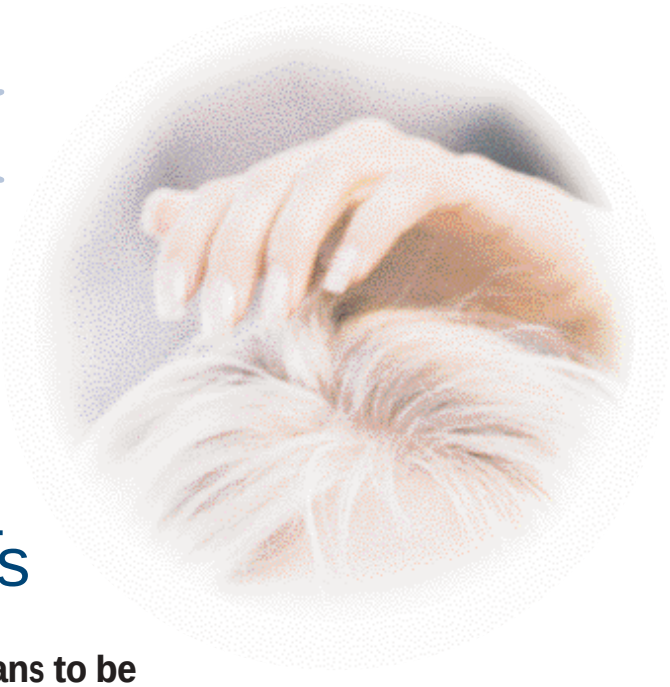
Why Am I Losing My Hair?

A Differential Diagnosis



As hair loss affects a large segment of our population, it is important for family physicians to be aware of the different causes, so patients can access treatment sooner.

By Louis Weatherhead, BSc, MBBS, FRCPC

Hair is a very important secondary sexual characteristic in both men and women, and contributes greatly to the personal psyche. The sheer number of hair cosmetics available attest to this, and thus, hair loss is often quite traumatic to many.

Why do people lose hair? There are many causes, the most common of which are classified as follows:

1. Androgenetic alopecia (AGA);
2. Diffuse alopecia;
3. Alopecia areata (AA);
4. Cicatricial alopecia;
5. Traumatic alopecia; and
6. Miscellaneous causes of alopecia.

This article will attempt to briefly outline these conditions.

Androgenetic Alopecia

This is, perhaps, the most common cause of hair loss occurring in many men, and some women, who carry the genes responsible for baldness. In susceptible people, certain follicles in the fronto-vertical region of the scalp show end-organ sensitivity and possess an enzyme, 5 alpha reductase type II. This enzyme converts testosterone to dihydrotestosterone (DHT), the active component, which acts on the susceptible follicles. This, in turn, causes progressive miniatur-

ization of the hair from normal terminal to vellous hairs, and then loss.[1] Attempts to classify hair loss have resulted in the modified Hamilton Scale for men and the Ludwig Scale for women (Figures 1 and 2). As a rule of thumb, 20% of men in their 20s, 30% of men in their 30s, 40% in their 40s, *etc.*, show evidence of AGA.

Dr. Weatherhead is clinical associate professor, division of dermatology, University of Ottawa, Ottawa, Ontario.

Therapy for AGA

There is no true cure for AGA. Massage, ultra violet light, shampoos and topical applications do not cure AGA.[2] There are, however, successes in restoration by medical and surgical means.

Minoxidil

This medication comes in a 2% and 5% solution. Its exact mechanism of action is unclear, but it is thought that it may act by stimulating the blood supply at the follicular papillary level and by modulating follicular deoxyribonucleic acid (DNA). The effect is unpredictable and seems to show a better response in women. In men, the best response seems to occur in those under 30 with non-extensive male pattern hair loss. The treatment requires a long-term commitment of six months minimum.[1]

