An Approach to Hair Loss in Women

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Hair loss is a common symptom presenting in women, and there are a range of conditions that can cause hair loss in women. Conditions for hair loss can be scarring (cicatricial) or nonscarring (noncicatricial). Nonscarring conditions include female pattern hair loss, telogen effluvium, and alopecia areata. Trichotillomania and tinea capitis are also causes of nonscarring alopecia. Scarring alopecias comprise primary and secondary conditions. Primary scarring alopecias include lichen planopilaris and discoid lupus erythematosus. Secondary causes of scarring alopecia include tumors. Hair shaft disorders, or trichodystrophies, and traction alopecia are other forms of hair loss in women.

Hair loss is stressful and cosmetically concerning for women. In some cases, the degree of concern may seem inconsistent to the severity of the hair loss.[1] Given the ready availability of information on the Internet and advice from friends and family, patients often have misconceptions of the causes of hair loss and may have tried several expensive over-the-counter remedies.[1] A comprehensive history and physical examination to identify the cause of hair loss and any underlying systemic diseases should be performed. Spending time with patients, acknowledging their complaints, and providing information will give patients a better understanding of the problem and more satisfaction that it is being taken seriously.[1]

HAIR GROWTH

Approximately 100,000 hair follicles exist on the scalp, and humans do not form more hair follicles after birth.[2,3] Hair growth is cyclical and with each new hair cycle, the hair follicle remodels itself.[3] Each hair follicle undergoes 10 to 30 cycles in a lifetime.[3,4] Hair stem cells form the new hair follicle and are found in the permanent bulge region of the follicle.[4] Damage to the stem cells can result in a cicatricial (scarring) alopecia and the hair cannot regrow.[3] Hair follicle growth is asynchronous. Each hair follicle goes through a cycle: anagen, a growth phase; catagen, a regression phase; telogen, a resting phase; and exogen, a shedding phase.[3,4] Eighty percent to 85% of hair follicles on the scalp are in anagen, which lasts 2 to 8 years, and the remainder of the hair follicles are in catagen and telogen.[3,5] Telogen lasts 3 to 5 months and 50 to 150 telogen hairs are lost per day.[5] Different factors (eg, hormones such as androgens) can influence the hair follicle characteristics and alter the normal hair cycle.[3]

CAUSES OF NONSCARRING HAIR LOSS

Female Pattern Hair Loss

Pattern hair loss (PHL), or androgenetic alopecia, begins after the onset of puberty.[6] It is thought to occur in genetically predisposed individuals, but the role of androgens in the pathogenesis of female PHL (FPHL) is unclear.[6-8] Women with normal circulating androgen levels who show no other clinical signs of hyperandrogenism, such as hirsutism, irregular menstrual periods, or acne, can still present with FPHL.[6-8] However, FPHL is seen in women with hyperandrogenism, and some women with hyperandrogenism can present with a male type of PHL.[6,9] Some women present with complaints of a decrease in hair

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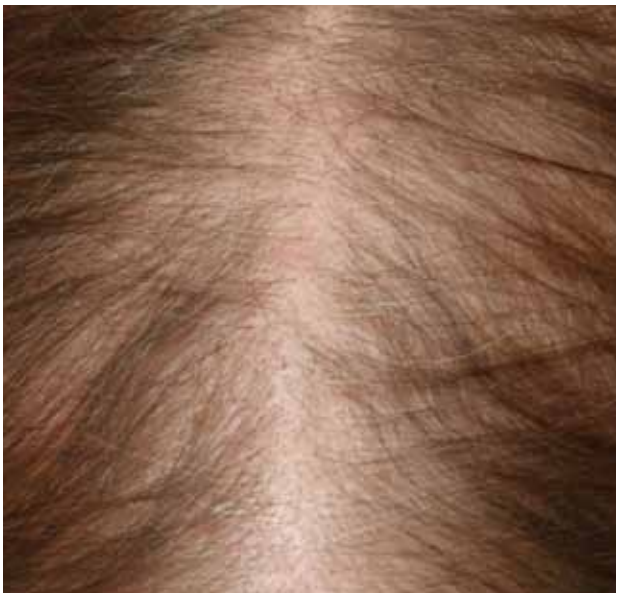

Figure 1. Patient with female pattern hair loss, with loss of scalp hair, diffuse thinning over the crown/vertex scalp, and a widening of the central midline part.

density over the crown or with episodic shedding and no apparent loss in density.[10] They can give a positive family history for androgenetic alopecia.[6] On clinical examination, FPHL typically presents with a characteristic pattern of scalp hair loss, with diffuse thinning over the crown/vertex scalp, and with a widening of the central midline part (Figure 1).[11,12] Preservation of the frontal hair line is most common[6]; however, some women exhibit a focus of hair loss in the frontal region described as a "Christmas tree" pattern of hair loss.[6,7] Complete baldness is not typically seen in FPHL.[6]

Miniaturization of large terminal scalp hairs into small vellus hairs is characteristic of FPHL and can be shown by scalp biopsy.[6,13-15] Laboratory testing can exclude other causes of hair loss, and in women with signs of hyperandrogenism, an androgen screen is essential.[6]

