

Common Alopecia

Rattapon Thuangtong, M.D.

Department of Dermatology, Faculty of Medicine Siriraj Hospital, Mahidol University, Bangkok 10700, Thailand.

Siriraj Med J 2007; 59: 30-31

E-journal: <http://www.sirirajmedj.com>

Hair loss (alopecia) is a very common patient's problem and often a significant source of patient distress. There are numerous potential etiologic factors in alopecia. Endocrine abnormalities, genetic factors, systemic illness, drugs, psychological abnormalities, diet, trauma, infections, and structural hair defects may all cause hair loss. Evaluation of the patient must be thorough and include history, physical examination, and appropriate laboratory work-up.

History is of utmost importance in focusing on the correct diagnosis (Table 1). The duration of hair loss, family history, location of hair loss (diffuse vs. focal), drug intake history, hair care habits (bleaching, back brushing, permanent waving), and presence of coincidental acne and abnormal menstrual cycles are all important clues in determining the correct diagnosis. It is important to determine whether the hair falls out from the roots or breaks off along the shaft since there are separate distinct differential diagnoses for each of these problems (Table 2).

Clinical examination of all hair-bearing areas should be performed in three stages. First inspect for inflammation, infection and scarring (loss of the hair follicle) is of utmost importance. Second, examine the pattern of density and distribution of hair. Finally, study the quality of hair shaft in terms of caliber, fragility, length and shape. Pull test, Plug test and hair count sometime helpful in the diagnosis. The differential diagnosis of hair loss is based on whether the hair loss is scarred or not (Table 3).

Regarding laboratory testing, nonscarring alopecia may require a complete blood count, thyroid-stimulating hormone, serum ferritin, and VDRL. In women with androgenetic alopecia and other virilizing signs, an androgen work-up for free testosterone, androstenedione, and dehydroepiandrosterone (DHEAS) is advised. Scarring alopecias are difficult to differentiate from one another

TABLE 1. Hair loss history questionnaire

What is the duration of the hair loss problem?
Is the hair coming out by the roots, or is it breaking?
Increased shedding or increasing thinning?
Age of onset
Drugs
Menses, pregnancy, menopause
Past health
Family history
Hair care, hair cosmetics
Diet

TABLE 2. Differential diagnosis

Hair coming out by roots	Hair breaking
Telogen effluvium	Tinea capitis
Androgenetic alopecia	Structural hair shaft abnormalities
Alopecia areata	Breakage due to improper use of hair care cosmetics
Drugs	Anagen arrest

clinically and almost always require a 4-mm scalp biopsy for determination of the correct diagnosis.

Androgenetic Alopecia

Most cases of hair loss are due to androgenetic alopecia (AGA). Fifty percent of men by age 50 years and 40% of women by menopause have some degree of AGA. Hair loss is gradual, with miniaturization of genetically programmed hair follicles. Uptake, metabolism, and conversion of testosterone to dihydrotestosterone by 5-alpha-reductase is increased in balding hair follicles. AGA appears different in men as compared with women. In men with AGA, hair loss occurs in the fronto-temporal regions and on the vertex of the scalp, depending on severity. In female AGA patients, it is more diffuse and located centroparietally. The frontal hairline is usually intact in women.

Treatment is either medical or surgical. The only proven medications that are indicated for AGA are topical minoxidil (Rogaine[®]) and oral finasteride (Propecia[®]). Minoxidil's success for cosmetically acceptable regrowth is approximately 10% in men. In women, 50% show minimal regrowth and 13% moderate regrowth. Treatment is lifelong. Seven percent of patients may experience some irritation (burning, itching, redness) from the minoxidil solution. The 5% minoxidil solution has greater efficacy than the 2% minoxidil solution (Rogaine[®]). Finasteride

TABLE 3. Causes of alopecia

Non-scarring	Scarring
Androgenetic alopecia	Discoid lupus erythematosus
Telogen effluvium	Lichen planus
Alopecia areata	Pseudopelade
Tinea capitis	Severe fungal, viral or bacterial infection

TABLE 4. Causes of telogen effluvium

Childbirth
Severe infection
Severe chronic illness
Severe psychological stress
Major surgery
Hypo or hyperthyroidism
Crash diets inadequate protein
Drugs

recently has been shown to have significant efficacy in male AGA. Finasteride reversed hair loss in 66% of men and stabilized hair loss in 83% of men after two years of follow-up. In women, the use of a systemic antiandrogen such as spironolactone (Aldactone®) 50 to 200 mg per day, cyproterone acetate (Androcur®), or flutamide (Eulexin®) may have some benefit in reducing the amount of hair thinning. Finasteride is not indicated in women.

