CLINICAL PRACTICE

Hair Loss in Women

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This Journal feature begins with a case vignette highlighting a common clinical problem. Evidence supporting various strategies is then presented, followed by a review of formal guidelines, when they exist. The article ends with the author's clinical recommendations.

A 45-year-old white woman presents with a 1-year history of scalp-hair loss. She was hospitalized with appendicitis 14 months ago. She has been a vegetarian for 20 years. She takes no medications. Her father was bald. On physical examination, she has diffuse, nonscarring hair thinning with a widened part over the central portion of the scalp. How should this problem be evaluated and treated?

THE CLINICAL PROBLEM

Hair loss, or alopecia, is a very common presenting symptom, and more than one third of women have clinically significant hair loss during their lifetime. The effect of hair loss on patients' emotions is often greatly underestimated by physicians.

After bone marrow, hair is the second fastest growing tissue of the body. As a result, many metabolic derangements can be manifested with alopecia, and hair loss may be the first clinical sign of systemic disease.

HAIR BIOLOGY

The scalp contains, on average, 100,000 hairs. More than 90% of these hairs are actively growing, and they are referred to as anagen hairs. Anagen hairs are anchored deeply into the subcutaneous fat and cannot be pulled out easily. Hair is constantly cycling and regenerating on the scalp. Each hair shaft may persist on the scalp for 3 to 7 years before falling out and being replaced by a new hair. The anagen phase, which lasts for most of this period, is followed by a 2-week phase of catagen, during which there is programmed apoptosis; the trigger factor for catagen is unknown. After catagen, the hair goes into telogen, a resting phase that lasts 3 months. As compared with anagen hair, telogen hair is located higher in the skin and can be pulled out relatively easily. Normally, the scalp loses approximately 100 telogen hairs per day.

In addition to the ratio of anagen hair to telogen hair, the diameter of the hair follicles determines scalp coverage. Vellus hairs have a hair-shaft diameter of less than 0.03 mm, whereas terminal hairs have a diameter greater than 0.06 mm. The optimal hairs for scalp-hair growth and scalp coverage are anagen and terminal hairs.

CAUSES OF HAIR LOSS

Hair loss is typically categorized as scarring (which occurs in discoid lupus, lichen planopilaris, and folliculitis decalvans)¹ or nonscarring. This review focuses on non-scarring alopecia.

The most common cause of such hair loss, female-pattern hair loss, is frequently referred to as androgenetic alopecia; however, the role of androgens in this type of hair loss remains uncertain.^{2,3} This condition is often familial.² Female-pattern hair loss can develop any time after the onset of puberty³; by 70 years of age, 38% of

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women have female-pattern hair loss.⁴ It affects the central portion of the scalp, sparing the frontal hairline, and is characterized by a wider midline part on the crown than on the occipital scalp (Fig. 1). In some women, hair thinning over the lateral area of the scalp also occurs. The severity of hair loss is staged according to the Ludwig classification,⁵ in which increasing stages (I to III) correspond to increasing widths of the midline part. If hair thinning is more evident in the frontal portion of the scalp, the part may resemble a fir tree in what is known as a "Christmas tree pattern" behind the frontal hair line (Fig. 2). This pattern is referred to as "frontal accentuation."³

Other manifestations of hair loss include hair thinning on the lateral scalp and male patterns involving thinning on the frontotemporal and vertex areas of the scalp. Male patterns of hair loss may be associated with hyperandrogenism, but the majority of women with female-pattern hair loss have normal serum androgen levels.⁶⁻⁸ One study⁶ showed a prevalence of biochemical hyperandrogenemia of 38.5% among women with moderateto-severe alopecia; approximately one quarter of these women had no other signs of hyperandrogenemia, such as hirsutism or menstrual disturbances.

Another common cause of alopecia is telogen effluvium. This condition results from an abrupt shift of large numbers of anagen hairs to telogen hairs on the scalp, with a corresponding change in the ratio of anagen hair to telogen hair from the normal ratio of 90:10 to 70:30. It is not unusual for women with telogen effluvium to lose more than 300 hairs per day. This form of alopecia generally begins approximately 3 months after a major illness or other stress (e.g., surgery, parturition, rapid weight loss, nutritional deficiency, high fever, or hemorrhage) or hormonal derangement (e.g., thyroid dysfunction); it has also been reported after the initiation of treatment with certain medications (Table 1). This process is distinct



Figure 1. Marked Thinning of Hair on the Crown of the Scalp in a Woman with Female-Pattern Hair Loss and Fairly Normal Occipital Density.