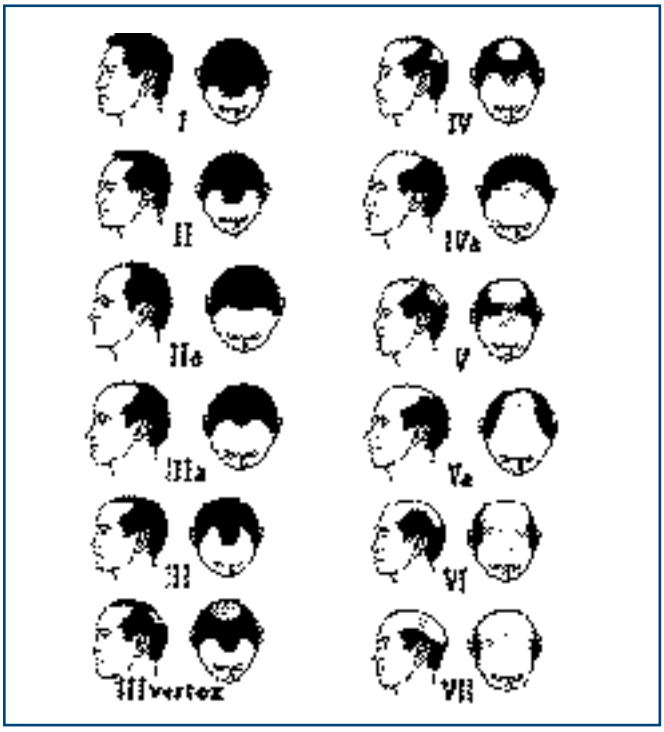


Figure 1. Modified Hamilton Scale for male-patterned androgenetic alopecia.

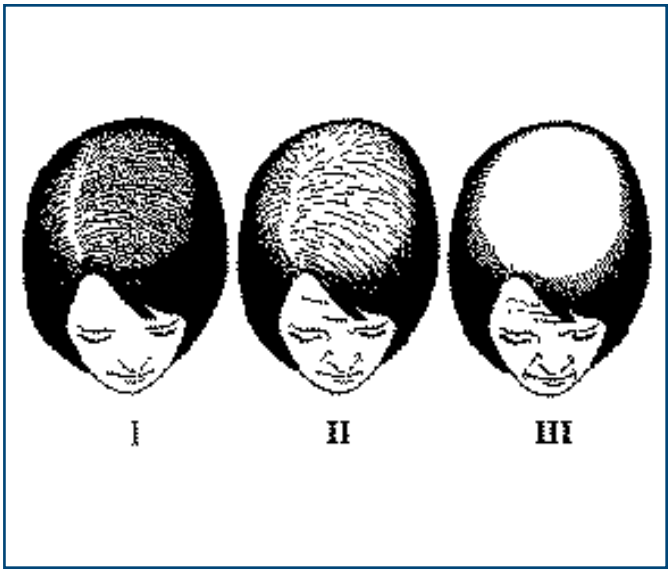


Figure 2. Ludwig scale for female-patterned androgenetic alopecia.

Finasteride

This is primarily indicated for AGA in men of Type I to Type IV pattern loss. Finasteride causes reversible inhibition of Type II 5-alpha reductase enzyme. Eighty-five per cent of people using the medication show reduction or cessation of further hair loss, and 66% of this population show varied amounts of regrowth from mild to marked. A long-term commitment, a minimum of six months, is required. Finasteride is not indicated in women of any age, and is actually contraindicated in women of childbearing years due to the possibility of virilization of the female fetus. Anyone prescribing this medication should be familiar with the product monograph and should counsel their patients as to the potential side-effects.

Hair transplantation

This is a surgical procedure in which hair is taken from the inferior occipital area of the scalp as a donor strip. This strip is then sectioned into follicular unit grafts, which are transplanted into recipient slits in the bald scalp area. As the occipital scalp is not effected by DHT, the transplanted hairs similarly will be unaffected when placed in the recipient area and will grow normally. The final result is dependent on the extent of hair loss and the total number of transplanted hairs.

Also known as spot baldness, alopecia areata may affect up to 1.7% of Canadians by age 50.

Diffuse Alopecia

The most common cause of diffuse alopecia is telogen effluvium.[1,3] In the normal,

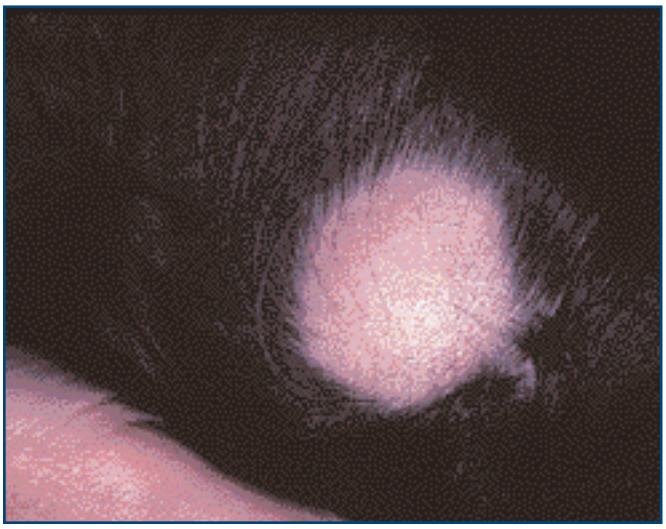


Figure 3. Alopecia areata.

