CME

Practical management of hair loss

Jerry Shapiro, MD, FRCPC Marni Wiseman, MD Harvey Lui, MD, FRCPC

abstract

OBJECTIVE To describe an organized diagnostic approach for both nonscarring and scarring alopecias to help family physicians establish an accurate in-office diagnosis. To explain when ancillary laboratory workup is necessary to confirm the diagnosis.

QUALITY OF EVIDENCE Current diagnostic and therapeutic interventions for hair loss are based on randomized controlled studies, uncontrolled studies, and case series. MEDLINE was searched from January 1966 to December 1998 with the MeSH words alopecia, hair, and alopecia areata. Articles were selected on the basis of experimental design, with priority given to the most current large multicentre controlled studies. Overall global evidence for therapeutic intervention for hair loss is quite strong.

MAIN MESSAGE The most common forms of nonscarring alopecias are androgenic alopecia, telogen effluvium, and alopecia areata. Other disorders include trichotillomania, traction alopecia, tinea capitis, and hair shaft abnormalities. Scarring alopecia is caused by trauma, infections, discoid lupus erythematosus, or lichen planus. Key to establishing an accurate diagnosis is a detailed history, including medication use, systemic illnesses, endocrine dysfunction, hair-care practices, and family history. All hair-bearing sites should be examined. A 4-mm punch biopsy of the scalp is useful, particularly to diagnose scarring alopecias. Once a diagnosis has been established, specific therapy can be initiated.

CONCLUSIONS Diagnosis and management of hair loss is an interesting challenge for family physicians. An organized approach to recognizing characteristic differential features of hair loss disorders is key to diagnosis and management.

résumé

OBJECTIF Décrire une approche diagnostique structurée pour l'alopécie cicatrisante et non cicatrisante afin d'aider les médecins de famille à poser un diagnostic exact en cabinet. Expliquer les circonstances où il est nécessaire de procéder à des épreuves auxiliaires en laboratoire pour confirmer le diagnostic.

QUALITÉ DES DONNÉES Les interventions diagnostiques et thérapeutiques actuelles pour la perte de cheveux et de poils se fondent sur des études aléatoires contrôlées, des études non contrôlées et des séries de cas. Une recension a été effectuée dans MEDLINE, de janvier 1966 à décembre 1998, à l'aide des termes MeSH en anglais pour alopécie, cheveux et alopecia areata ou pelade. Les articles ont été sélectionnés en se fondant sur la conception expérimentale, accordant la priorité aux études multicentriques contrôlées les plus récentes. Dans l'ensemble, les données probantes sur les interventions thérapeutiques pour la perte de cheveux et de pilosité sont assez solides.

PRINCIPAL MESSAGE Les formes les plus courantes d'alopécie non cicatrisante sont l'alopécie androgène, l'effluvium télogène et la pelade. Au nombre des autres problèmes figurent la trichotillomanie, l'alopécie de traction, la teigne et les anomalies de la tige capillaire. L'alopécie cicatrisante est causée par les traumatismes, les infections, le lupus discoïde, l'érythème ou le lichen plan. Il est essentiel à un diagnostic précis de faire une anamnèse détaillée, notamment l'usage de médicaments, l'existence de maladies systémiques, une dysfonction endocrine, les pratiques dans les soins capillaires et les antécédents familiaux. Il faudrait examiner toutes les parties du corps portant des cheveux ou des poils. Une biopsie au poinçon de 4 mm de cuir chevelu peut se révéler utile, en particulier pour le diagnostic des alopécies cicatrisantes. Une fois le diagnostic posé, une thérapie précise peut être entreprise.

CONCLUSIONS Le diagnostic et la prise en charge de l'alopécie posent des défis intéressants aux médecins de famille. Une approche structurée pour reconnaître les caractéristiques différentielles distinctives des problèmes de perte de cheveux et de poils est essentielle au diagnostic et à la prise en charge.

This article has been peer reviewed.
Cet article a fait l'objet d'une évaluation externe.
Can Fam Physician 2000;46:1469-1477.

Hair loss is a common problem and often a source of distress for patients. Alopecia can be either scarring or nonscarring. Androgenic alopecia (AGA), alopecia areata, and telogen effluvium are the most common causes of nonscarring alopecia.

The first task of physicians is to address patients' concerns fully, exploring the effect of alopecia on psychosocial well-being. Next, an organized diagnostic approach can help family physicians recognize characteristic features of each disorder to identify the cause of alopecia and guide therapy. Ancillary laboratory evaluation is sometimes necessary to confirm a diagnosis. Many effective therapeutic options are available and easily performed in an office. Patients often appreciate a supportive diagnostic and therapeutic approach.