The centroparietal portion of the scalp, which shows decreased hair density (Panels A and B), would be classified as Ludwig stage II (a moderately widened central part). In this patient, hair thinning also extends laterally (Panels C and D).

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Figure 2. Frontal Accentuation of Hair Loss in Female-Pattern Hair Loss.

from anagen effluvium, the hair loss associated with chemotherapeutic agents that cause immediate destruction and release of anagen hair.

If the cause of telogen effluvium is removed, hair loss lasts for up to 6 months after removal of the trigger. Chronic telogen effluvium refers to hair loss lasting more than 6 months. In some patients, this type of hair loss lasts for years.¹⁰ Prolonged telogen effluvium may be due to multiple sequential triggers, although in some patients, no trigger is identified.¹⁰ Telogen effluvium may evolve into or reveal female-pattern hair loss, but the frequency of such cases is unclear.

A less frequent cause of nonscarring alopecia is alopecia areata. The estimated lifetime incidence of this condition is 1.7% (Fig. 3).¹¹ It is usually manifested as round patches of alopecia that may become multifocal and may coalesce into large areas affecting more than 50% of the scalp. Occasionally, there is diffuse generalized alopecia, requiring a scalp biopsy for confirmation. Alopecia areata is frequently reversible, but it tends to be recurrent, and it can progress to total loss of scalp hair (alopecia totalis) in 5% of women and total loss of body hair (alopecia universalis) in 1% of women. The cause is unknown, but it is thought to be autoimmune. Other causes of nonscarring alopecia are certain hair-care practices (Table 2), compulsive hair pulling (trichotillomania), severe bacterial infections, tinea capitis, and, in rare cases, abnormalities causing fragility or breakage of irregularly shaped hair.

In some cross-sectional studies, iron deficiency and reduced iron levels have been associated with hair loss, including female-pattern hair loss and telogen effluvium, but data are limited.¹²⁻¹⁴ Such a relationship might be explained by the observation that iron is required as a cofactor for the activity of ribonucleotide reductase, a rate-limiting enzyme controlling DNA synthesis and required by rapidly dividing hair matrix cells.

STRATEGIES AND EVIDENCE

EVALUATION

The history taking should include an assessment of the duration and pattern of hair loss, including whether hair is shedding (suggesting alopecia areata or telogen effluvium) or is primarily thinning (suggesting female-pattern hair loss). It is important to establish whether the hair is falling out from the root (suggesting telogen effluvium, female-pattern hair loss, or alopecia areata) or breaking off along the shafts (as occurs with certain hair-care practices, trichotillomania, or tinea capitis) (Table 2).

Patients should be asked about hair-care practices that may damage hair (e.g., braiding that causes traction alopecia), as well as about the loss of eyelashes, eyebrows, and axillary, pubic, or body hair, since any hair-bearing area can be affected by alopecia areata or trichotillomania. A history of illness, childbirth, surgery, psychosocial stress, or a new medication predating the onset of hair loss by 1 to 3 months suggests telogen effluvium. Acne, irregular menstrual cycles, or hirsutism may indicate androgen excess contributing to femalepattern hair loss. Symptoms of hyperthyroidism or hypothyroidism should also be assessed, and current and previous medications should be carefully reviewed (Table 1). A history of following a strict vegetarian diet or heavy menses may suggest iron-deficiency anemia.

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Table 1. Medication	s Associated with Ha	ir Loss.*	
Type of Hair Loss	Interval between Start of Treatment and Hair Loss	Medications	Estimated Incidence (%)
Telogen effluvium	2–3 mo	Acetretin, heparin, interferon alfa, isotretinoin, lithium, ramipril, terbinafine, timolol, valproic acid, warfarin	>5
		Acyclovir, allopurinol, buspirone, captopril, carbamazepine, cetirizine, cyclosporine, gold, lamotrigine, leuprolide, lova- statin, nifedipine	1–5
		Amiodarone, amitriptyline, azathioprine, dopamine, naproxen, omeprazole, paroxetine, prazosin, sertraline, venlafaxine, verapamil	<1
Anagen effluvium	7–14 days	Bleomycin, busulfan, cisplatin, cyclophosphamide, daunoru- bicin, doxorubicin, fluorouracil, vasopressin, vinblastine, vin- cristine	>10

* This list is not comprehensive. Data are from Litt.⁹



Figure 3. Alopecia Areata with Round, Random Patches of Hair Loss That Coalesce.