Telogen Effluvium

Telogen effluvium is another common cause of hair loss in women and arises due to an interruption in the normal hair cycle.[5,16] Women experience excessive hair shedding of up to 300 telogen hairs per day, which is above normal.[9,17] Clinical examination usually reveals a diffuse hair thinning over the entire scalp (Figure 2). Telogen hairs can be obtained with a hair pull test. Telogen effluvium can be categorized into acute (<6 mo) and chronic (>6 mo) hair shedding (Figure 3).[9,18] Acute telogen effluvium is defined as an acute onset telogen hair loss 2 to 3 months after a sudden triggering event.[5,16] A detailed history is important to determine an accurate timeline of the relationship between possible triggers and hair loss. Multiple conditions can trigger telogen hair shedding. A real trigger of telogen hair loss is reversible, with cessation of the hair shedding following removal of the stress, and return of the shedding with recurrence of the trigger or stress.[19] Accepted triggers for acute telogen effluvium are surgery, high fever, severe illness, postchildbirth (telogen gravidarum), and dieting.[5,20] If the trigger is identified and removed, the shedding tends to resolve by 6 months and recovery can be expected to be complete.[17]

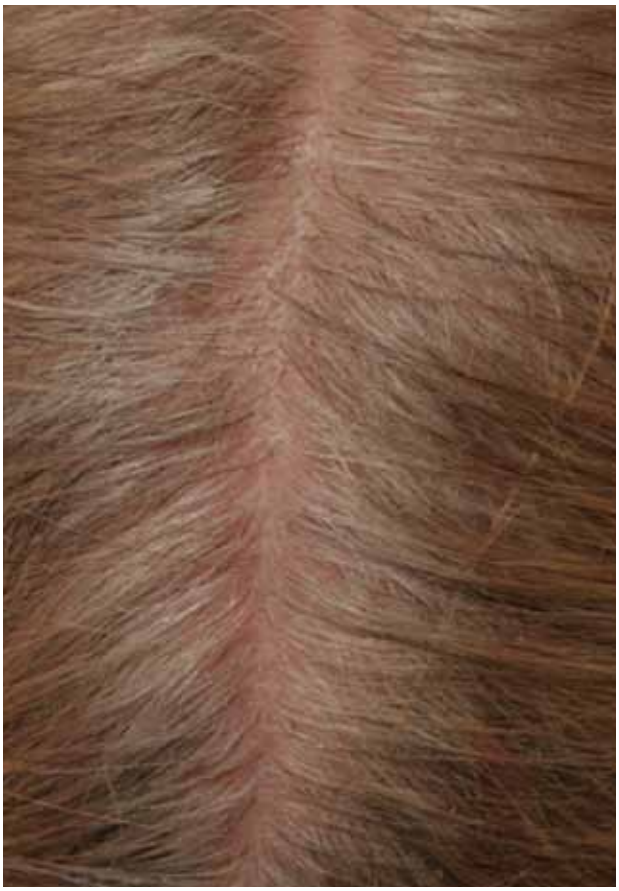

Figure 2. Patient with acute telogen effluvium, with diffuse hair thinning over the entire scalp.

Chronic telogen effluvium (CTE) refers to the idiopathic primary condition, first described by Whiting in 1996, of chronic telogen hair shedding of more than 6 months duration with no identifiable triggers.[21,22] With these patients, there is usually no family history of androgenetic alopecia and the patients tend to report thick hair at baseline.[21,22] On examination, there is no widening of the midline part as in FPHL, and these patients often present with a full head of hair.[21,22] Bitemporal recession can be seen in some cases.[22] It is a diagnosis of exclusion of other causes of chronic diffuse telogen hair loss and FPHL.[21,22] The natural history of this condition is