Quality of evidence

A computerized MEDLINE database was searched from January 1966 to December 1998 to identify articles published in English with the MeSH words alopecia, hair, and alopecia areata. Articles were selected on the basis of experimental design, with priority given to the most current large multicentre controlled studies. Studies with objective hair growth end points, such as photographs, hair counts, or hair weight methods, were also given priority.

Use of minoxidil for alopecia areata and AGA is based on randomized placebo-controlled trials. Use of finasteride for AGA is based on multicentre randomized placebo-controlled studies. Use of diphenylcyclopropenone for alopecia areata is also supported by controlled trials. Other data are supported by clinical trials, case series, review articles, and 12 years of experience at a tertiary hospital–based specialty hair clinic.

General diagnostic approach

Many factors cause clinical hair loss, or alopecia, including endocrine abnormalities, genetic predisposition, systemic illness, drugs, psychological abnormalities, diet, trauma, infections, autoimmunity, and structural hair defects. Because of the multiplicity of disorders that can result in hair loss, a thorough history and physical examination are important, and ancillary laboratory workup could be necessary.

The history is critical in developing an initial differential diagnosis (**Table 1**). Duration and pattern (ie, diffuse versus focal) of hair loss are important to determine. A full list of current and past medications should be obtained because many medications can induce hair loss. A family history of alopecia areata or AGA can point to a genetic predisposition for hair loss. In addition, coincidental acne and abnormal menstrual cycles can indicate an androgen excess causing AGA. Answers to thyroid screening questions can point to

Dr Shapiro *is a Clinical Associate Professor in the Division of Dermatology at the University of British Columbia (UBC) in Vancouver and founding Director of the UBC Hair Research and Treatment Centre.*
Dr Wiseman *is a resident in the Division of Dermatology at UBC.* **Dr Lui** *is an Associate Professor in the Division of Dermatology at UBC and is Associate Director of the UBC Hair Research and Treatment Centre.*

Table 1. Questionnaire on history of hair loss

What is the duration and pattern of hair loss?
Is hair coming out by the roots, or is it breaking?
Is increased shedding or increased thinning apparent?
What was age of onset?
Does patient take any drugs?
Is there a relationship with menses, pregnancy, or menopause?
What is present and past health?
Is thyroid gland functioning?
Is there a family history of hair loss?
Does patient have unusual hair care or use hair cosmetics?
What is patient's diet?

Table 2. Differential diagnosis

HAIR COMING OUT BY ROOTS
• Telogen effluvium
• Androgenic alopecia
• Alopecia areata
• Drugs
HAIR BREAKING
• Tinea capitis
• Structural hair shaft abnormalities
• Breakage due to improper use of hair-care cosmetics
• Anagen arrest

hypothyroidism, and a strict vegetarian diet can implicate iron deficiency anemia. Some hair-care practices (eg, bleaching, back-combing, permanent waving) break hair. It is important to establish whether the hair falls out from the roots or breaks off along the shafts, because there are completely different causes for each (**Table 2**). It is also important to ask about loss of axillary and pubic hair, eyelashes, eyebrows, and body hair, because any hair-bearing area can be affected by alopecia areata or trichotillomania.

Clinical examination should be performed in three stages. First inspect the scalp for inflammation, scale, and erythema. It is important to determine whether hair loss is associated with scalp scarring (**Table 3**). Nonscarring alopecias demonstrate visible follicular units, while scarring alopecias are devoid of follicular units (**Figure 1**). Second, examine the pattern of distribution and density of hair. Finally, study the quality of the hair shaft in terms of calibre, fragility, length, and shape.

Table 3. **Causes of alopecia**

NONSCARRING
• Androgenic alopecia
• Telogen effluvium
• Alopecia areata
• Traction alopecia
• Tinea capitis
SCARRING
• Discoid lupus erythematosus
• Lichen planus
• Severe fungal, viral, or bacterial infection
• Injury or burn

To determine the ongoing activity of hair loss, a useful ancillary test, the "pull test" should be conducted. Approximately 60 hairs are grasped between the thumb, index finger, and middle fingers from the base near the scalp, and firmly, but not forcefully, tugged away from the scalp (**Figure 2**). If more than 10%, or six hairs, are pulled away from the scalp, this constitutes a positive pull test and implies active hair shedding. If fewer than six hairs can be easily pulled out, this is considered normal physiologic shedding. The patient must not shampoo for at least 1 day before the pull test. The pull test helps to assess severity and location of hair loss.

