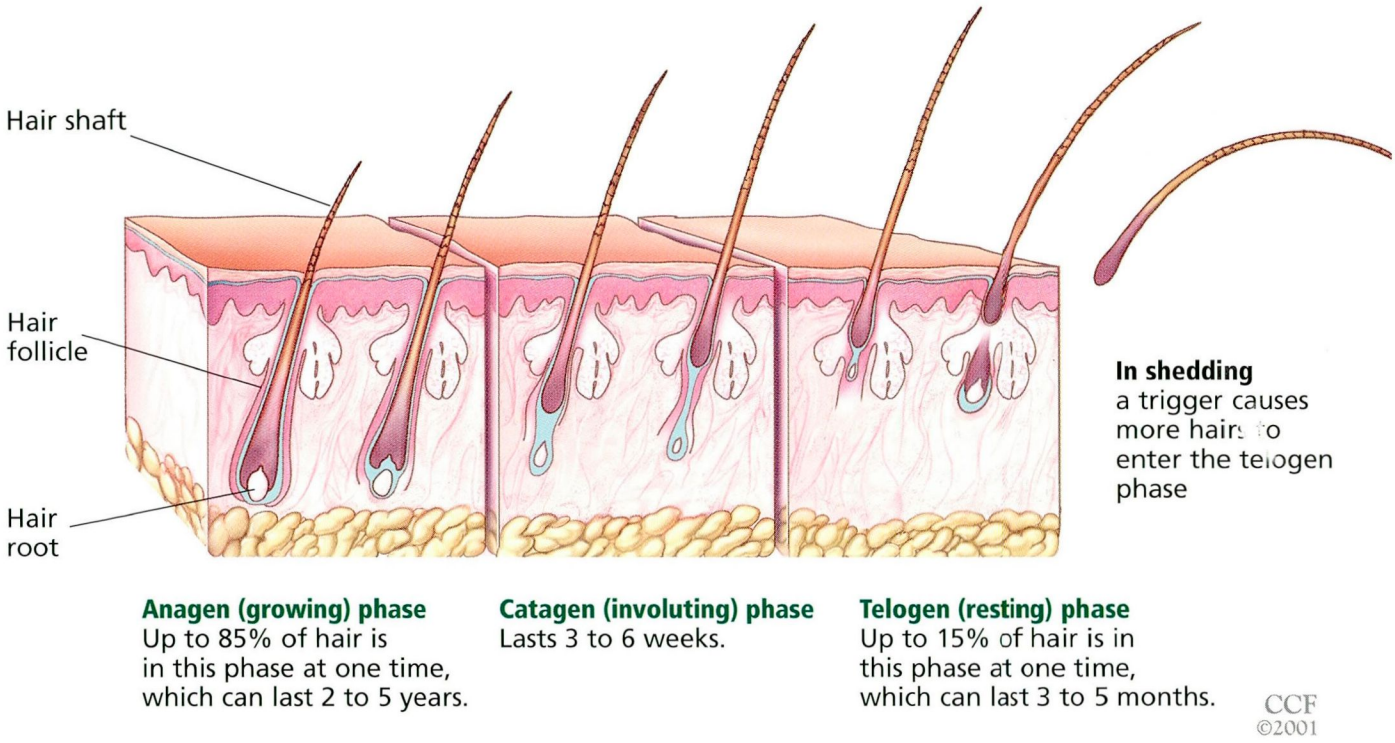

FIGURE 1. Hair cycle. Each hair follicle has its own individual three-phase life cycle, with a growing (anagen) phase, an involution (catagen) phase, and a resting (telogen) phase. Hair falls out during the telogen phase. The growing phase for scalp hair can last 2 to 5 years, and 85% of scalp hair is in this phase at any time. In shedding, a trigger causes a higher proportion of hairs to enter the telogen phase.

Ask about drugs and dietary restrictions

hair loss reaches 20% to 30%. Clinicians, however, may not recognize a reduction in the patient's normal hair density until thinning reaches 30% to 50%.[2,13]

The goal of diagnosis is to correlate the volume of hair loss, duration, and previous episodes with triggers (TABLES 1 AND 2).