The clinical examination should be performed in four stages. First, the scalp should be inspected for inflammation, scale, and erythema (Fig. 4). Next, scarring associated with hair loss should be assessed. Nonscarring alopecia (Fig. 5A) is characterized by visible follicular openings (ostia), whereas scarring alopecias (Fig. 5B) are devoid of ostia. The third step is to examine the pattern of distribution of hair loss and the density of hair, and the fourth step is to assess the quality of the hair shaft in terms of caliber, fragility, length, and shape. If the hair tips are blunt, hair breakage may be implicated. Tapered tips are normal. To assess the ongoing activity and severity of hair loss, a pull

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Table 2. Characteristi	cs of Nonscarring Hair Loss.				
Characteristic	Female-Pattern Hair Loss	Telogen Effluvium	Alopecia Areata	Tinea Capitis	Hair-Care Practices, Traction Alopecia, or Trichotillomania
Distribution	Usually central portion of the scalp, sparing the frontal hairline (less commonly, hair thinning on the lateral, frontotemporal, or vertex portions of the scalp)	Generalized	Usually patchy, but may be multifocal and patches may co- alesce; total alopecia in 5–10% of patients with this condition	Any area of the scalp; may be focal or multifocal	Any area of the scalp; may be patchy with irregular angular borders; trac- tion alopecia frequently affects the frontal and temporal edges of the scalp
Onset	Gradual with progression	Abrupt with a trigger factor (e.g., blood loss, iron deficien- cy, thyroid imbal- ance, or initiation of drug treatment)	Abrupt, usually waxes and wanes	Gradual or abrupt	Gradual or abrupt, depending on the cause
Appearance	Hair thinning with or without bare patches; wide midline part on the crown	Hair thinning with no bare patches	Hair thinning with abrupt bare patches; "excla- mation point" hairs	Inflammation or no inflam- mation; scale present	Broken hairs with blunt rather than ta- pered tips; degree of inflammation due to hair-care practices depends on the offending agent; no inflam- mation with traction alopecia or trichotillomania
Degree of shedding	Minimal	Prominent	Prominent	Prominent	Broken hairs can be shed; varies with offending hair-care agent; minimal with traction alopecia and trichotil- lomania
Patient's age at onset	Puberty or older	Any age, but not common in childhood	Any age; most patients have first patch be- fore 20 yr of age	Any age; common in childhood	Any age
Result of pull test	Usually negative	Positive	Positive	Positive	Usually negative
Other history	Often family history of hair loss	Previous major ill- ness or stress	May be personal or family history of other autoimmune disease	Previous contact with ani- mals (e.g., kittens) as- sociated with certain dermatophytes	Back brushing (i.e., brushing or comb- ing hair in a direction different from that of hair growth); use of perma- nent waves, bleach, or relaxants or braiding; trichotillomania may be associated with other psychiatric conditions

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Figure 4. Marked Scarring Hair Loss with a Central Distribution, Follicular Hyperkeratosis, and Pustules in a 44-Year-Old Woman.

A biopsy specimen revealed folliculitis decalvans, a rare permanent scarring condition that occurs with folliculitis.

test (Fig. 6) should be performed. Table 2 summarizes characteristics that help in distinguishing among common nonscarring hair-loss conditions.

LABORATORY TESTING

Clinicians often assess the ferritin level to rule out iron deficiency (particularly in menstruating women, vegetarians, and women with a history of anemia¹³⁻¹⁵) and the thyrotropin level to rule out thyroid dysfunction in women with diffuse hair loss, although the yield of such universal testing has not been proved. If the ferritin level is less than 70 ng per milliliter, iron supplementation is recommended.15 However, its effects on hair loss and regrowth have not been rigorously evaluated in controlled trials; data suggestive of efficacy are limited to case series indicating cessation of hair loss and new hair growth with iron supplementation in women with low ferritin levels,16 and not all case series have shown a benefit of iron supplementation.12

In women with female-pattern hair loss and other conditions suggesting androgen excess (e.g., hirsutism, acne, or irregular menses), assessment of free testosterone is recommended; the yield of testing is expected to be low in the absence of other features suggesting androgen excess. A Venereal Disease Research Laboratory test is recommended if the patient has any risk factors for syphilis.

If tinea is suspected, scale from the area of alopecia should be examined by means of a potassium hydroxide scraping for hyphae and sent for



culture. Hair shafts should be plucked for culture as well. Examination with a Wood's lamp will show a green fluorescence if a specific group of dermatophytes (*Microsporum canis*) is present.