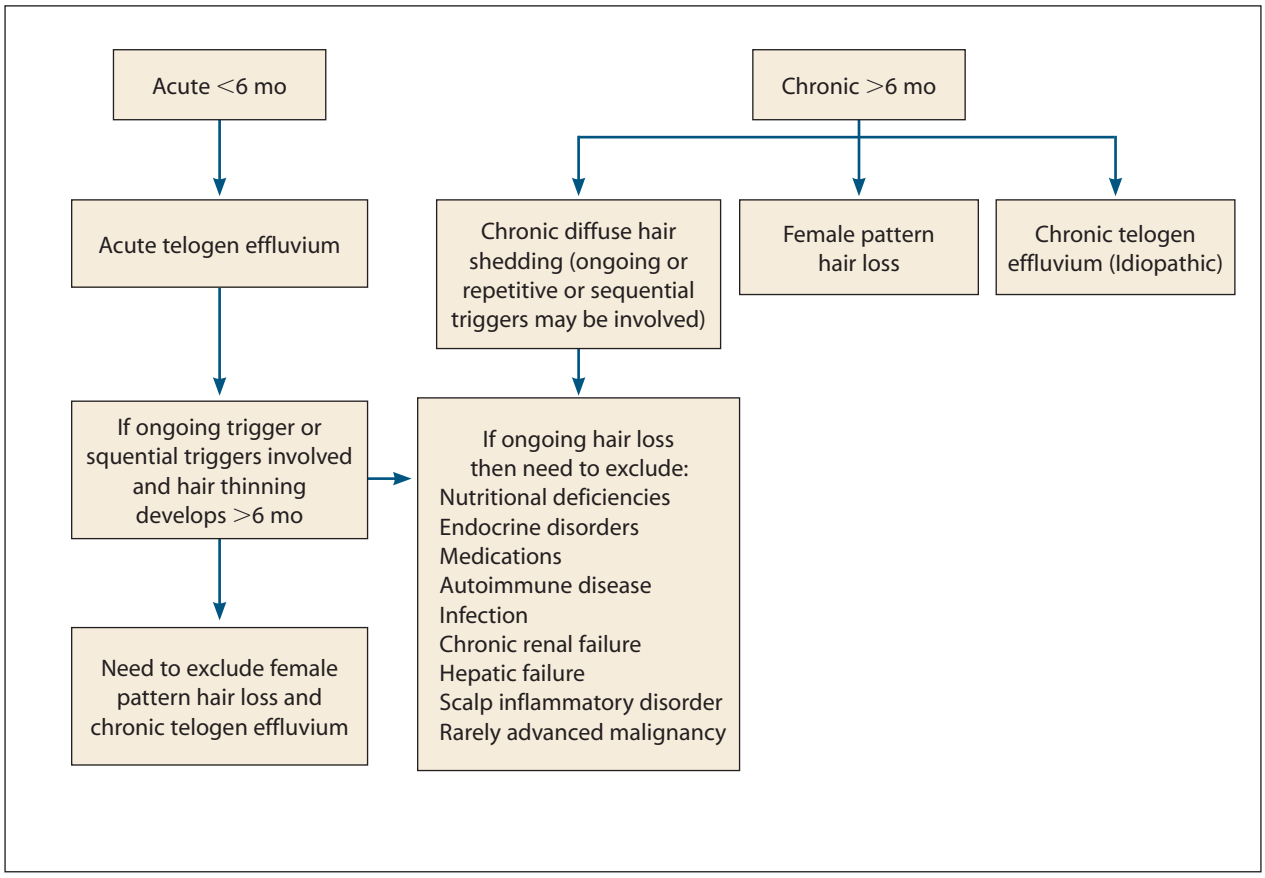

Figure 3. Telogen hair shedding.

unknown, but telogen hair shedding can fluctuate during many years.[21,22] In some cases, patients may develop both conditions, and in other cases, CTE may progress to FPHL.[9,21,22] Scalp biopsy can distinguish between FPHL and CTE, with no follicle miniaturization being seen in CTE.[21,22]

In contrast to idiopathic CTE, chronic diffuse telogen hair loss of more than 6 months' duration can be secondary to a variety of triggers.[17,20] Difficulties can arise as patients will often have multiple triggers that may occur concomitantly, sequentially, or repetitively (Figure 3).[17] Multiple repetitive or sequential triggers will cause ongoing chronic telogen hair loss and can be mistaken for idiopathic CTE or early FPHL.[17] Laboratory investigations can assist in confirmation of suspected triggers of hair loss. Some triggers include hypothyroidism, hyperthyroidism,[20,23] and nutritional disorders such as zinc deficiency or iron deficiency.[17,20] Rarer nutritional causes of telogen hair loss are essential fatty acid deficiency, biotin deficiency, and severe protein and caloric restriction with chronic starvation.[17,20,24] Vitamin A deficiency can also cause telogen hair loss.[17] Since vitamin D is an important vitamin in cell proliferation, vitamin D deficiency may also play a role in hair loss.[17] Diffuse telogen hair loss has also been reported in chronic systemic disorders, such as hepatic insufficiency,[5,20] chronic renal failure,[5] inflammatory bowel disease,[5,24] lymphoproliferative disorders,[20] secondary syphilis,[5,20] and autoimmune diseases such as systemic lupus erythematosus.[24] Emotional stress and local scalp inflammatory disorders, such as psoriasis and seborrheic dermatitis, have been reported to trigger a diffuse telogen hair loss.[17,24]

All drugs ingested by a patient presenting with hair loss should be suspected as a cause of hair loss, including botanicals.[17,24] Drug-induced telogen hair loss starts 6 to 12 weeks after commencing the drug and continues while on the medication.[25] Dosage changes can also trigger a telogen hair shed.[17] Many drugs, such as cardiac medications, anticonvulsants, oral contraceptives, and antidepressants, can cause telogen effluvium.[24,25]

Alopecia Areata

Alopecia areata (AA) is an autoimmune, nonscarring form of hair loss that is unpredictable and recurring.[9,26,27] Genetic, immunological, and environmental factors are likely involved.[26,27] It commonly occurs

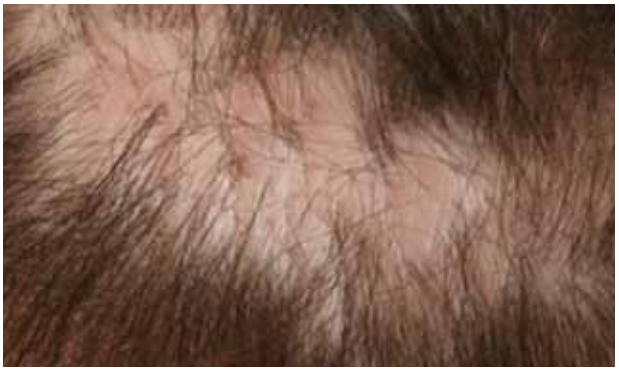

Figure 4. Patient with cicatricial alopecia, with loss of visible follicular openings on the scalp.

