Hair Loss in Women: Common Causes, Diagnosis, and Management

Susan L. Mayne, DNP, FNP-C, DCNP[a] | Loren E. Shannon, FNP-C[a]

[a]University Hospitals of Cleveland, Cleveland, OH

Hair loss in women is a complicated condition that can significantly impact quality of life and psychological well-being. Women's health nurse practitioners are often the first point of contact for women who have concerns about hair loss. Conducting initial assessments, detecting red flags for scarring hair loss conditions, and making timely referrals to dermatology specialists for diagnosis and treatment can halt progression and optimize outcomes. This article explores the causes and the clinical presentation of female hair loss conditions, outlining the distinction between those that are scarring and nonscarring. Female pattern hair loss (FPHL), the most common nonscarring condition, affects up to 50% of women over 50. For this reason, the authors provide details on pharmacologic and nonpharmacologic treatment modalities for FPHL, including topical and oral medications, natural remedies, platelet-rich plasma, and low-level laser therapy.

Keywords: antiandrogen, female pattern hair loss, low-level laser therapy, minoxidil, nonscarring alopecia, platelet-rich plasma, scarring alopecia

Hair loss manifests as a wide variety of disorders that can be challenging to diagnose and treat. All hair loss disorders have the potential to cause significant distress and affect quality of life. Hair represents health, beauty, and youth. It provides individuality, portrays femininity, and boosts self-confidence. Hair loss can negatively affect an individual's self-esteem, cause anxiety and depression, and impact interpersonal and professional relationships.[1] While women's health nurse practitioners (WHNPs) might not be providing treatment, they will see patients of all ages experiencing hair loss. Knowledge about potential causes and an initial evaluation can expedite a referral to a dermatologist for accurate diagnosis and treatment. Early treatment can make a difference in the trajectory of hair loss conditions.

It is relevant to be able to distinguish between scarring (cicatricial) hair loss and nonscarring loss (noncicatricial). Cicatricial alopecias lead to follicular scarring and permanent hair loss. These are pathologic conditions typically caused by inflammation that can wax and wane or progress rapidly and cause irreversible damage to the follicle. In contrast, nonscarring hair loss is potentially reversible.

This article focuses on the causes and clinical presentation of the most common causes of hair loss in women. Female pattern hair loss (FPHL) is one of the most common types of noncicatricial alopecias, estimated to affect up to 50% of women aged 50 and over.[2,3] Treatment options for FPHL are discussed. Knowledge about structure, function, and hair cycle regulation sets the foundation for evaluation of hair loss disorders.

Anatomy and Physiology of Hair

Hair follicles are classified into two types: terminal and vellus. Terminal hair is coarse, thick hair, mainly found on the scalp, eyebrows, eyelashes, armpits, and genital areas. Vellus hair is short, fine, and soft, and found on the remaining hair-bearing areas of the body. Terminal hair follicles extend deeper into the skin than vellus follicles.

There are approximately 100,000–150,000 terminal hair follicles on the human scalp. Important structures of these hair follicles include the bulb, inner and outer root sheath, sebaceous gland, and the bulge region. The bulb, the deepest portion of the follicle, resides in the dermal papilla and is essential for hair growth. The inner and outer root sheaths protect and support the hair shaft. Sebaceous glands produce sebum and provide lubrication, and the bulge region harbors stem cells that are essential for hair growth and follicular cycles (see Figure 1).[4]

Hair Growth Cycle

Hair follicles are in a constant cycle of growth (anagen), transition (catagen), and rest (telogen) phases. It is important to note that hair follicle cycles do not occur at the same time. Every follicle is independent of its

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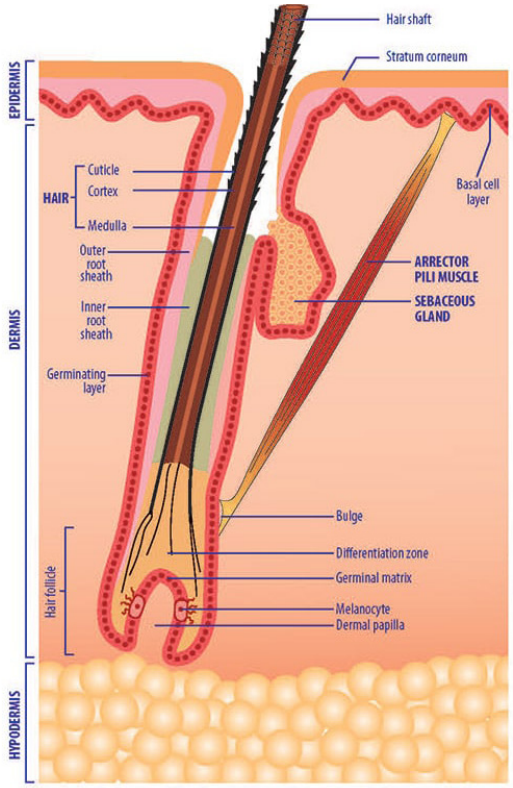

Figure 1. Hair follicle anatomy.