Drugs. Ask what prescription and nonprescription drugs the patient is taking, including vitamins and over-the-counter and homeopathic medications, and their dosages. Discontinuation of prescription and nonprescription drugs may be necessary to halt shedding.

Diet. Patients should be asked if they are following a restricted diet. Vegetarians often present with hair shedding, most likely associated with iron and protein deficiency.[4]

Menstrual history. In women, a careful menstrual history should be taken. Heavy periods can lead to an underlying iron deficiency, and amenorrhea can be associated with endocrine disorders. The clinician should inquire about the use of oral contraceptives, a recent pregnancy or miscarriage, any hair loss after previous pregnancies, signs of androgen excess, and polycystic ovaries.

Androgenetic alopecia. In both sexes, a family history of baldness should be sought, as both acute and chronic shedding can overlap with androgenetic alopecia. A fluctuating and prolonged course of chronic shedding may herald androgenetic alopecia.[6,7]

Other diseases. Autoimmune diseases, hepatic and renal disorders, and chronic infections such as syphilis should be excluded by history or complementary exams.[4–6]

Onset and duration of shedding. Patients with shedding most likely recall when the hair loss began and often will report having a sudden increase in hair shedding. In contrast, the hair loss characteristic of androgenetic alopecia is continuous, and thinning is more evident than shedding.

Laboratory evaluation

Screening laboratory evaluation should include a complete blood count, a metabolic panel, and thyroid evaluation. Testing blood ferritin levels is a good way to measure and