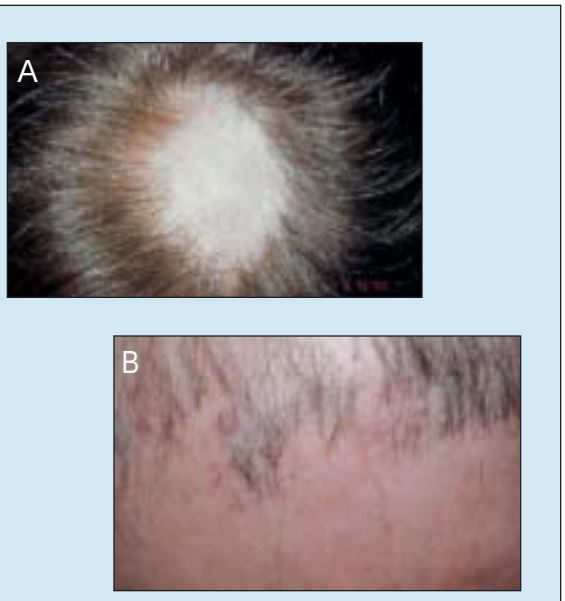


Figure 1. **Alopecia**: *A) Nonscarring alopecia demonstrating multiple follicular units; B) Scarring alopecia demonstrating a smooth scalp devoid of follicular units.*

Laboratory tests can help to establish a diagnosis. Evaluation of serum ferritin could be necessary to exclude iron deficiency anemia, particularly in women with diffuse alopecia. If thyroid dysfunction is suspected, thyroid-stimulating hormone level should be investigated. In women with AGA and such virilizing signs as hirsutism, acne, or irregular menses, an endocrinologic workup consisting of free testosterone, androstenedione, and dehydroepiandrosterone (DHEA) is advised to rule out hyperandrogenism.[1,2] In cases of confirmed scarring alopecia due to discoid lupus erythematosus, an antinuclear antibodies (ANA) examination should performed.

Not only are various scarring alopecias difficult to differentiate from each other clinically but they can also be difficult to distinguish from nonscarring alopecias. For these reasons, it is almost always necessary to perform a 4-mm scalp biopsy when evaluating patients suspected of having scarring alopecia.

Patients' concerns and expectations should be acknowledged and fully explored. Many patients with hair disorders become frustrated when their worries about hair loss are either ignored or dismissed as insignificant. Explanation and discussion could resolve problems without specific intervention. Occasionally underlying depression or dysmorphophobia (pathologic fixation on body image) is present.

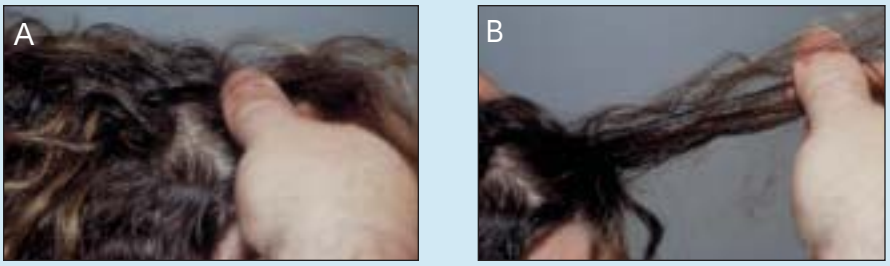


Figure 2. **Hair pull test:** *A) Approximately 60 hairs are grasped between the thumb, index finger, and middle fingers near the scalp; B) Hair is firmly, but not forcibly, tugged away from scalp as fingers slide along the hair shaft.*

These psychiatric conditions must be recognized and managed before any further treatment is initiated.

Nonscarring alopecias

In nonscarring alopecias, follicles appear preserved on clinical and histologic examination, although they can sometimes be difficult to appreciate when miniaturized. The three most common forms of nonscarring alopecias are AGA, telogen effluvium, and alopecia areata. **Table 4** compares key clinical features that distinguish these three conditions from one another.

Androgenic alopecia. Approximately 95% of hair loss cases are AGA. Fifty percent of men by age 50 years, and 40% of women by menopause exhibit some degree of AGA.[3] Hair loss is gradual, with miniaturization of genetically programmed hair follicles being mediated by increased uptake, metabolism, and conversion of testosterone to stanolone by 5-α reductase in susceptible hair follicles.