Source: Adapted from OpenStax College. (2013). *Illustration from Anatomy and Physiology*. http://cnx.org/content/col11496/1.6/

surrounding follicles, ensuring uniform hair density and preventing synchronized shedding.[5]

The anagen (growth) phase lasts 2–6 years. Approximately 90% of follicles are in this phase. Depending on the location, hair growth and cycle rates for terminal hairs range from 0.3 mm per day for 2–6 years (scalp) to 0.1 mm per day for 2–3 months (eyebrows). Shorter anagen phases account for shorter maximum length for both terminal and vellus hairs.

The catagen (transition) phase usually lasts around 3 weeks; hair production in this phase ceases, then transitions to the telogen (resting) phase. Less than 1%, or 500–1,000 scalp follicles, are in this phase at any given time.

Up to 10% of scalp follicles are in the telogen (resting) phase, which lasts for 2–3 months. Approximately 50–200 telogen hairs shed per day. This fact provides reassurance to anxious patients who present to the clinical setting with bags of hair. Once hair sheds, the follicle returns to the anagen phase and the cycle is repeated.

Causes of Female Hair Loss

Acute or chronic illness, nutritional deficiencies, medications, stress, and damaging hair care practices can cause hair loss. Causes may be multifactorial, and in some hair loss conditions, there may be a genetic component. When evaluating a patient with hair loss, it is crucial to focus on the precipitating factors, symptoms, and associated comorbidities. In other words, listen to your patients; they will tell you much of the information needed to determine if an immediate referral to a dermatology specialist is needed or if some initial treatments might be initiated in the primary care setting.

Cicatricial (Scarring) Alopecias

The most common cicatricial (scarring) alopecias affecting women are briefly discussed for the sake of clinical recognition and prompt treatment. These conditions often challenge clinicians due to their insidious onset and lack of distinct clinical findings. Cicatricial alopecias have a variety of causes that can be difficult to differentiate and diagnose in the primary care clinical setting. Most of the cicatricial alopecias have common symptoms including pain, burning, and pruritus. A biopsy is often required for diagnostic confirmation. Prompt referral to a dermatology specialist is important for an accurate diagnosis and initiation of appropriate treatment. Early diagnosis and treatment can stop the progression of disease and prevent further extensive hair loss. Regrowth in areas where follicles have already been replaced by scarring is not possible.

Central Centrifugal Cicatricial Alopecia

Central centrifugal cicatricial alopecia (CCCA) predominantly affects adult African American women, with a prevalence of 3%–6% of this demographic.[6] The exact pathogenesis is unknown, but likely multifactorial. Genetic mutations have been implicated. Traumatic hair care practices such as hot combs, traction-inducing styles, and chemical relaxers exacerbate the disease. Patients with CCCA will present with smooth patches without an abnormal follicular pattern expanding from the crown (upper back of head) and vertex (top of head) areas of the scalp. Skin inflammation is typically present. In darker skin types, this inflammation may be subtle and appear gray, violet, or deep red. As with most scarring alopecias, symptoms often include burning or pruritus in the areas of hair loss.

Lichen Planopilaris

Lichen planopilaris (LPP) is a condition in which there is chronic inflammation that leads to scarring alopecia, with a disease course that can have a brisk onset or take an insidious course, making diagnosis difficult. The etiology of lichen planus is not exactly known but is presumably due to a T-cell-mediated immunity dysfunction, which attacks affected tissue.

If untreated, LPP often leads to permanent destruction with scarring due to inflammatory cells infiltrating the follicle. The primary affected population is adults

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between the ages of 40 and 70 years with a female-to-male ratio of 4:1.[7] There are several variants of LPP, but all have similar clinical features, including perifollicular inflammation and scale, which is difficult to assess without magnification. The hair loss pattern is highly variable; most commonly, there are several localized areas of partial loss with scarring and associated inflammation.[8] Most importantly, the patient will often complain of increased hair shedding, burning, severe itch, and tenderness.

Discoid Lupus Erythematosus

Discoid lupus erythematosus most commonly affects African Americans between the ages of 20 and 40 years. Primarily affecting the scalp, face, and neck, the condition carries a 5–10% risk of developing systemic lupus erythematosus.[9] Early disease manifestations include inflamed papules and plaques with scale on sun-exposed areas. If the condition goes untreated, atrophic plaques devoid of hair will develop. The clinical presentation of skin inflammation on darker skin colors may be subtle. Look for violet, grayish, or pink patches and plaques. Symptoms often include tenderness, burning, and pruritus, all of which are worsened by sun exposure.