before the age of 20 but can occur at any age.[28] Some patients have a positive family history for AA.[26] Many autoimmune associations also occur with AA including autoimmune thyroid disease, pernicious anemia, vitiligo, and diabetes mellitus.[26,27]

Typically, AA presents as nonscarring, single or multiple coin-shaped areas of hair loss, complete hair loss of the scalp (alopecia totalis), or loss of all hair on the scalp and body (alopecia universalis).[27] Occasionally, pain, burning, or pruritus can be experienced prior to the onset of a patch.[27] Diffuse AA is characterized by a severe, sudden onset of diffuse scalp hair shedding and thinning over the whole scalp.[27] These patients may also experience acute graying of the hair.[1] Diffuse AA can be mistaken for the shedding seen in early FPHL and telogen effluvium.[1,9]

Characteristically, the area of alopecia shows early preservation of follicular openings[26] and exclamation mark hairs (broken off short hair that tapers nearer to the scalp) at the periphery of the active area.[26,27] A hair pull is positive from the margin of the active patch.[26] Nail dystrophy, such as nail pitting, longitudinal ridging, Beau lines, and trachyonychia, can be associated with AA.[26,27]

If the diagnosis of AA is unclear, then scalp biopsy should confirm the diagnosis with histology early in the disease showing peribulbar inflammatory infiltrate surrounding anagen hair follicles and an increased number of catagen and telogen follicles.[22] Laboratory tests should examine for related autoimmune diseases.

Traction Alopecia

Traction alopecia is common in women, with certain hair styles more likely to cause hair loss.[29] Tight braids, ponytails, or chignons direct a traction force on the hair and can cause hair loss where the force is maximal.[30] Chignons can cause hair loss over the occiput, whereas ponytails or braids can cause hair loss at the scalp margins.[31,32] An inflammatory folliculitis can be seen in some patients or the inflammation can be subclinical.[33] An area of patchy alopecia with broken hairs is usually found.[30,33] Sustained traction can lead to permanent hair loss.[30,33] Diagnosis is usually made from patient evaluation.

TRICHOTILLOMANIA

Trichotillomania is characterized by the *Diagnostic and Statistical Manual of Mental Disorders* (Fourth Edition) as compulsively pulling or tugging the hair.[34] Linear or irregular patches of partial hair loss occur.[35,30] Scalp biopsy may be needed to confirm the diagnosis, and hair microscopy can show fractured hair shafts.[30]

Tinea Capitis

The fungal infection of the hair, tinea capitis, presents with pruritic patches of incomplete scalp hair loss. Hair microscopy and fungal culture reveal the diagnosis.[36]

CAUSES OF SCARRING HAIR LOSS

Cicatricial (scarring) alopecias are a group of disorders that cause permanent hair loss and are characterized by patches of hair loss, which are usually inflammatory, with loss of visible follicular openings on the scalp (Figure 4).[37,38] Inflammatory changes, such as erythema, pustules, scaling, and follicular plugging, can be seen.[39] Patients can experience scalp scaling, itching, and burning associated with hair loss. These can be divided into primary disorders, such as lichen planopilaris and discoid lupus erythematosus; and secondary disorders due to infection, malignancy, and trauma; or due to bullous disorders, such as mucous membrane pemphigoid and epidermolysis bullosa.[37,38] Scalp histology can provide the diagnosis, with all scarring alopecias having in common loss of the hair follicle or pilosebaceous unit.[37,38] Treatment is guided by the type of scarring alopecia present and aims to slow the rate of progression and suppress inflammation.[39]

TRICHODYSTROPHIES AND HAIR SHAFT DISORDERS

Another complaint from women regarding hair loss involves hair that does not grow long and hair that easily breaks. Hair shaft disorders, or trichodystrophies, are commonly seen in women who excessively style and groom their hair.[40,41] Certain hair care practices, such as chemical straightening, perming, and coloring, can damage the hair shafts and cause weathering. Other grooming styles that involve hot curling irons or metal straighteners can also injure the hair shafts, causing fragility. Environmental factors such as UV light and water also contribute to hair fragility. Some trichodystrophies are inherited, uncommon disorders of hair loss that will not be discussed further. Light microscopy can assist in the diagnosis of trichodystrophies. Trichorrhexis nodosa, a nodelike defect, is a feature seen in weathered hair.[40]