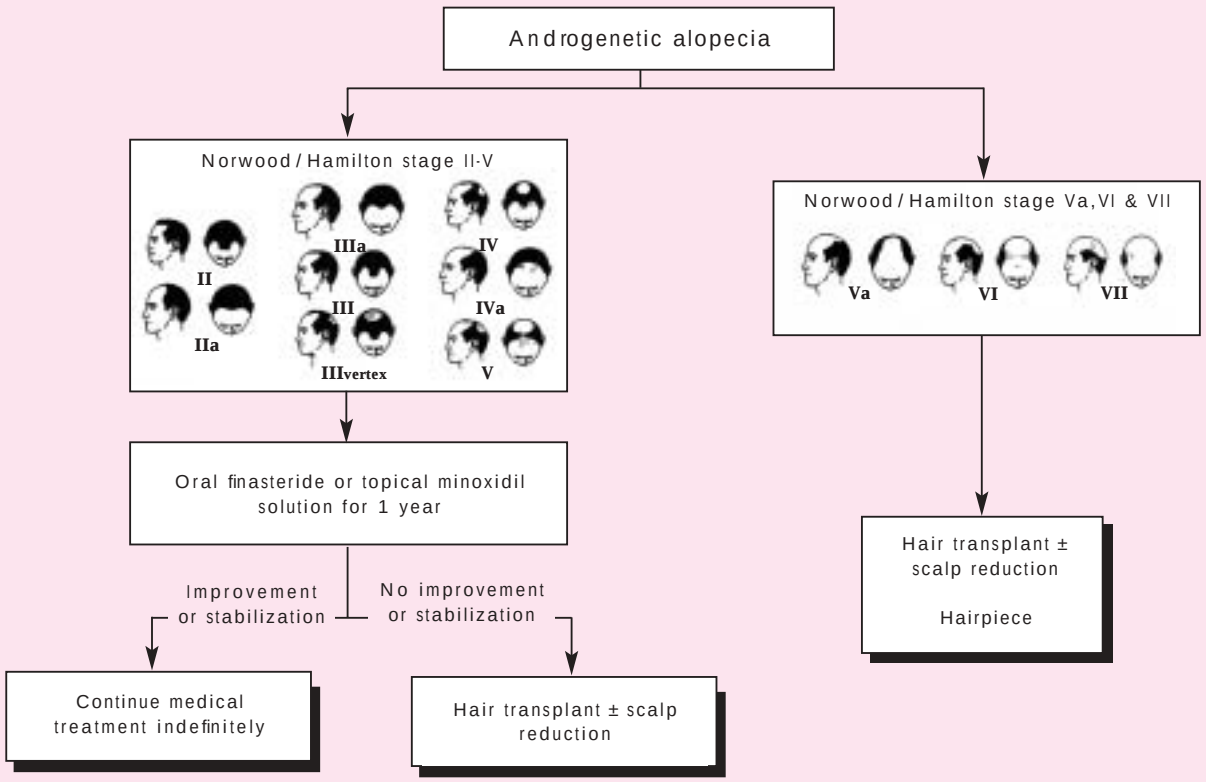
Androgenic alopecia presents differently in men than in women. Men with AGA lose hair in the frontotemporal and vertex regions of the scalp in varying degrees depending on the severity of the disorder. A formal staging system is the Norwood/Hamilton scale[4] (**Figure 3**). In female AGA patients, balding is more diffuse with a more centro-parietal distribution and relative preservation of the frontal hair line. In women, the analogous grading system is the Ludwig classification (**Figure 4**).

Treatment of AGA is either medical or surgical. Only two medications with proven efficacy are indicated for AGA: oral finasteride and topical minoxidil.[5] Finasteride (1 mg) has been shown to

Table 4. **Common hair loss disorders**

CHARACTERISTIC	ANDROGENIC ALOPECIA	TELOGEN EFFLUVIUM	ALOPECIA AREATA
Hair loss distribution	Focal balding pattern: Hamilton/Norwood (men) Ludwig (women)	Generalized	Usually patchy but can be generalized
Course	Gradual onset with progression	Onset abrupt, often with trigger factor	Onset abrupt; often waxes and wanes with relapses
Appearance	Thinning with or without bare patches. Bare patches are gradual, not abrupt	Thinning with no bare patches	Thinning with abrupt bare patches; exclamation mark hairs
Shedding	Minimal	Prominent	Prominent
Age of onset	Onset at puberty or older	Onset at any age, but usually not childhood	Onset at any age; most have their first patch before age 20
Pull test	Usually negative	Positive; telogen hairs	Positive; dystrophic anagen and telogen hairs

Figure 3. **Treatment plan for AGA in men**



With permission © University of California, San Francisco (UCSF) and University of British Columbia (UBC).Courtesy of Jerry Shapiro, MD, Vera H. Price MD, and Harvey Lui, MD.