Traction Alopecia

Although not inflammatory in nature, this condition can result in permanent hair loss, which can be avoided with early intervention. Frequent tight braiding, cornrows, tight rollers, excessive heat, and excessive brushing weaken the hair shaft. Chemical relaxers weaken tight disulfide bonds, resulting in thinning and drying of the hair shaft. Traction alopecia can also occur because of work-related head gear such as headbands, hair caps, and helmets.[10] The temporal and frontal hair margins are often affected, and the condition is often asymptomatic. Look for broken hairs and receding hairline corresponding to areas of pulling and braiding.

Noncicatricial (Nonscarring) Alopecias

Inflammation is often mild or absent with noncicatricial alopecias, and hair follicle destruction does not occur in most cases. The distribution of hair loss helps narrow the differential diagnosis. If hair loss is focal, think alopecia areata or early traction alopecia. If diffuse, consider telogen effluvium. If patterned, androgenetic alopecia (FPHL) is often the culprit.

Alopecia Areata

Seen equally in both sexes, with onset often in childhood and young adult years, alopecia areata is an autoimmune disease of the hair follicle. The disease can affect the scalp, eyelashes, eyebrows, or body hair. Most cases present with one or two focal, smooth, round patches of hair loss, which often spontaneously regrow. The disease course is unpredictable, with variability in duration and extent of hair loss occurring from patient to patient.[11] A positive hair pull test can indicate active disease. A hair pull test is done by grasping a small clump of hair and gently tugging. If more than 10% of the hairs are pulled out, the test is considered positive and indicates disease activity.

Telogen Effluvium

Telogen effluvium is a common cause of diffuse hair shedding. It is a result of abnormal follicular cycling. As discussed earlier, approximately 90% of scalp hair is in the growth (anagen) phase and 10% is in the resting (telogen) phase. This condition prematurely moves scalp hair from the anagen phase to the telogen phase, with resultant premature shedding. A wide variety of triggers have been linked to the condition, which can be transient or chronic. Triggers include acute or chronic major illness, childbirth, medications, malnutrition, emotional stress, low vitamin D and/or ferritin levels, and endocrine disorders. Chronic telogen effluvium is idiopathic and rare, most often diagnosed in women between the ages of 30 and 60 years.[12] Shedding occurs between weeks and a few months of the inciting event. Patients will complain of a sudden increase in hair loss over the entire scalp and are usually otherwise asymptomatic. Shedding may continue for 6–12 months of loss exceeding 150 hairs per day. The diagnosis of acute telogen effluvium can often be made based on physical exam, including a hair pull test, and a thorough patient history. If history and exam fail to identify a trigger, lab tests can be beneficial. The following tests are typically ordered: complete blood count (anemia), complete metabolic panel (systemic causes), iron studies, vitamin D, and thyroid-stimulating hormone level. The condition is self-limited with treatment of any underlying cause.

Androgenetic Alopecia (FPHL)

Androgenetic alopecia or FPHL is a common nonscarring hair loss condition and a frequent complaint of women over the age of 40. While inheritable in nature, the specific genetic pattern of FPHL remains unclear. Current studies substantiate a multifactorial cause, implicating hormonal (androgen) and genetic factors.

Classic features of FPHL are progressive conversion of terminal hairs to vellus hairs (follicular miniaturization) and increased shortening in the duration of the growth (anagen) phase with successive hair cycles.[2] The progressive loss results in a visible reduction in hair density over the frontal and vertex areas of the scalp. It is postulated that genetics plays a large role in FPHL. In genetically susceptible patients, androgens have been shown to initiate and propagate FPHL.

In addition to genetics, the hormone dihydrotestosterone (DHT), along with an androgen-metabolizing enzyme, 5-alpha reductase, has been implicated in female

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and male pattern hair loss. Testosterone is converted to DHT by 5-alpha reductase. As DHT levels increase, so does hair loss. DHT appears to cause miniaturization of hair follicles. Decreased levels of DHT in both skin and blood have been associated with decrease and reversal of miniaturization.[5,13]

Making the Diagnosis

Clinical assessment of alopecia includes recognizing the precipitating factors, identifying comorbidities, and obtaining pertinent information necessary to guide treatment. A thorough history should uncover the information that is critical for diagnosis and treatment. Most nonscarring hair loss conditions can be diagnosed based on the history and exam findings (see Boxes 1 and 2). The severity of FPHL can be assessed by determining central hair loss width using the Sinclair Scale (see Figure 2).

Management

The primary goals of treatment are to limit further hair loss and promote terminal hair growth if feasible. Cicatricial (scarring) conditions should be managed by a dermatology specialist. Telogen effluvium is often self-resolving once a trigger is found and eliminated. If hair loss is limited, most cases of alopecia areata are often best left untreated because spontaneous regrowth often occurs within 1 year. There is no evidence that the course of mild disease is altered with treatment.[14]

Before initiating treatment of FPHL, rule out cicatricial alopecias, other noncicatricial alopecias, and hyperandrogenism. Inflammation, scale, or scarring should not be present. While managing FPHL, be aware that due to the psychosocial impact of this condition, the patient's perception of hair loss may not be consistent with the clinical assessment. One way to mitigate this is to take serial photos before and throughout therapy to document progress.

