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Diagnosis and Treatment of Nonscarring Hair Loss in Primary Care in 2021

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The first step in diagnosing hair loss (alopecia) is to determine whether the process is nonscarring or scarring. This review focuses on nonscarring alopecia, the category of hair loss most frequently encountered in primary care. Scarring alopecia, characterized by permanent destruction of follicular structures, presents with pruritus, pain, erythema, scale, and/or crust and obliteration of follicular pore markings leading to an abnormally smooth appearance of the skin. These patients may require scalp biopsy for diagnosis and referral for specialty care. Once hair loss has been categorized as nonscarring, the next step is to determine the distribution of loss on the scalp as patterned, diffuse, or focal (**Figure**). Diagnosis and management of the most common nonscarring disorders in each of these categories will be discussed.

Patterned

In patterned hair loss, thinning occurs symmetrically and most notably at the front, top, and sides of the scalp (frontal, vertex, temporal, parietal), while greater density is retained at the back of the scalp (occipital). Although thinning of the frontotemporal hairline occurs in both sexes, pronounced recession is more typical of men. In patterned hair loss, follicles miniaturize (ie, become finer and grow less long).

Example Diagnosis: Androgenetic Alopecia

Androgenetic alopecia (AGA) is the most common form of patterned hair loss. AGA begins at puberty, with follicular miniaturization mediated by the androgen dihydrotestosterone. Patients describe gradual hairline recontouring and decreased volume and length (eg, "my scalp is more visible on top," "my hair no longer grows as long").

AGA is a heritable polygenic trait. Female patients with irregular menses, acne, hirsutism, and acanthosis nigricans should be screened for polycystic ovary syndrome.[1] Managing polycystic ovary syndrome may improve patterned hair loss.

A meta-analysis of randomized placebo-controlled trials showed that US Food and Drug Administration (FDA)–approved medications in men; minoxidil 2% solution in women; and low-level light devices, compared with placebo, were associated with greater improvement in hair density per cm^2 from baseline ($P < .00001$).[2] FDA-approved therapies for men include minoxidil 2% solution (mean hair density change of +8.11 hairs/cm^2), 5% solution (+14.94 hairs/cm^2), or 5% foam twice daily and the 5_α-reductase type II inhibitor finasteride, 1 mg, once daily (+18.37 hairs/cm^2).[2] Women may use minoxidil 2% solution twice daily (+12.41 hairs/cm^2) or 5% foam once daily.[2] Low-level light devices (+17.66 hairs/cm^2) emitting low-frequency red light have FDA clearance for safety.[2]

Up to 15% of patients who use topical minoxidil experience transient hair shedding in the first few months of treatment; 2% to 5% of women develop unwanted facial hair that resolves with treatment discontinuation. Although adverse effects with oral finasteride are uncommon and clear causality between 5_α-reductase inhibitors and sexual adverse effects have not been established, the FDA has revised labeling to include reports of disorders involving libido, ejaculation, and orgasm, which, in some cases, continued after drug discontinuation; 5_α-reductase inhibitors may reduce serum prostate-specific antigen levels.[3]

Diffuse

Diffuse hair loss occurs uniformly across the entire scalp.

Example Diagnosis: Telogen Effluvium

Telogen effluvium (TE) is the most common cause of diffuse scalp hair loss. An inciting event (severe acute or chronic illness; major surgery; thyroid disease; pregnancy; iron deficiency anemia; malnutrition; rapid weight loss; vitamin D Deficiency; certain medications, most commonly lithium, sodium valproate, fluoxetine, warfarin, metoprolol, propranolol, retinoids, and isoniazid; and the discontinuation of estrogen-containing oral contraceptive pills) disrupts the hair cycle, leading to a loss of greater than 200 scalp hairs a day.[4,5] Although decreased hair density is apparent to the patient, it may not be to the clinician. Results of a pull test, performed by grasping the base of 40 to 50 hairs and pulling away from the scalp, will be positive (ie, dislodges >4 telogen hairs, identifiable by their club-shaped bulbs) in patients with TE.[5]

In TE, hair shedding occurs 2 to 4 months after the inciting event. Laboratory evaluation for causes of hair cycle dysfunction consists of thyroid-stimulating hormone, ferritin, and 25-hydroxyvitamin D_2 and D_3. Medical, surgical, and medication history should be reviewed.[6] Although TE is typically self-limited, correction of ongoing triggers must be pursued. Shedding takes 6 to 9 months to normalize. Patients with concomitant patterned hair loss may benefit from topical minoxidil to improve the density of regrowth.