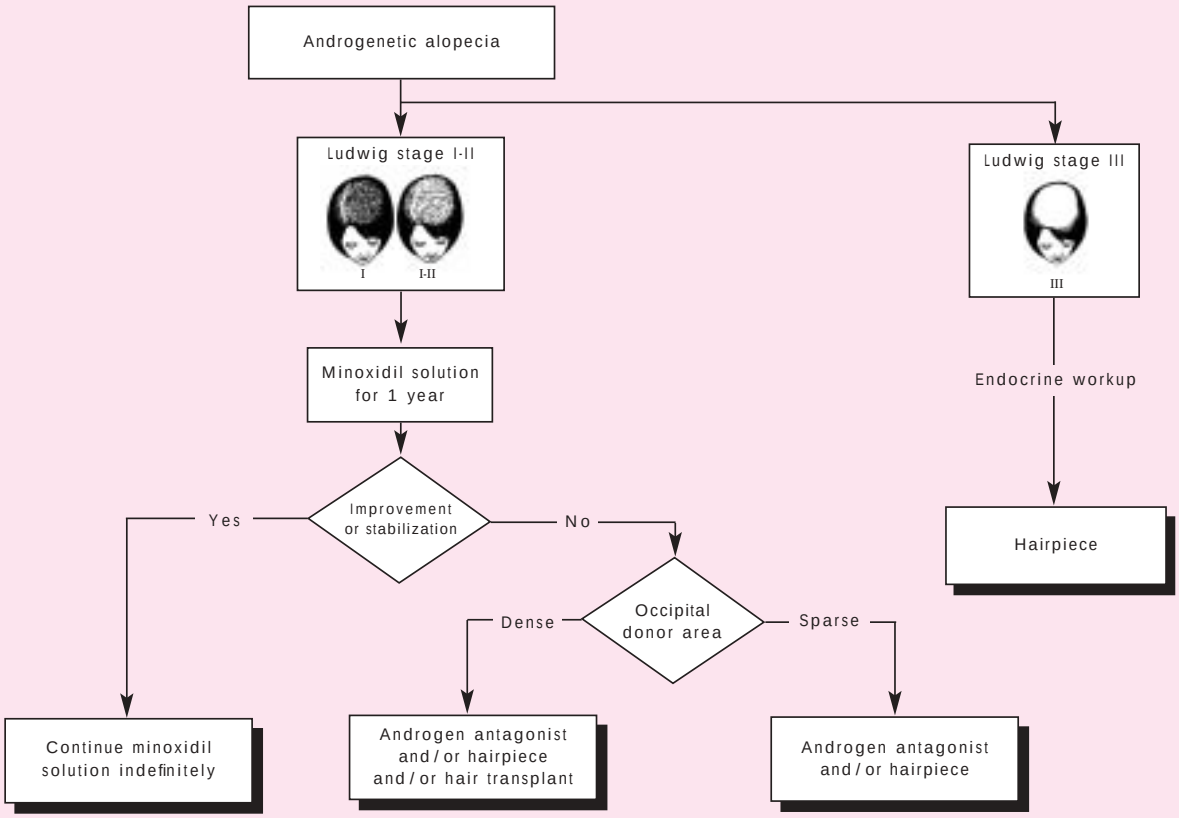
have efficacy in male AGA patients aged 18 to 41 with Norwood/Hamilton stage II-V. Sixty-six percent showed regrowth and 83% showed stabilization after a 2-year follow up in a double-blind, placebo-controlled clinical trial of 1553 men.[5] Finasteride is contraindicated in women of childbearing potential because of the risk of external genitalia abnormalities in male fetuses. Randomized controlled studies have not supported use of finasteride in postmenopausal women. Finasteride is prescribed as a once daily oral dose of 1 mg.

Topical minoxidil can regrow cosmetically acceptable terminal hair in approximately 10% of men.[2] In women, 50% show minimal regrowth and 13% moderate regrowth.[6] Seven percent of patients experience some irritation (burning, itching, redness) from topical minoxidil, and 3% to 5% of women develop facial hypertrichosis.[3] The 5% minoxidil solution has greater efficacy than the 2% minoxidil solution and is currently available without a prescription in the United States, but not in Canada. Treatment with either finasteride or minoxidil must be continued indefinitely to maintain any clinical effect.[7] In women, use of systemic antiandrogens, such as spironolactone (50 to 200 mg daily),[8] cyproterone acetate, or flutamide, might reduce hair thinning slightly, but insufficient hard clinical data support this.[9]