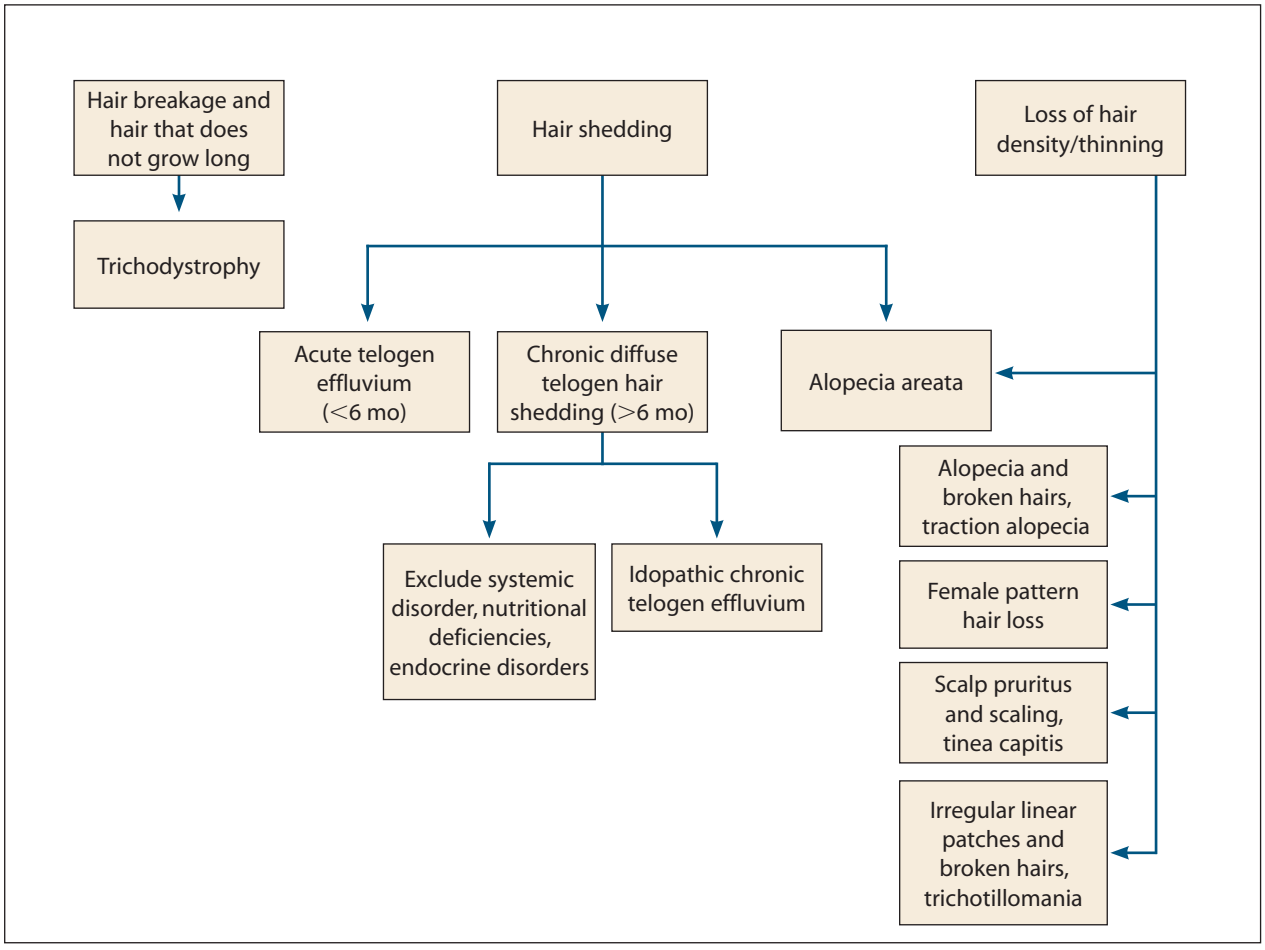

Figure 5. Types of hair loss.

CLINICAL HISTORY

A thorough history is crucial in diagnosing women with hair loss. Recognition of the type of hair loss, whether the hair is shedding, whether the hair density is decreasing, or whether the hair is breaking and not growing long is very helpful (Figure 5).[9] The onset and chronicity of hair loss are important, and an approximation of the amount of hair lost should be made by the patient. It is also important to know whether the hair loss has been continuous or episodic, or if the patient has had episodes of alopecia in the past. Episodic hair loss could suggest telogen effluvium or AA. With telogen effluvium, shedding hair is usually easily recognizable and may be found in a shower drain, on a pillow, or in a hairbrush.[17] Loss of hair density is usually observed by the patient when there is a 30% to 50% loss of hair density.[42,43] Hair fragility and breakage tend to be noted by the patient particularly after styling and if the hair does not grow long (Figure 5). Hair breakage is noted in trichodystrophies, traction alopecia, trichotillomania, and tinea capitis.[9] The distribution and pattern of hair loss are important. Loss of body hair or eyelashes and eyebrows suggest AA or trichotillomania, which can also affect any hair-bearing body site.[9] Symptoms of scalp pruritus, scaling, or burning should also be recorded. Scalp itch may be a symptom of an infection like tinea capitis, seborrheic dermatitis, or a scarring alopecia. Any previous investigations and treatments tried for hair loss by the patient should be noted.