As discussed, since hair growth cycles are asynchronous, expect results to take some time. Interventions

Box 1. Focused History for Initial Assessment of Hair Loss

- Onset, duration, and pattern of hair loss
- Presence of associated symptoms: burning sensation, pain, and pruritus
- Characteristics of associated symptoms: localized or diffuse, intermittent, or continuous
- Current or recent illness, medications, hormonal changes, and stressors
- Any known potential triggers for hair loss

Box 2. Focused Physical Exam and Lab Tests for Initial Assessment of Hair Loss

- Color of scalp in hair loss area: gray, violet, or deep red (inflammatory scalp disorders)
- Signs of hyperandrogenism: severe patterned or early-onset hair loss, which may suggest a pathologic hyperandrogenism
- Hair loss due to reduction in hair density (FPHL) or generalized shedding (telogen effluvium)
- If there is increased hair shedding, determine if the hair breaks or comes out at the root
- Balding pattern
  - Patchy: alopecia areata, lichen planopilaris, trichotillomania (hair-pulling disorder)
  - Diffuse: telogen effluvium, diffuse alopecia areata, anagen effluvium (chemotherapy)
  - Patterned: FPHL, scarring alopecias
- Severity of FPHL using central part width (see Figure 2)
- Lab tests as indicated to rule out underlying disease: hormone levels (androgen, thyroid stimulating hormone), complete blood count, complete metabolic panel, serum iron and ferritin, autoimmune markers (ANA for lupus)
- In dermatology, two punch biopsies are performed for both vertical and horizontal pathology evaluation; typically scalp biopsies are reserved for inflammatory conditions

Abbreviations: ANA, Antinuclear Antibody test; FPHL, female pattern hair loss.

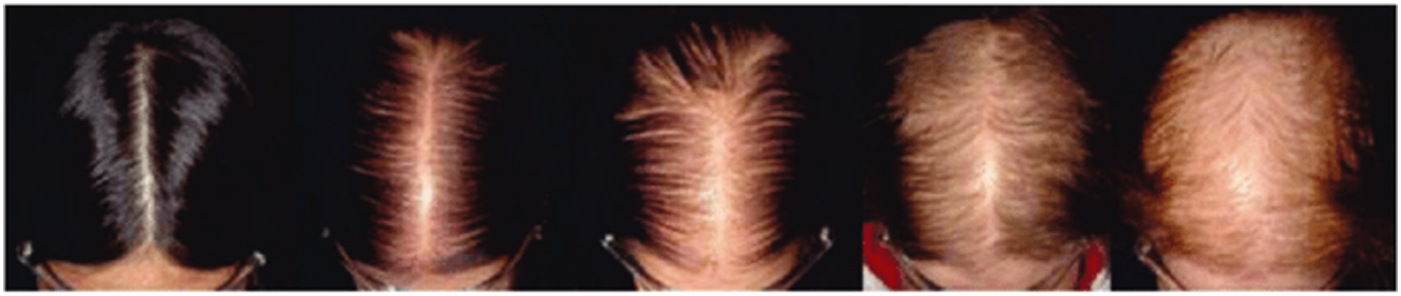

Figure 2. Sinclair scale.

Source: Adapted from Sinclair R, Torkamani N, Jones L. Androgenetic alopecia: new insights into the pathogenesis and mechanism of hair loss. *F1000Res*. 2015;4(F1000 Faculty Rev):585. doi:10.12688/f1000research.6401.1

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require at least 4–6 months before any clinical signs of improvement are achieved. Set realistic expectations with patients before initiation of therapy and advise them that pharmacologic therapy, if effective, must be continued indefinitely.

Topical Agents

Topical minoxidil is the only medication approved by the U.S. Food and Drug Administration (FDA) for FPHL; all other prescription medications discussed in this article are off-label. Oral antiandrogens include spironolactone, finasteride, and dutasteride.

Topical minoxidil (2% solution, 2% foam, 5% foam) should be incorporated in the treatment plan for all patients with FPHL. It has the strongest evidence for efficacy compared with other interventions and is well-tolerated.[15,16] Minoxidil thickens existing hair and affects growth cycles; it does not promote new growth. The exact mechanism of action is not completely understood. It is thought that the drug may prolong the growth (anagen) phase, promote enlargement of miniaturized follicles, and shorten the resting (telogen) phase. During the first 2–8 weeks of treatment, shedding commonly occurs. This is due to the release of telogen hairs, as follicles quickly transition from the resting (telogen) phase to the growth (anagen) phase and resolves with continued treatment.[16] Adverse reactions include irritation and mild scalp dryness. Topical minoxidil should be avoided during pregnancy. Data are not available on whether topical minoxidil is excreted in breastmilk.