The surgical approach to AGA, which is based on transplanting permanent hairs from the back and sides of the scalp to balding areas in the front, usually requires two to four operative sessions before adequate hair density can be achieved at recipient sites. Use of mini-grafts and micrografts containing two to four follicles rather than 2- to 4-mm cylindrical plug grafts, has revolutionized hair transplantation by allowing for a more natural-looking result that is free of obvious hair clumping or tufting. Furthermore, harvesting donor hairs in the form of strips rather than plugs has made donor sites more cosmetically acceptable. Hair transplantation is useful not only for men but also for women. Treatment algorithms for male and female AGA based on clinical staging are presented in **Figures 3 and 4.**

Telogen effluvium. Telogen hairs are resting hairs that have a nonpigmented club tip at the proximal root

Figure 4. **Treatment plan for AGA in women**



With permission © University of California, San Francisco (UCSF) and University of British Columbia (UBC).Courtesy of Jerry Shapiro, MD, Vera H. Price MD, and Harvey Lui, MD

Table 5. **Causes of telogen effluvium**

• Childbirth
• Severe infection
• Severe chronic illness
• Severe psychological stress
• Major surgery
• Hypothyroidism or hyperthyroidism
• Iron deficiency anemia
• Crash diets
• Drugs

and can be pulled easily from the scalp (**Figure 5**). Excessive generalized shedding of normal telogen club hairs can be induced by several stresses: parturition, febrile illness, psychological stress, crash diets, and drugs (**Table 5**). These factors induce hair loss by abruptly shifting hair follicles from their growing phase (anagen) into their resting phase (telogen). Hair loss does not actually occur until 2 to 4 months after the period of initial stress, since hair shafts are usually not shed until 2 to 4 months after entering the telogen phase. Thus, hair loss may be seen 2 to 4 months into the postpartum period.

An average normal scalp has 100 000 hairs, with approximately 90% of these normally in anagen and 10% in telogen.[2] Average normal daily hair fall-out is 100 telogen hairs. With telogen effluvium, the anagen-telogen ratio is shifted to 70% anagen, 30% telogen, with average daily shedding of 300 hairs.[2] Treatment is

Figure 5. **Telogen club hair.**

based on identifying the specific cause and correcting it. In most cases, complete recovery occurs over 4 to 6 months, provided the triggering factor is resolved.[2,10] Chronic telogen effluvium of unknown origin, lasting months to years, can also occur in women.

Alopecia areata. Alopecia areata is considered an autoimmune disease that affects 1.7% of the population.[2] It usually presents with an oval patch or multiple confluent patches of asymptomatic, well-circumscribed, nonscarring alopecia. Ten percent of cases proceed to alopecia totalis, in which the entire scalp is devoid of hair, and 1% develop alopecia universalis, which affects all body hair, including eyebrows and eyelashes.[2]

Fifty percent of cases resolve spontaneously within 1 year of onset of hair loss, but relapses are common.[2] Severe alopecia areata is less likely to resolve on its own, especially in children or atopic individuals. Fingernails might show dystrophic changes, such as pitting, ridging, and thinning of the nail plate, in association with alopecia areata. Exclamation mark hairs, short hairs that taper proximally, are seen at the periphery of the patch and are specific for alopecia areata.

Treatment of alopecia areata depends primarily on extent of disease and age of patients. Once the natural history of alopecia areata is explained, patients can elect to be observed for several months and defer active treatment. For small patchy disease, intralesional corticosteroid is the treatment of choice. Triamcinolone acetonide suspension diluted to 5 mg/mL is injected with a 30-gauge needle directly into the balding patches at volumes of 0.1 mL per injection. Injections are dispersed over affected areas. Total dose of injected triamcinolone acetonide should not exceed 10 mg (2 mL of 5 mg/mL) per visit. Injections are repeated every 4 to 6 weeks. If hair growth is observed, injections can be continued. If no hair growth is observed after 3 months, injections should be stopped.[3]

Figure 6. **A 40-year-old woman with alopecia universalis of 20 years' duration who received topical immunotherapy for 24 weeks to only the left side of the scalp:** *Only the treated side grew cosmetically acceptable terminal hair.*

Other treatment options for alopecia areata include topical steroids, minoxidil, or anthralin. Use of topical minoxidil and steroids has been supported by clinical trials.[3,11-14] For more extensive disease, defined as 50% or more scalp involvement, topical immunotherapy with a contact allergen called diphenylcyclopropenone is the treatment of choice, with some studies showing 40% rates of hair regrowth (**Figure 6**).[3,15,16] Systemic steroids or phototherapy with psoralens in combination with ultraviolet A radiation (PUVA) can also be used for severe alopecia areata, but use of systemic steroids is controversial, because patients might have to continue the

Table 6. **Treatment of alopecia areata**

ADULTS WITH < 50% HAIR LOSS
Observe for several weeks
Administer intralesional corticosteroids
Apply minoxidil 5% solution ± corticosteroid cream or anthralin
ADULTS WITH > 50% HAIR LOSS
Apply topical immunotherapy with diphenylcyclopropenone
Administer psoralens ultraviolet A light treatment
Apply minoxidil 5% solution ± corticosteroid cream or anthralin
CHILDREN
Administer minoxidil 5% solution ± corticosteroid cream or anthralin

drug for a long time.[3,15] Although systemic steroids are highly effective, hair shedding promptly occurs upon their discontinuation. **Table 6** lists treatments for alopecia areata.