A female history is important and should include menstrual history, history of oral contraceptive use, type of contraceptive presently used, pregnancy history, history of infertility, age at menopause, and hormone replacement.[2] Menorrhagia may suggest a possible underlying iron deficiency or endocrine disorder such as hyperthyroidism. Features of hyperandrogenism, such as irregular periods, acne, seborrhea, and hirsutism, should also be noted and may suggest FPHL or an androgen excess disorder as the cause of alopecia.[9] Some hair loss disorders have a hereditary component; therefore, a family history of PHL, AA, polycystic ovarian syndrome, thyroid disease, autoimmune conditions, and estrogen-dependent cancers should be obtained. Events in the recent past, such as weight loss, new medication, childbirth, recent illness, or surgery, may suggest a telogen effluvium as the

cause of the hair loss. A thorough dietary history should be performed, with special emphasis on vegetarian diets and crash dieting, and may identify a low-protein or low-iron status contributing to the hair loss.[9] A history of hair care involving styling and cosmetic practices is required and can identify possible causes for hair breakage and loss.[9]

Past medical history and systems review will identify any history of autoimmune disease, malignancies, or nail or skin disorders, and suggest an underlying systemic disease such as thyroid disease as a cause of the hair loss. A history of changes in dosage of medication or new medications is also important.[9] Any over-the-counter and herbal preparations should also be noted and are a possible cause of hair loss. Time should also be spent assessing the stresses affecting the patient's life and the psychological impact the hair loss has made on the patient.

CLINICAL EXAMINATION

Careful examination of the scalp and all areas bearing hair on the body, including eyelashes and eyebrows, should be performed because hair loss can affect any hair bearing site. The distribution and amount of hair loss at each site should be noted. Clinical photographs can accurately document the site and extent of hair loss. The Ludwig[11] and Savin[44] scales are useful for grading FPHL, whereas Olsen et al[45] has devised a scale useful in estimating hair loss in AA. Loss of body hair or eyelashes and eyebrows is mostly seen in AA but could also be evidence of trichotillomania. An assessment of whether the hair loss is patterned, localized, or diffuse should be made.[9] Patterned hair loss over the crown suggests FPHL.[46] Localized regular round patches of hair loss can be seen in AA, whereas irregular patches of alopecia are suggestive of trichotillomania, tinea capitis, traction alopecia, and some types of scarring alopecia. A decision should be made whether the type of hair loss is scarring or nonscarring.[9] Usually, visible follicular openings support a type of nonscarring alopecia, and loss of visible follicular openings support the diagnosis of a type of scarring alopecia.[9]

Length, diameter, and breakage of the hair shafts should also be assessed.[9] The presence of scalp inflammation with erythema, scale, and follicular plugging should be carefully noted.[9] Dermatoscopic examination of the scalp can also enhance evaluation of surface changes.[17,47] Videodermatoscopic examination can also add to this evaluation.[48] Scalp inflammation can be seen with inflammatory scalp conditions, such as psoriasis or eczema, or an inflammatory scarring alopecia, or an infective disorder such as seborrheic dermatitis or tinea capitis. A Wood lamp examination of the scalp can demonstrate scaling more effectively[17] and reveal *Malassezia* (*Pityrosporum*) species, the causative organism for seborrheic dermatitis,[36] fluorescing orange,[17] whereas some species causing tinea capitis fluoresce green.[36]

Skin and nail assessment is helpful and an important component of the clinical examination.[17] Autoimmune conditions, AA, thyroid disorders, and nutritional deficiencies may be associated with nail dystrophy.[20] The nail change of Beau lines may suggest the diagnosis of telogen effluvium signaling a relatively recent acute severe physiological stress.[17] Acne, seborrhea, and hirsutism, which could be signs of underlying hyperandrogenism should be noted.[6,9] Rashes in photosensitive sites are suggestive of lupus erythematosus, and skin findings that suggest an underlying systemic disorder such hypothyroidism or hyperthyroidism should be documented.

Further diagnostic tests, such as hair pulls and collections (shed hair), are very helpful (Table 1). A hair collection is done daily for 2 weeks by the patient at home.[17] Loss of more than 100 hairs per day suggests an effluvium.[17] The hair pull test is performed at the office and can be useful.[17] Light microscopy can identify telogen and dystrophic anagen hairs and tinea capitis infection. Hair shaft microscopy can also identify a trichodystrophy and some underlying nutritional deficiencies.[17,20]

LABORATORY INVESTIGATIONS

Laboratory workup is essential in women experiencing hair loss (Table 1). Possible triggers and contributing causes of hair loss can be screened for with blood work. A complete blood count and serum ferritin level can be performed to look for anemia and iron deficiency.[49] A comprehensive metabolic panel will exclude chronic renal or liver disease. A thyroid-stimulating hormone and free thyroxine level can identify thyroid disease.[9] A serum zinc level screens for zinc deficiency.[17] If warranted clinically, an antinuclear antibody level can screen for systemic lupus erythematosus, and syphilis serology can screen for syphilis infection as causes for hair loss.[9,20] A basic androgen workup of total and free testosterone, dehydroepiandrostenedione sulfate, and sex hormone–binding globulin levels is needed to exclude a virilizing tumor, polycystic ovarian syndrome, or other endocrine disorder, if hyperandrogenism features are present or if a woman presents with a male type PHL (Table 1).[6,9] Blood work for autoimmune screening is also important. Antimicrosomal or antithyroid peroxidase antibodies should be done to exclude autoimmune thyroid disease.[17] A vitamin B12 level, antinuclear antibodies, and a glucose level can screen for pernicious anemia, lupus erythematosus, and diabetes mellitus, respectively. Levels of vitamins A and D can also be investigated if warranted from the history (Table 1).