A systematic review comparing 2% with 5% minoxidil found no significant difference in efficacy.[17] Minoxidil 5%

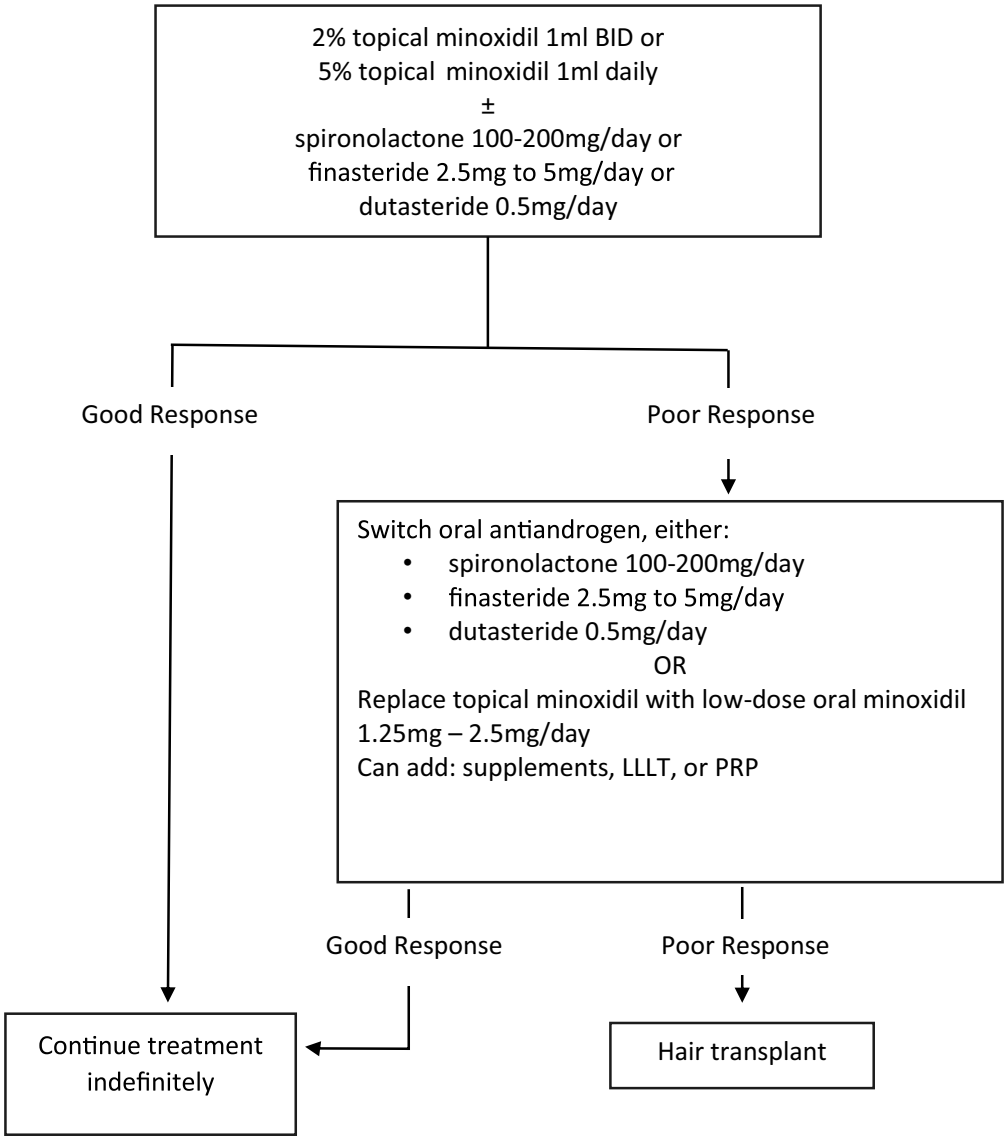

Figure 3. Female pattern hair loss treatment algorithm.

Abbreviations: BID, twice a day; LLLT, low-level laser light therapy; PRP, platelet-rich plasma.

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foam is associated with an increased risk of dandruff, scalp irritation, and sideburn hypertrichosis.[17] The 2% formulation dosage is 1 mL applied twice daily to affected areas; the 5% formulation is applied once daily to the scalp, not the hair. Continue treatment for 12 months before concluding inefficacy. Treatment with topical minoxidil alone is an alternate initial approach for those who prefer to avoid oral antiandrogenic medications. Consider replacing topical minoxidil with oral minoxidil if there is a poor response after 6–9 months. Alternatives to topical and oral medications include hair transplantation, laser therapy, and platelet-rich plasma (PRP; see Figure 3).