Patients with alopecia areata require encouragement and emotional support. Physicians need to take time with them to discuss the social and psychological effects of their disease and to address their concerns. Many cities now have support groups, which are an invaluable resource for many patients. The National Alopecia Areata Foundation, 710 C St Suite 11, San Rafael, CA 94901 USA, is also an excellent resource for patients.

Trichotillomania. A compulsion to pull or pluck one's hair repetitively is called trichotillomania, and can result in unnatural or even bizarre patterns of hair loss. Clinical diagnosis can sometimes be quite difficult, and a scalp biopsy could be required for confirmation. Examination of distal hair shafts for bizarre twisting and fracturing should increase physicians' index of suspicion. Fewer than 5% of such patients have deep-seated psychological disorders, and most cases resolve spontaneously, particularly among children.[17] Clomipramine can be prescribed for severe cases of trichotillomania. A double-blind crossover trial comparing clomipramine with desipramine demonstrated that clomipramine decreased compulsion intensity more than desipramine and was partially effective in controlling this disorder.[17]

Traction alopecia. Traction alopecia is usually due to excessive stretching of hair shafts by hair-styling practices. Bitemporal alopecia is commonly caused by binding hair back and is seen most often in women, particularly East Indian and black women. Hair loss will be focal, depending on the way the hair is being pulled. Prolonged traction alopecia can scar the new hair follicle and cause permanent hair loss.

Tinea capitis. Tinea capitis is due to a dermatophyte infection of the hair follicle and can be inflammatory or noninflammatory. Causative organisms depend on geographic location and environmental exposure to animals. In North America, *Trichophyton tonsurans* and *Microsporum canis* account for most cases of tinea capitis.[2,9,18] Diagnosis is made by potassium hydroxide scraping, mycologic culture, and Wood's light examination. Hair infected by *M canis* fluoresces green under Wood's light, whereas hair infected by *T tonsurans* does not.

Key points

- Most common causes of nonscarring alopecia are androgenic alopecia, alopecia areata, and telogen effluvium.
- Scarring alopecia is caused by trauma, infections, discoid lupus erythematosus, or lichen planus.
- History, examination of hair and skin on the scalp, selected laboratory tests, and 4-mm punch biopsy should make the diagnosis apparent.
- Oral finasteride and topical minoxidil are the only proven medications for androgenic alopecia.
- Management should always involve assessment of the psychological effects of alopecia.

Points de repère

- Les causes les plus communes de l'alopécie non cicatrisante sont l'alopécie androgène, la pelade et l'effluvium télogène.
- L'alopécie cicatrisante a pour cause les traumatismes, les infections, le lupus discoïde, l'érythème ou le lichen plan.
- L'anamnèse, l'examen des cheveux et du cuir chevelu, certaines épreuves en laboratoire et une biopsie au poinçon de 4 mm devraient confirmer le diagnostic.
- La finastéride par voie orale et le minoxidil topique sont les seuls médicaments éprouvés pour l'alopécie androgène.
- La prise en charge devrait toujours comporter l'évaluation des effets psychologiques de l'alopécie.

Antifungal treatment needs to be systemic to penetrate the hair follicle. Griseofulvin, itraconazole, and terbinafine are all effective. Griseofulvin therapy is usually continued for 6 to 8 weeks, at which time a clinical and mycologic cure can be detected. Itraconazole is also efficacious in treating tinea capitis and is well tolerated. Terbinafine has the advantage of a once-daily dosing regimen and minimal drug interaction, but response varies with dermatophyte species. It is important to investigate household pets as a possible source of dermatophyte infection of patients with tinea capitis.

Hair shaft abnormalities. If a patient complains of hair breakage rather than hair falling out at the roots, one of many hair shaft problems could be the culprit. Microscopic examination of the actual shafts (hair mount) is necessary to make the diagnosis. The most common cause of hair fragility is trichorrhexis nodosa, in which hair shafts fracture at the sites of nodes. This defect usually appears as a result of trauma. Overbleaching is another common cause of hair breakage.