Fungal scrapings and cultures of scalp scale and hair shafts should be performed if tinea capitis is suspected.[9] Pustules require bacterial culture and sensitivities. Light microscopy of clipped hair can demonstrate features of trichodystrophies and nutritional deficiencies.[17,20] Histology is also useful and two 4-mm scalp biopsies are most helpful with horizontal and vertical sectioning (Table 1).[9,17]

TABLE 1

Hair Loss Investigations for Women

Basic Laboratory Investigations	Reason for Screening
Complete blood count	Anemia
Comprehensive metabolic panel	Renal and liver diseases
Nutritional Screening Investigations	
Iron studies	Anemia
Ferritin level	Iron deficiency
Zinc level	Zinc deficiency
Vitamin A	Vitamin A deficiency
Vitamin D (25-hydroxy vitamin D)	Vitamin D deficiency
Endocrine Screening Investigations	
Thyroid function tests Thyroid-stimulating hormone level Free thyroxine level	Thyroid disorder
Androgen screening Testosterone level (free and total) Sex hormone–binding globulin Dihydroepiandrostenedione	Hyperandrogenism
Autoimmune Screening Investigations	
ANA titer	Systemic lupus erythematosus
Autoimmune screening Thyroid autoantibodies Fasting glucose level Vitamin B12 level	Autoimmune associations
Hair Shaft Investigations	
Hair pull test	Hair shedding disorder
Hair collection (done by patient)	Hair shedding disorder
Light microscopy of hair shaft clippings	Hair shaft defect
Two 4-mm scalp biopsies (horizontal and vertical sectioning)	Helpful in hair loss conditions
Microbiological Investigations	
Fungal scraping and culture	Fungal infection
Bacterial culture	Bacterial infection
Syphilis serology	Syphilis infection

Abbreviation: ANA, antinuclear antibody.

TREATMENT

The most important part of treatment of hair loss in women is support and education of the patient.[18] It can be helpful to discuss the normal hair growth cycle and provide the patient with written information regarding their diagnosis. Nutritional deficiencies should be corrected.[18] Education regarding a balanced healthy diet should be discussed and may support hair growth. Oral zinc replacement is the therapy of choice for zinc deficiency.[17] Zinc levels should be monitored and supplementation stopped when zinc levels return to normal. Ferritin levels have been suggested to be kept above 70 ng/ml for hair loss regardless of the presence of anemia.[17,49] Hemoglobin, iron, and ferritin levels need to be monitored to avoid iron overload.[17] In some cases, subclinical biotin deficiency may contribute to hair loss, and biotin supplements may promote hair growth.[17] Biotin levels are not formally performed, but supplementation is quickly metabolized and has a low potential for toxicity.[17] Vitamin D is an important vitamin in cell growth, and theoretically, low levels of vitamin D may be an exacerbating factor in hair loss.[17] Vitamin A levels should be within normal range as fluctuations in the level may precipitate hair shedding.[17] Reduction of emotional stress through yoga or exercise may also aid in reducing the patient's stress level and assist in coping with the anxiety of hair loss (M. Piliang, MD, W.F. Bergfeld, MD, oral communication, January 2009). In certain hair loss conditions, hair styles, hair pieces, and hair transplants can assist in the management of some women.

Antidandruff and antifungal shampoos can be helpful in a variety of hair loss conditions in women. Zinc pyrithione and ketoconazole shampoos are not only useful in treating seborrheic dermatitis and tinea capitis, but can also be useful to debride scales from the scalp in inflammatory alopecias (W.F. Bergfeld, MD, oral communication, January 2009). These shampoos have an additional effect of partially enhancing hair growth in a telogen effluvium.[17] Scalp inflammation should also be treated with short-term moderate potency topical corticosteroid preparations until the inflammation has settled.

For FPHL, treatment options are topical minoxidil and antiandrogens. Topical minoxidil 2% twice daily is the only approved treatment by the US Food and Drug Administration (FDA) for women over 18 years of age for FPHL (Table 2).[6] The minoxidil 5% solution is clinically superior when used twice daily but is not FDA approved for women.[6,17,50] Facial and generalized hypertrichosis can occur as side effects.[6] Scalp irritation, erythema, and dryness are more common adverse effects.[6] Topical minoxidil should not be used in pregnant or nursing women.[6] Systemic antiandrogen therapy, with androgen receptor antagonists like spironolactone or cyproterone acetate, is helpful for FPHL but studies are limited and it is an off-label usage.[16] Spironolactone is given 50 mg to 200 mg daily for FPHL.[6,51] Cyproterone acetate is not approved by the FDA.[6,52,53] Other therapeutic options include the combined oral contraceptive pill with a low androgenic progestogen, such as drosperinone, norgestimate, or gestodene, but no controlled studies are available.[42,53] In postmenopausal women, finasteride has failed to improve FPHL[54] and is contraindicated in premenopausal women (pregnancy category X). Although there are no controlled trials using combination therapy of topical minoxidil and an antiandrogen for the treatment of FPHL, they are commonly used together (M. Piliang, MD, W.F. Bergfeld, MD, oral communication, January 2009).