Oral Agents

Spironolactone is an aldosterone antagonist that inhibits androgen synthesis and blocks androgen receptors. It is typically dosed between 100 and 200 mg/day. Start at 50 mg/day in one or two divided doses. If tolerated after 2–4 weeks, increase to 100 mg/day in one to two divided doses. Titrate from 50 to 100 mg after 2–4 weeks to a maximum dose of 200 mg/day. Monotherapy with spironolactone is ineffective if dosed below 100 mg/day.[18] Adverse effects include decreased libido, headache, menstrual irregularities, hyperkalemia, fatigue, and orthostatic hypotension. Serum potassium levels should be checked periodically for patients over 45 years old; those with cardiac disease, renal disease, or multiple medical problems; and patients taking medications that increase the risk for hyperkalemia. Spironolactone is contraindicated during pregnancy. Limited data indicate this drug is poorly excreted in breastmilk and is acceptable to use during lactation.[19] There is no evidence linking breast or estrogen-dependent breast tumors to spironolactone.[20,21] The same holds true for the 5-alpha reductase inhibitors (finasteride and dutasteride).[22]

Finasteride is a 5-alpha reductase inhibitor that reduces the effects of DHT on hair follicles by inhibiting the testosterone-to-DHT conversion. For FPHL, daily doses of 2.5 and 5 mg appear to be effective.[23] Although well-tolerated in female patients, male patients have reported breast swelling and tenderness and decreased libido. Avoid finasteride in patients who are pregnant or considering pregnancy. Pregnant individuals should not handle broken or crushed tablets. Data on the excretion of finasteride in breastmilk are not available.[24]

Dutasteride is also a 5-alpha reductase inhibitor. It is dosed at 0.5 mg/day, which provides a greater reduction in scalp and serum DHT level as compared with finasteride 5 mg/day. More recent studies found dutasteride to be more effective than finasteride with comparable side effects.[25] It is contraindicated for use in women of childbearing age and should be reserved for postmenopausal women.[26] Side effects for dutasteride are similar to finasteride and include decreased libido and breast swelling and tenderness.

Oral low-dose minoxidil 0.25–1.25 mg has been shown to be an effective and safe option for FPHL.[27] Although not FDA-approved to treat FPHL, it is often used as an alternative treatment and its vasodilating action results in increased cutaneous blood flow and ensuing hair growth. It appears to be as effective as 5% topical minoxidil, but if taken orally, there is a slight increased risk of hypertrichosis (excessive hair growth) of the face and body.[28] Oral minoxidil carries a black box warning for cardiac conditions, including pericardial effusion. The use of low-dose minoxidil greatly reduces the cardiac risks associated with higher doses, with the exception of a slight increase in heart rate and lower leg edema.[15] That said, until further studies are conducted, the use of low-dose oral minoxidil should not be prescribed to elderly patients with an increased risk of heart failure, chronic renal failure, myocardial infarction, or severe hypertension. It is contraindicated in patients with previous hypersensitivity reactions or pheochromocytoma. Avoid the use of oral minoxidil during pregnancy and lactation.

Natural remedies, including vitamins, minerals, and nutraceuticals, are commonly used as alternative treatments. Nutraceuticals have been widely advertised for the treatment of hair loss. They often contain phytochemicals or naturally derived, biologically active compounds. The FDA regulates these products but does not approve them. They are not as rigorously tested as pharmaceuticals.[29] Caution should be used when selecting supplements for the treatment of hair loss. A recent systematic review has shown potential benefits for some of them and no evidence of benefits for others.[29] Biotin, part of the B complex group of vitamins, is a common supplement taken for hair loss. There is no evidence to support its efficacy. The efficacy of other supplements, including saw palmetto, caffeine, plant-based oils, and other vitamins and minerals, remains unclear.[29]

Procedures

PRP is a more recent therapy based on the concept that the concentrated platelets, cytokines, and growth factors in blood plasma may modulate signaling pathways that aid in regrowth and regeneration of hair. To prepare PRP, 10–30 mL of the patient's blood is centrifuged to separate blood into its various components. Once separated, 4–8 mL of the PRP is injected into the patient's scalp. Intact hair follicles are essential for this treatment; therefore, it is used more often for patients with early-stage FPHL. Although studies are limited, there are data that suggest improvement in hair density and caliber with PRP.[30] There is no standardization of PRP preparations and treatments are expensive, ranging from $500 to $2,500 per treatment.[15]

Low-level laser therapy (LLLT) is used to treat several types of alopecia. The proposed mechanism assumes photons emitted from LLLT may decrease inflammation and