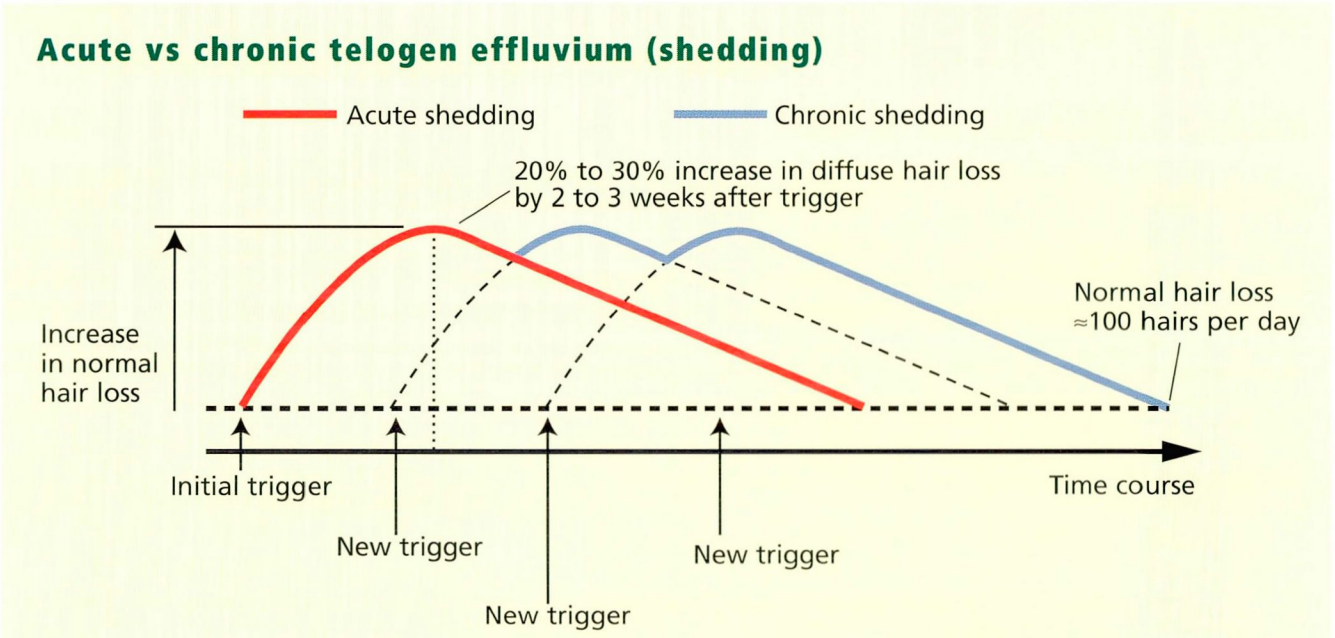

FIGURE 2. Normal hair loss (100 hairs a day) increases immediately after a trigger, although the increase may not be noticed until 2 to 3 weeks later. Acute shedding usually lasts 2 to 6 months and is followed by complete recovery in 12 to 18 months. If new triggers occur and the causes are not removed or treated, hair loss does not fully resolve, and shedding may persist for years.

follow the status of iron storage: when the quantity of iron in the plasma falls, iron is removed from ferritin quite easily.[14] This situation is frequent in women during menstrual periods and crash diets.

Measuring hair loss

Several approaches may be used to measure hair loss.

A count of the hairs shed daily can be done, and is usually around 100 to 200.[9,10]

The hair pull test should be done in all patients with hair loss. This simple technique involves the gentle traction from the base to the terminal ends of a group of 25 to 50 hairs. Normally only 1 or 2 shafts are dislodged. However, in shedding 10 or 15 shafts may be dislodged.

Scalp biopsy. Failure of the history to identify a trigger, a persistently positive pull-test, evidence of decreased hair shaft diameter (which may indicate androgenetic alopecia), and concern for possible alopecia areata are all indications for scalp biopsy.[2] Punch biopsy is preferred, and evaluation by an experienced dermatopathologist is recommended.

Biopsy specimens in acute shedding show increased resting-phase follicle counts, with an anagen/telogen ratio of about 1:4 (approximately the inverse of the normal ratio) and a decreased number of hair follicles.[10,15] The presence of fibrous tracts (hair sacs) without hair follicles suggests chronic shedding. When regeneration is not clinically evident, biopsy specimens show a diminished number of hair follicles and adnexal structures, and increased fibrous tracts and vellus or miniaturized hair follicles.[10,16]

REASSURING AND EDUCATING THE PATIENT

Reassure the patient that shedding does not lead to baldness

A key part of management is reassuring and educating the patient about the typical course of shedding. Acute shedding usually lasts 2 to 6 months and is followed by complete recovery, without the need for intervention.[1,13] Chronic forms of shedding may last months to years and may require therapy, as discussed below.[2,5,6,10]

TABLE 3

Management of shedding (telogen effluvium)

Identify triggers: patient should keep a "health calendar"
Remove or treat the trigger
Educate patient about hair's growth and life cycle
Treat nutrient deficiencies (eg, iron, vitamins, protein)
Prescribe minoxidil to promote hair growth and improved self-image
When appropriate, prescribe anti-inflammatories (steroids, tar and zinc pyrithione shampoo) or antiandrogens (spironolactone, flutamide, estrogen)

Shedding increases right after a trigger, although it is only noted when highly increased, 2 or 3 weeks after that. Even if the cause is removed or treated, the amount of shedding will be high for up to 6 months (FIGURE 2).

Regrowth and enhanced aesthetic appearance take 12 to 18 months, depending on the patient's hairstyle. However, if a new episode is triggered or if the cause is not removed, shedding may persist for years.

Acute shedding triggered by stress is often self-limited

MANAGEMENT STRATEGIES

The management strategy depends on whether the diagnosis determines that the shedding is acute or chronic. Acute shedding is usually defined as an episode of hair loss lasting from 2 to 6 months. Shedding is considered chronic if it lasts longer than 6 months. In some cases, shedding may persist for years. In either case, reassuring the patient that the condition will not lead to baldness and that hair is being replaced is a key part of management (TABLE 3).

Acute shedding

Acute shedding triggered by drugs, emotional stress, or illnesses is often self-limited and requires no treatment other than eliminating the trigger. An underlying scalp condition such as seborrheic dermatitis should be controlled. Drugs suspected of triggering the shedding should be discontinued or changed, if possible.