Scarring alopecia

Localized areas of scarring alopecia of the scalp can result from trauma, burns, acute fungal infections (such as tinea capitis), viral infections (such as herpes zoster), and bacterial infections. Discoid lupus erythematosus is the most common primary cause of scarring alopecia; lichen planus is also common. Lesions of discoid lupus erythematosus demonstrate marked erythema, atrophy, telangiectasia, and follicular hyperkeratosis. A biopsy is necessary to establish an accurate diagnosis. Evidence of cutaneous disease elsewhere on the skin, oral or genital mucous membranes, and nails should be looked for carefully. Scarring alopecias are considered true trichologic emergencies, as hair loss is irreversible once hair follicles are scarred. Prompt appropriate therapy is crucial. Treatment of discoid lupus erythematosus of the scalp includes intralesional corticosteroids and, if severe, antimalarials, systemic steroids, or retinoids.

Conclusion

Most common hair disorders can be readily diagnosed in physicians' offices through recognition of characteristic features of each disorder. The first task of physicians is to acknowledge patients' concerns and have an empathetic approach to hair loss. Next, an organized diagnostic and management strategy will help to both identify the cause of alopecia and direct therapy. Evaluation of hair disorders can be very interesting and challenging for physicians. Patients often appreciate a supportive diagnostic and therapeutic approach.

Correspondence to*: Dr Jerry Shapiro, University of British Columbia Hair Research and Treatment Centre, Vancouver Hospital and Health Sciences Centre, Division of Dermatology, 835 W 10th Ave, Vancouver, BC V5Z 4E8; telephone (604) 875-4747; fax (604) 873-9919; e-mail* shapiro@interchange.ubc.ca

References

1. Olsen E. Androgenetic alopecia. In: *Disorders of hair growth*. New York, NY: McGraw-Hill, Inc; 1994. p. 257-83.
2. Shapiro J. Diseases of the hair. In: Raykel R, editor. *Conn's current therapy*. Philadelphia, Pa: WB Saunders; 1996. p. 739-41.
3. Shapiro J, Price V. Hair regrowth, therapeutic agents. *Dermatol Clin* 1998;16(2):341-56.
4. Norwood D. Male pattern baldness: classification and incidence. *South Med J* 1975;68:1359-65.
5. Kaufman K, Olsen EA, Whiting O, Savin R, DeVillez R, Bergfeld W, et al. Finasteride in the treatment of men with androgenetic alopecia. *J Am Acad Dermatol* 1998;39:578-89.
6. DeVillez R, Jacobs J, Szpunar CA, Warner ML. Androgenetic alopecia in the female: treatment with 2% minoxidil solution. *Arch Dermatol* 1994;130:303-7.
7. Olsen E. Five-year follow-up of men with androgenetic alopecia treated with topical minoxidil. *J Am Acad Dermatol* 1990;22(4):643-6.
8. Burke B, Cunliffe W. Oral spironolactone therapy for female patients with acne, hirsutism or androgenic alopecia. *Br J Dermatol* 1985;112:124-5.
9. Legendre R, Esola-Macre J. Itraconazole in the treatment of tinea capitis. *J Am Acad Dermatol* 1990;23:559-60.
10. Whiting D. Chronic telogen effluvium. *Dermatol Clin* 1996;14(4):723-31.
11. Leyden J, Kligman A. Treatment of alopecia areata with steroid solution. *Arch Dermatol* 1972;106:924.
12. Montes LF. Topical halcinonide in alopecia areata and alopecia totalis. *J Cutan Pathol* 1977;4:47-50.
13. Price V. Double-blind, placebo-controlled evaluation of topical minoxidil in extensive alopecia areata. *J Am Acad Dermatol* 1987;16:730-6.
14. Winter R, Kern F, Blizzard R. Prednisone therapy for alopecia areata. *Arch Dermatol* 1976;112:1549-52.
15. Shapiro J. Alopecia areata update. *Dermatol Clin* 1993;11:35-45.
16. Shapiro J. Topical immunotherapy in the treatment of chronic severe alopecia areata. *Dermatol Clin* 1993;11:611-7.
17. Swedo SE, Leonard HL, Rapoport JL, Lenane MC, Goldberger EL, Cheslow DL. A double blind comparison of clomipramine and desipramine in the treatment of trichotillomania (hair pulling). *N Engl J Med* 1989;321:497-501.
18. Baudraz-Rosselet F, Monod M, Jaccoud S, Frenk E. Efficacy of terbenafine treatment of tinea capitis in children varies according to the dermatophyte species. *Br J Dermatol* 1996;135:1011-2.