If the diagnosis remains in question, a 4-mm punch biopsy of tissue from the scalp may be useful. This test is especially useful when evaluating patients suspected of having scarring alopecia.

MANAGEMENT

Therapies for female-pattern hair loss include topical minoxidil, antiandrogen medication, and hair transplantation in selected patients. Baseline photographs (typically of the midline part) should be taken and used on subsequent visits for comparison. Six months to 1 year of treatment may be required before there is considerable improvement.

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The examiner grasps approximately 60 hairs (Panel A) and tugs at them from proximal to distal ends (Panels B and C). Removal of more than six hairs indicates a positive pull test and active hair loss, as shown in this patient (Panel D).

MINOXIDIL SOLUTION

Topical 2% minoxidil solution is approved by the Food and Drug Administration (FDA) for women with thinning hair due to female-pattern hair loss. In a double-blind, placebo-controlled trial, 2% minoxidil used twice daily resulted in minimal hair regrowth in 50% of women and moderate hair regrowth in 13% of women after 32 weeks of treatment,¹⁷ as compared with rates of 33% and 6%, respectively, in the placebo group (P<0.001). Efficacy can be assessed definitively after 6 to 12 months of treatment. Side effects of topical minoxidil therapy include contact dermatitis (attributed in many cases to irritation from propylene glycol in the solution) and symmetric facial hypertrichosis manifested as fine hairs on the cheeks or forehead in up to 7% of women. Hypertrichosis disappears within 4 months after discontinuation of the drug. Minoxidil should not be used in pregnant or nursing women.

The use of 5% minoxidil (Fig. 7) may be considered in women who do not have a response to the 2% formulation or who want more aggressive management.18 A double-blind, randomized trial comparing a 5% minoxidil solution with a 2% minoxidil solution used twice daily in women with mild-to-moderate female-pattern hair loss showed no significant difference between the two solutions with respect to investigator assessments of efficacy, but it showed significantly greater patient satisfaction with the 5% preparation.¹⁸ However, the incidences of hypertrichosis and contact dermatitis were higher with the 5% solution than with the 2% solution. A new 5% minoxidil foam formulation that contains no propylene glycol appears to be much less likely to cause contact dermatitis than topical minoxidil solution. Although they are prescribed by many dermatologists in practice, neither the 5% minoxidil solution nor the foam preparation is FDAapproved for use in women.

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Figure 7. Female-Pattern Hair Loss with Frontal Accentuation before and after Treatment with Topical 5% Minoxidil Solution in a 58-Year-Old Woman.

Panel A shows hair loss before treatment, and Panel B shows regrowth after 6 months of treatment.

ANTIANDROGEN THERAPIES

Antiandrogen agents (including the androgenreceptor blockers spironolactone, cyproterone acetate, and flutamide and the 5α -reductase inhibitor finasteride) and oral contraceptives are not commonly used to treat female-pattern hair loss in North America, but they are used more commonly in Europe. None of these agents are FDAapproved for female-pattern hair loss. Cyproterone acetate is not approved in the United States, and neither flutamide nor finasteride is approved for any indication in women, although finasteride is approved for the treatment of hair loss in men.

In an open-label study of cyproterone acetate (50 to 100 mg daily for 10 days of the menstrual cycle) or spironolactone (200 mg daily) in women with female-pattern hair loss,¹⁹ more than 80% of women had either hair regrowth or stabilization of hair loss, but this study was uncontrolled. In a

randomized trial comparing topical 2% minoxidil solution plus an oral contraceptive with cyproterone acetate (52 mg per day) plus an oral contraceptive in women with female-pattern hair loss, the latter combination resulted in greater hair density in women with hyperandrogenism, whereas in women without hyperandrogenism, minoxidil had a greater effect.²⁰ If antiandrogen agents are used in women of reproductive age, an oral contraceptive should be prescribed concomitantly, since these agents are known teratogens.

In two small, uncontrolled studies, finasteride (Propecia) at a minimum dose of 2.5 mg per day appeared to have a benefit for women with femalepattern hair loss.^{21,22} However, in a double-blind, controlled trial²³ involving postmenopausal women with female-pattern hair loss, treatment with finasteride at a dose of 1 mg per day was not significantly better than placebo. Like the antiandrogens,

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finasteride is a known teratogen, and its use is not recommended in women of reproductive age.