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increase adenosine triphosphate production and cellular activity.[31] A near-infrared light range of 650–900 nanometers with a power output of 5 milliwatts is often used for this type of therapy. A 2021 systematic review of cap, comb, hairband, and helmet LLLT devices showed a significant increase in hair density in both men and women when compared with sham (placebo) controls. In fact, LLLT appears to be as efficacious as conventional FPHL treatments such as topical minoxidil. Limitations of the review include short-term follow-up with a maximum of 26 weeks and no head-to-head studies comparing efficacy between devices.[32] Laser caps, bands, and combs range in price from $200 to over $2,000.[15] Unlike ultraviolet light, which can cause skin cancer, research has shown that red light therapy does not increase the risk of any type of skin cancer. Darker skin types are more sensitive to red light. Caution should be used to prevent hyperpigmentation in this type of skin, which can be longer-lasting than similar hyperpigmentation caused by sunlight. Although more research is needed for the long-term effects of LLLT, in the short term, the side effect profile of LLLT is minor and includes skin irritation and temporary mild discomfort.[33]

Surgical Treatment

When all else fails, patients may opt for a hair transplant. Studies have shown that women can benefit from transplantation as much as men.[15] The cost of hair transplants ranges from $4,000 to over $20,000.[15] Advances in technique have significantly improved results and are a good option for patients who have failed other therapeutic options.

Implications for WHNP Practice

Hair loss in women is a complex and often distressing condition with many potential causes, from genetic predispositions and hormones to systemic illness and lifestyle influences. Early recognition, accurate diagnosis, and timely intervention are key in minimizing progression and optimizing outcomes. Distinguishing between scarring and nonscarring alopecias enables providers to make appropriate decisions about management and referral. WHNPs are often the first point of contact for women who have concern about hair loss. Even though WHNPs may not be treating each type of alopecia, their ability to conduct full initial assessments, detect red flags, and make timely referrals to dermatology specialists can have a significant impact on the quality of life for their patients.

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Author Correspondence

Susan L. Mayne, DNP, FNP-C, DCNP
Email: susan.mayne@uhhospitals.org
Loren E. Shannon, FNP-C
Email: loren.shannon@uhhospitals.org

References

1. Davis, DS, Callender VD. Review of quality of life studies in women with alopecia. *Int J Womens Dermatol*. 2018;4:18–22. doi:10.1016/j.ijwd.2017.11.007
2. Starace M, Orlando G, Alessandrini A, Piraccini BM. Female androgenetic alopecia: an update on diagnosis and management. *Am J Clin Dermatol*. 2020;21:69–84. doi:10.1007/s40257-019-00479-x
3. Norwood OT. Incidence of female androgenetic alopecia (female pattern alopecia). *Dermatol Surg*. 2001;27(1):53–54. PMID: 11231244.
4. Childs JM, Sperling LC. Histopathology of scarring and nonscarring hair loss. *Dermatol Clin*. 2013;31(1):43–56. doi:10.1016/j.det.2012.08.001
5. Myung P, Ito M. Dissecting the bulge in hair regeneration. *J Clin Invest*. 2012;122(2):448-454. doi: 10.1172/JCI57414
6. Ncoza DC, Salkey KS, Callendar VD, McMichael AJ. Central centrifugal ciciatricial alopecia: new insights and a call for action. *J Investig Dermatol Symp Proc*. 2017;18(2):S54–S56. doi:10.1016/j.jisp.2017.01.004
7. Mangold AR, Pittelkow MR. Lichen planus. In: Kang S, Amagai M, Bruckner AL, Enk AH, Margolis DJ, McMichael AJ, Orringer JS, eds. *Fitzpatrick's Dermatology*. 9th ed. McGraw-Hill Education; 2019.
8. Sperling LC, Sinclair RD, Shabrawi-Caelen L. Alopecias. In: Bolognia J, Schaffer J, Cerroni L, eds. *Dermatology*. 5th ed. Elsevier; 2024.
9. McDaniel B, Sukumaran S, Koritala T, et al. *Discoid Lupus Erythematosus*. StatPearls Publishing. Accessed January 2, 2025. https://www.ncbi.nlm.nih.gov/books/NBK493145/
10. Larrondo J, McMichael AJ. Traction alopecia. *JAMA Dermatol*. 2023;159(6):676. doi:10.1001/jamadermatol.2022.6298.21075478
11. Sibbald C. Alopecia areata: an updated review for 2023. *J Cutan Med Surg*. 2023;27(3):241–259. doi: 10.1177/12034754231168839
12. Rebora A, Guarrera M, Baldari M, Vecchio F. Distinguishing androgenetic alopecia from chronic telogen effluvium when associated in the same patient: a simple noninvasive method. *Arch Dermatol*. 2005;141(10):1243–1245. doi:10.1001/archderm.141.10.1243
13. Nestor MS, Ablon G, Gade A, Han H, Fischer DL. Treatment options for androgenetic alopecia: efficacy, side effects,

https://doi.org/10.1891/CJNPWH-2025-0010

compliance, financial considerations, and ethics. *J Cosmet Dermatol*. 2021;20(12):3759–3781. doi:10.1111/jocd.14537