Hair transplantation of permanent hairs from the back and sides of the scalp to balding areas in the front is a successful procedure but usually requires three to four sessions over 2 years to fill in an area with adequate density. The advent of mini- and micrografting has revolutionized hair transplantation into a more natural-looking process, eliminating clumping or tufting. Donor harvesting with strips rather than plugs has made the donor site more cosmetically acceptable. Hair transplantation is useful not only in men but also in women.

Alopecia Areata

Alopecia area (AA) is considered an autoimmune

PRACTICAL MANAGEMENT OF HAIR LOSS

TABLE 5. Treatment of common hair diseases

Androgenetic alopecia	Alopecia areata	Telogen effluvium
Males:	Adults:	Identify and
1. Finasteride 1.0 mg/day	<50% hair loss:	correct triggering
2. Minoxidil 5% solution	1. Intralesional	factor (e.g. thyroid
3. Hair transplantation	corticosteroids	imbalance,
	2. Minoxidil 5%	nutritional
	solution ±	deficiency,
	corticostero	drugs, etc)
	id cream or	Minoxidil 5%
	anthralin	solution for
		chronic telogen
		effluvium
Females:	>50% hair loss	
1. Minoxidil 5% solution	1. Topical	
2. Antiandrogens:	immunothe-	
o spironolactone	rapy with	
o cyproterone acetate	diphencyprone	
o flutamide	2. PUVA	
3. Hair transplantation	3. Minoxidil 5%	
	solution ±	
	corticosteroid	
	cream or	
	anthralin	
	Children:	
	Minoxidil 5%	
	solution ±	
	corticosteroid	
	cream or	
	anthralin	

disease that affects 1% of the population. It usually presents with an oval patch or multiple confluent patches of asymptomatic nonscarring alopecia. Twenty percent of cases may proceed to alopecia totalis, affecting the entire scalp. One percent may go on to alopecia totalis, affecting all body hairs, including eyebrows and eyelashes. The disease frequently resolves spontaneously within 1 year. Recurrences, however, are common. Severe disease has less of a tendency to resolve on its own, especially in children or atopic individuals. Frequently, fingernails may show dystrophic changes such as pitting, ridging, and thinning of the nail plate.

Treatment depends on the extent of the disease, and the age of the patient. For small patchy disease, intralesional corticosteroid is the treatment of choice. Triamcinolone acetonide (Kenalog®) suspension 5-10 mg per mL is injected with a 30-gauge needle directly into the patches with tiny injections of 0.1 mL each spread over affected areas. The total amount should not exceed 10 mg of triamcinolone acetonide per visit. Injections are repeated every 4 to 6 weeks. Other options include topical minoxidil, anthralin, and steroid. For more extensive disease, the use of a contact allergen (diphencyprone) is treatment of choice, in Hair Clinic, Siriraj Hospital showing 40-60% success rates. Other treatment options include psoralens and ultraviolet A radiation (PUVA) and the use of systemic steroids. The use of systemic steroids is controversial, since steroids have a high side effect profile and patients may have to be on the drug for a long time. In Hair Clinic of Siriraj Hospital we use triamcinolone acetonide (Kenalog®) suspension 40 mg per mL intramuscular every one month for three times, some patients were response to the treatment, but when the systemic steroid is discontinued, hair shedding occurs.

Telogen Effluvium

Excessive shedding of normal club hairs can be brought about by a number of stresses: parturition, febrile illness, psychological stress, crash diets, drugs (Table 4). These factors may cause termination of the growing phase (anagen) of the hair follicles and transformation into the resting phase (telogen), causing a telogen fallout 2 to 4 months later. The normal average scalp has 100,000 hairs. Approximately 90% are in anagen, 10% in telogen. Normal average daily hair fallout is 100 hairs. With telogen effluvium, the anagen/telogen ratio may be shifted to 70% anagen, 30% telogen, with average daily shedding of 300 hairs. Treatment depends on determining the cause and correcting it.

Treatment

The treatment are summarized in the Table 5.