HAIR TRANSPLANTATION

Hair surgery is increasingly used to treat many women with female-pattern hair loss.²⁴ Clinical experience indicates that when the newer technique of follicular-unit transplantation is performed by an experienced surgeon, a natural result is possible (Fig. 8). However, data on long-term outcomes are lacking, and rates of graft failure, although considered to be very low, remain uncertain. Costs vary, but they may range from \$4,000 to \$15,000 per session, depending on the size of the area treated and the surgeon. One or two sessions are usually sufficient for a cosmetically acceptable result. Hair density in the donor (occipital) area must be sufficient to yield the required number of grafts



Figure 8. Female-Pattern Hair Loss before and after Hair Transplantation in a 45-Year-Old Woman.

Panel A shows hair loss before transplantation, and Panel B shows the results, at 9 months, of grafting with 1575 follicular units. with no visible scarring. Complications, which are rare, include infection, permanent scalp dysthesias, and arteriovenous malformations (which occur in less than 1% of patients). Many surgeons use minoxidil therapy in patients who have undergone hair transplantation (Fig. 9), although this strategy has also not been rigorously studied.²⁵

TREATMENT OF OTHER CAUSES OF HAIR LOSS

Tinea resulting in hair loss is treated with systemic antifungal agents. Adverse hair-care practices should be discontinued. Detailed discussions of the treatments of trichotillomania and alopecia areata are beyond the scope of this article. Briefly, trichotillomania may improve with counseling, cognitive behavioral therapy, or pharmacotherapy (e.g., antidepressants).26,27 For alopecia areata, treatment depends on the extent of scalp involvement. Limited patches affecting less than 50% of the scalp are generally treated with intralesional corticosteroid injections, whereas for more extensive scalp-hair loss, treatments include topical minoxidil solution, anthralin, psoralen and ultraviolet A (PUVA) therapy, and topical immunotherapy with a contact sensitizer or allergen; data on optimal therapy are limited.11,28

AREAS OF UNCERTAINTY

There are limited data from randomized, doubleblind, controlled trials to evaluate and compare various therapies for female-pattern hair loss. The roles of iron deficiency in causing hair loss and iron supplementation in treatment remain uncertain. Critical evaluation of graft survival and other outcomes of hair transplantation is needed.

GUIDELINES

The American Academy of Dermatology published guidelines in 1996 for the management of hair loss in women,²⁹ but these guidelines antedated many current treatment options. An updated review of the evaluation and treatment of female-pattern hair loss was published in 2005.³⁰ Guidelines for the treatment of alopecia areata have been issued by the British Association of Dermatologists.²⁸

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Figure 9. Hair Transplantation with Grafts Obtained from an Elliptical Strip from the Back of the Scalp.

An elliptical strip averaging in size from 8 to 14 mm wide and 8 to 12 cm long is excised from the occipital portion of the scalp (Panels A and B). This strip is subdivided into 1200 to 2000 follicular units of two to three hairs each (Panel C). Slits are made with a tiny spear or needle (Panel D). The needle is then removed, and follicular units are planted in these slits (Panel E). With appropriate placement and orientation of follicular units, it is possible to increase hair density from Ludwig stage II (Panel F) to Ludwig stage I (Panel G) in a patient with female-pattern hair loss.

CONCLUSIONS AND RECOMMENDATIONS

Determining the cause or causes of hair loss in women can be difficult and should be guided by the patient's history — including the pattern of hair loss, other medical conditions, the use of hair treatments, and the family history of hair loss — as well as by the physical examination. The history of the patient in the vignette suggests telogen effluvium from appendicitis or iron deficiency related to her vegetarian diet; her family history suggests female-pattern hair loss. Her hair loss on the central portion of the scalp also suggests female-pattern hair loss, which may have been revealed by telogen effluvium. Although data are lacking to provide support for routine testing and treatment for iron deficiency in the management of hair loss, I would check the patient's ferritin and thyrotropin levels.

Although objective data are lacking to show the superiority of 5% minoxidil solution over 2% minoxidil solution, on the basis of clinical experience and reports of greater patient satisfaction with the former, I would initiate treatment with topical 5% minoxidil solution twice daily, with the plan to continue this treatment indefinitely if there is evidence of efficacy within 1 year. If the results are unsatisfactory, hair transplantation might be considered, if this procedure is available and affordable to the patient and if the hair in the donor area has sufficient density. I would carefully review the patient's expectations regarding therapy, with attention to the magnitude of improvement that can be realistically anticipated. Results of treatment are usually seen in 6 months to 1 year.

Dr. Shapiro reports receiving consulting fees from Pfizer. No other potential conflict of interest relevant to this article was reported.

The North American Hair Research Society Web site (www. nahrs.org) is a resource for patients with hair loss.

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