14. Malhotra K, Madke B. An updated review on current treatment of alopecia areata and newer therapeutic options. *Int J Trichology*. 2023;15(1):3–12. doi:10.4103/ijt.ijt_28_21
15. Devjani S, Ezemma O, Kelley KJ, Stratton E, Senna M. Androgenetic alopecia: therapy update. *Drugs*. 2023;83(8):701–715. doi:10.1007/s40265-023-01880-x
16. van Zuuren EJ, Fedorowicz Z, Schoones J. Interventions for female pattern hair loss. *Cochrane Database Syst Rev*. 2016;2016(5):CD007628. doi:10.1002/14651858.CD007628.pub4
17. Blume-Peytavi U, Hillmann K, Dietz E, Canfield D, Garcia Bartels N. A randomized, single-blind trial of 5% minoxidil foam once daily versus 2% minoxidil solution twice daily in the treatment of androgenetic alopecia in women. *J Am Acad Dermatol*. 2011;65(6):1126–1134.e2. doi:10.1016/j.jaad.2010.09.724
18. Aleissa M. The efficacy and safety of oral spironolactone in the treatment of female pattern hair loss: a systematic review and meta-analysis. *Cureus.* 2023;15(8):e43559. doi:10.7759/cureus.43559
19. Aldactone (spironolactone) tablets. Package insert. G.D. Searle LLC; 2001.
20. Wei C, Bovonratwet P, Gu A, Moawad G, Silverberg JI, Friedman AJ. Spironolactone use does not increase the risk of female breast cancer recurrence: a retrospective analysis. *J Am Acad Dermatol*. 2020;83(4):1021–1027. doi:10.1016/j.jaad.2020.05.081
21. Bommareddy K, Hamade H, Lopez-Olivo MA, Wehner M, Tosh T, Barbieri JS. Association of spironolactone use with risk of cancer: a systematic review and meta-analysis. *JAMA Dermatol*. 2022;158(3):275–282. doi:10.1001/jamadermatol.2021.5866
22. Seyed Jafari SM, Heidemeyer K, Hunger RE, de Viragh PA. Safety of antiandrogens for the treatment of female androgenetic alopecia with respect to gynecologic malignancies. *J Clin Med*. 2024;13(11):3052. doi:10.3390/jcm13113052
23. Iamsumang W, Leerunyakul K, Suchonwanit P. Finasteride and its potential for the treatment of female pattern hair loss: evidence to date. *Drug Des Devel Ther*. 2020;14:951–959. doi:10.2147/DDDT.S240615
24. Finasteride tablets. Package insert. Camber Inc; 2025.
25. Arif T, Dorjay K, Adil M, Sami M. Dutasteride in androgenetic alopecia: an update. *Curr Clin Pharmacol*. 2017;12(1):31–35. doi:10.2174/1574884712666170310111125
26. Dutasteride tablets. Package insert. GlaxoSmithKline; 2011.
27. Ramos PM, Sinclair RD, Kasprzak M, Miot HA. Minoxidil 1 mg oral versus minoxidil 5% topical solution for the treatment of female-pattern hair loss: a randomized clinical trial. *J Am Acad Dermatol*. 2020;82(1):252–253. doi:10.1016/j.jaad.2019.08.060
28. Randolph M, Tosti A. Oral minoxidil treatment for hair loss: a review of efficacy and safety. *J Am Acad Dermatol*. 2021;84(3):737–746. doi:10.1016/j.jaad.2020.06.1009
29. Drake L, Reyes-Hadsall S, Martinez J, Heinrich C, Huang K, Mostaghimi A. Evaluation of the safety and effectiveness of nutritional supplements for treating hair loss: a systematic review. *JAMA Dermatol*. 2023;159(1):79–86. doi:10.1001/jamadermatol.2022.4867
30. Gupta AK, Cole J, Deutsch DP, et al. Platelet-rich plasma as a treatment for androgenetic alopecia. *Dermatol Surg*. 2019;45(10):1262–1273. doi:10.1097/DSS.0000000000001894
31. Darwin E, Heyes A, Hirt PA, Wikramanayake TC, Jimenez JJ. Low-level laser therapy for the treatment of androgenic alopecia: a review. *Lasers Med Sci*. 2018;33(2):425–434. doi:10.1007/s10103-017-2385-5
32. Jimenez JJ, Wikramanayake TC, Bergfeld W, et al. Efficacy and safety of a low-level laser device in the treatment of male and female pattern hair loss: a multicenter, randomized, sham device-controlled, double-blind study. *Am J Clin Dermatol*. 2014;15(2):115–127. doi:10.1007/s40257-013-0060-6
33. Hernández-Bule ML, Naharro-Rodríguez J, Bacci S, Fernández-Guarino M. Unlocking the Power of Light on the Skin: A Comprehensive Review on Photobiomodulation. *Int J Mol Sci*. 2024;25(8):4483. doi:10.3390/ijms25084483

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