Focal

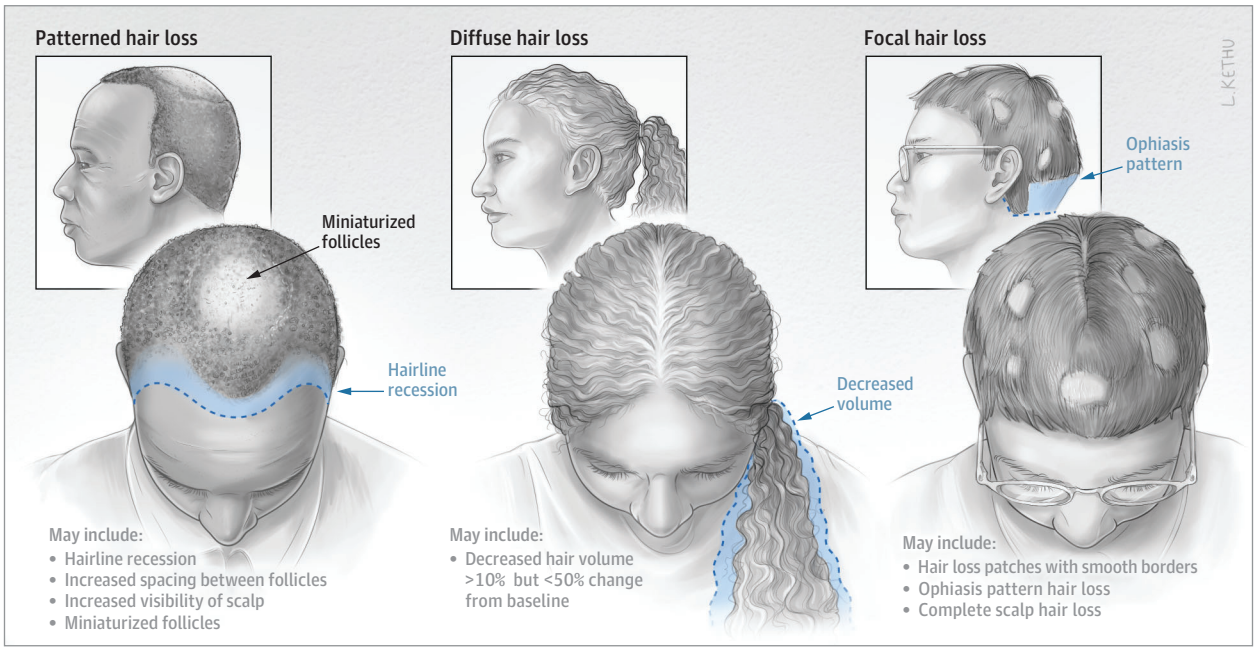
Focal hair loss occurs in discrete patches on the scalp and sometimes on the face and body.

Example Diagnosis: Alopecia Areata

The most common type of focal hair loss is alopecia areata (AA), an autoimmune disorder that often presents early in life but can affect individuals of any age, sex, or race. The lifetime incidence risk of AA is estimated at 2.1%.[7] Many patients with AA have atopic dermatitis, asthma, or allergic rhinitis. Patients with AA and family members of those with AA may have other autoimmune disorders, in particular, vitiligo, Hashimoto thyroiditis, and type 1 diabetes. In AA, a pathologic imbalance favoring cytotoxic $CD8^+NKG2D^+$ T cells over regulatory T cells leads to the targeting of growing anagen hairs and subsequent hair cycle dysfunction.[8]

Manifestations include bare, round patches (patchy AA), complete scalp hair loss (alopecia totalis), and complete scalp and body hair loss (alopecia universalis). Band-like, or *ophiasis*, loss along the occipital hairline can be difficult to manage. Although AA is unpredictable, spontaneous regrowth may be seen in up to 30% of patients with milder forms.[9] In active areas, the scalp may be pink and "exclamation point" hairs (hair with constricted proximal ends) may be observed. Pigmented hairs are preferentially lost, and regrowth

Figure. Prototypical Morphologies of Nonscarring Hair Loss



Under physiologic conditions, the neurovascular, hormonal, and immune systems work in concert to maximize hair shaft diameter and length as well as support optimal hair cycling; 90% of scalp hairs are in the anagen (growth) phase at any given time, while 10% are in the telogen (resting) or catagen (involution) phases.

is often initially nonpigmented or white. Shallow regularly spaced nail pits are a hallmark.

Treatment recommendations depend on patient age and extent of hair loss. Because spontaneous regrowth is common, young children or those with limited patches may not require treatment. For mild patchy AA, intralesional triamcinolone acetonide is effective. Other therapies include topical corticosteroids, topical minoxidil, contact irritants (anthralin), and contact allergens (squaric acid dibutyl ester, diphenylcyclopropenone). Oral janus kinase inhibitors, such as tofacitinib, ruxolitinib and baricitinib, have recently shown benefit for severe disease.[10]

Conclusions

An accurate diagnosis of nonscarring hair loss can be made by identifying the distribution of hair loss, reviewing the medical history, and performing appropriate laboratory workup. Treatment depends on the etiology of follicular disruption and includes medications that optimize follicular density and hair follicle cycling.

ARTICLE INFORMATION

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