Chronic shedding

Because of the potentially long duration of chronic shedding, the search for triggers is more difficult and may require frequent repeat visits. Patients should be reassured that the shedding hairs are being replaced and that the chances of becoming bald are remote. Encourage patients to keep a "health calendar" to record and grade shedding, stress, illnesses, and drug intake (changes in dosage, starting, stopping), which is very helpful in identifying triggers.

If a nutrient deficiency is identified, the patient should receive nutrient supplementation. Vitamin supplements, especially the B group, have shown good results in diffuse shedding.[17] Multivitamins with 3 to 5 mg of biotin (vitamin B_7) are preferred. If scalp erythema or scaling is present, anti-inflammatory shampoos (tar and zinc pyrithione) and topical steroids are recommended. Topical minoxidil, a hair promoter, can be helpful.

Systemic treatment

Systemic treatment is controversial but can be used in severe or chronic shedding. Corticosteroids may be given systemically or as intradermal injections. Prednisone 10 to 20 mg/day can reduce severe acute shedding.[13]

Some reports suggest that antiandrogen medications such as spironolactone (100–300 mg/day) and flutamide (250–500 mg/day) in combination with birth control pills are helpful, especially in women with androgen excess, where the shedding may reflect androgenetic alopecia.[5,13,18]

These treatments, if successful, may produce a noticeable decrease in shedding within 8 weeks. However, patients with shedding must be carefully followed to identify new triggers and causes.

REFERENCES

1. **Kligman AM.** Pathologic dynamics of human hair loss. I. Telogen effluvium. Arch Dermatol 1961; 83:175–198.
2. **Headington JT.** Telogen effluvium. Arch Dermatol 1993; 129:356–363.
3. **Pillans PI, Woods DJ.** Drug induced alopecia. Int J Dermatol 1995; 34:149–158.
4. **Goette DU, Odum RB.** Alopecia in crash dieters. JAMA 1976; 235:2622–2623.
5. **Whiting DA.** Chronic telogen effluvium. Dermatol Clin 1996; 14:723–731.

6. **Whiting DA.** Chronic telogen effluvium: increased scalp hair shedding in middle-aged women. J Am Acad Dermatol 1996; 35:899–906.
7. **Rand S.** Chronic telogen effluvium: potential complication for clinical trials in female androgenetic alopecia? [Letter] J Am Acad Dermatol 1997; 37:1021.
8. **Sperling LC, Mezebish DS.** Hair disease. Med Clin North Am 1998; 82:1155–1169.
9. **Epstein P, Cotsarelis G.** The biology of hair follicles. N Engl J Med 1999; 341:491–497.
10. **Bergfeld WF.** Alopecia. Histologic changes. Adv Dermatol 1989; 4:301–320.
11. **Odom RB, James WD, Berger TG.** Nutritional diseases. In: Andrew's diseases of the skin. 9th edition. Philadelphia: W.B. Saunders, 2000:606–615.
12. **Arnaud J, Beani JC, Favier AE, Emblard P.** Zinc status in patients with telogen defluvium. Acta Derm Venereol 1995; 75:248–249.
13. **Rebora A.** Telogen effluvium. Dermatology 1997; 195:209–212.
14. **Guyton AC, Hall JE.** Red blood cells, anemia, and polycytemia. In: Textbook of medical physiology. 10th edition. Philadelphia: W.B. Saunders, 2001:382–391.
15. **Sperling LC.** Hair anatomy for the clinician. J Am Acad Dermatol 1991; 25:1–17.
16. **Solomon AR, Templeton SF.** Alopecia. In: Farmer ER, Hood AF. Pathology of the skin. 2nd edition. New York: McGraw-Hill, 2000:833–858.
17. **Budde J, Tronnier H, Rahlfs VW, Frei-Kleiner S.** Systemic therapy of diffuse effluvium and hair structure damage. Hautarzt 1993; 44:380–384.
18. **García-Hernández MJ, Camacho FM.** Chronic telogen effluvium: incidence, clinical and biochemical features, and treatment. Arch Dermato 1999; 135:1123–1124.

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