In acute telogen effluvium, if the trigger can be removed, the shedding is short lived and requires no further treatment (Table 2).[18] Chronic diffuse telogen hair loss is more complex as multiple or sequential factors can be involved. Any underlying thyroid disease, systemic illness, or infection should be managed appropriately. If a drug is suspected as a cause of hair loss, it should be ceased or changed for at least 3 months.[17,19] Topical minoxidil 2% or 5% twice daily may be helpful in chronic diffuse telogen hair loss and CTE to promote hair regrowth (Table 2).[53]

There are no available FDA-approved treatments for AA (Table 2).[53] Support group information should be given to the patient.[53] In AA with isolated or limited disease, a potent topical corticosteroid daily or serial 6- to 8-weekly intralesional corticosteroid injections are used to suppress local inflammation.[27,53] Response can take up to 3 to 4 months in some patients and relapse can occur once treatment is stopped or once the injections have worn off.[53] For more widespread disease on the scalp, topical 5% minoxidil twice daily can be helpful but results are variable.[53] Other topical agents include anthralin cream (0.5%–1.0%), which can be used as short-contact therapy.[27,28,53] There are many reports of the topical contact sensitizer diphenylcyclopropenone being successful in the treatment of AA, but it is an unapproved FDA therapy.[53] In some institutions, ethics committee approval is needed for diphenylcyclopropenone use. A consent form should be signed by the patient before treatment after explanation of the risks and benefits and its status as an unapproved FDA therapy. Combination topical treatments have also been used in the treatment of AA.[53] Systemic therapies, such as corticosteroids or cyclosporin for severe disease, are not usually recommended due to adverse effects and recurrence of hair loss after stopping therapy.[27,28,53] The new biological agents have been unsuccessful in the treatment of AA.[55,56]

TABLE 2

Treatments for Hair Loss Conditions in Women

Condition	General	Topical	Systemic or Intralesional
FPHL	Multivitamin Biotin and zinc supplement Adequate dietary intake Stress reduction techniques	Antidandruff shampoo (1% zinc pyrithione or 1% ketoconzole) Topical minoxidil 2% or 5%	Spironolactone Cyproterone acetate Low androgenic oral contraceptive pills
Acute telogen effluvium	Remove or correct trigger Multivitamin Biotin and zinc supplement Adequate dietary intake Stress reduction techniques	Antidandruff shampoo (1% zinc pyrithione or 1% ketoconzole)	
CTE	Remove or correct trigger Multivitamin Biotin and zinc supplement Adequate dietary intake Stress reduction techniques	Antidandruff shampoo (1% zinc pyrithione or 1% ketoconzole) Topical minoxidil 2% or 5%	
AA	Multivitamin Biotin and zinc supplement Adequate dietary intake Stress reduction techniques	Antidandruff shampoo (1% zinc pyrithione or 1% ketoconzole) Topical corticosteroids Topical minoxidil 5% Topical anthralin Topical diphenylcyclopropenone	Intralesional corticosteroids
Traction alopecia	Avoid hair styles with traction	Topical minoxidil 2%	
Trichotillomania	Multivitamin Biotin and zinc supplement Adequate dietary intake Stress reduction techniques	Antidandruff shampoo (1% zinc pyrithione or 1% ketoconzole)	Psychiatric intervention with psychological and pharmacological therapies
Tinea capitis	Avoid sharing hats and scarves	Antifungal shampoo (1% zinc pyrithione or 1% ketoconzole)	Oral antifungal agent (griseofulvin)
Scarring alopecias	Multivitamin Biotin and zinc supplement Adequate dietary intake Stress reduction techniques	Antidandruff shampoo (1% zinc pyrithione or 1% ketoconzole) Topical potent corticosteroids	Intralesional corticosteroids Oral antibiotics
Acquired trichodystrophy	Avoid harsh cosmetic processes and overstyling of the hair	Antidandruff shampoo (1% zinc pyrithione or 1% ketoconzole) Leave-in conditioner	

Abbreviations: FPHL, female pattern hair loss; CTE, chronic telogen effluvium; AA, alopecia areata.

Treatment will be only briefly discussed for the remaining hair loss disorders (Table 2). The mainstay of management for trichodystrophies is ceasing cosmetic processing and styling with heat.[42] Certain hair care products may be helpful such as leave-on conditioners.[42] In traction alopecia, changing the hairstyle is essential to prevent permanent hair loss from the chronic traction.[42,30] Topical minoxidil 2% twice daily has also been reported for

traction alopecia.[33] Psychotherapy is an important part of management in trichotillomania, in addition to pharmacologic measures.[30,35] Treatment for tinea capitis is with an oral antifungal agent and antifungal shampoo.[36] Combination treatment is usually needed for scarring alopecias, but involves potent topical or intralesional corticosteroids.[39] Antibiotics like tetracycline or doxycycline can also be helpful in combination with topical or intralesional corticosteroids.[39]

SUMMARY

A detailed history and careful clinical examination are essential components of an approach to hair loss in women. Laboratory workup and scalp biopsy can assist in making the diagnosis. Treatment is based on the diagnosis, the clinician, and patient preference and convenience. Patient education is also an essential part of management of women with hair loss.

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