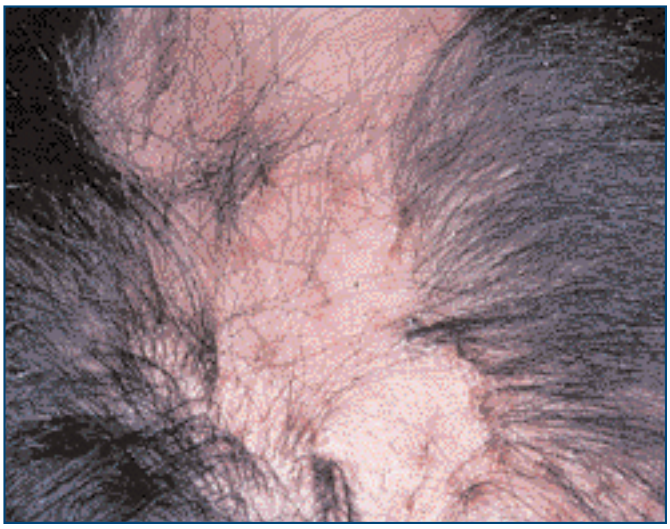


Figure 4. Folliculitis decalvans.

young adult scalp, approximately 85% of follicles are in the anagen (growth) phase of the hair cycle.

Telogen effluvium describes the increased shedding of telogen, the resting phase of the hair cycle, the hair having become a club hair and not growing further). The increased shedding of these hairs follows the acute precipitation of anagen follicles into telogen. This may occur due to multiple stress-related causes, such as:

1. Illness;
2. Post-operative state;
3. Medications;
4. Crash diets;
5. Emotional trauma; and
6. Post-partum state.

The only symptom of telogen effluvium is diffuse shedding of hair, which the patient notices on pillows, brushes, combs and with shampooing. The condition is triphasic. Phase 1 is accelerated hair loss; phase 2 is a plateau phase with no more loss or gain; and phase 3 is a spontaneous resolution phase whereby the amount of daily loss slowly returns to normal, less than 120 hairs lost per day. The total duration may range from six to 18 months. Complete baldness rarely occurs.

Although telogen effluvium is a spontaneously resolving condition, thyroid function testing and serum ferritin levels should be obtained, because thyroid disease and low serum ferritin (value less than 40) may play a role in prolongation of the problem. If these conditions are present, they should be corrected.

Alopecia Areata

Also known as spot baldness, AA is not uncommon and may affect up to 1.7% of Canadians by age 50. The etiology of AA is unknown, although it is thought to be an autoimmune disorder, as helper T-cells are commonly found in the lymphocytic infiltrate that exists around hair bulbs.[4] Factors implicated in the development of AA include:

1. Genetic considerations, as the role of human leukocyte antigen (HLA) class II antigens DQ3, DR4 and DRW11 genes

may increase susceptibility to AA;[5]
2. Atopy may play a role in susceptibility to development of AA; and
3. Emotional stress often is linked to the development of AA.

How these factors influence or trigger the onset of AA is, however, obscure in nature.[5]

Clinically, AA presents as a well circumscribed, totally hairless patch. It is often noticed by chance by a hairdresser, parent or friend, most commonly on the scalp (Figure 3), although it may involve the eyebrows, eyelashes or beard area. Examination at the periphery of the patch may reveal the presence of hairs that taper to the base (known as exclamation point hairs), which suggest ongoing activity.

In susceptible individuals, hair loss may extend to involve the loss of all scalp hair (alopecia totalis) and, in extreme cases, all scalp and body hair (alopecia universalis).

In AA, associated clinical changes may occur. Nail dystrophy may occur, varying from fine pitting to marked ridging, transverse lines, thickening and splitting of the nails.[6] There also have been reports of cataracts occurring with AA.

Treatment of AA is varied. As the condition often resolves spontaneously within a year, patients can be followed. More commonly, topical or intralesional steroids have been found to shorten this clinical course.

In persistent cases, immune modulation therapy using topical diphencyprone (DCP) or squaric acid dibutyl ester (SADBE) have shown good response rates with good regrowth, even in cases of alopecia totalis.[5]

Table 1
CLASSIFICATION OF CICATRICIAL ALOPECIA

1. Trauma

a) Burns—thermal, chemical
b) Radiation therapy/radiation dermatitis
c) Physical injuries

2. Infections

a) Bacterial—follicular decalvans (Figure 4), syphilis, cutaneous tuberculosis
b) Fungal—inflammatory fungal infections (kerion), favus
c) Viral—Herpes zoster

3. Neoplasms

a) Benign—epidermal nevi, cylindroma
b) Malignant—squamous cell carcinoma, angiosarcomas, cutaneous metastases

4. Inflammatory dermatoses

a) Lupus erythematosus
b) Lichen planopilaris (Figure 5)
c) Sarcoidosis
d) Scleroderma/morphea
e) Follicular mucinosis

5. Developmental disorders

a) Aplasia cutis
b) Facial hemiatrophy (Romberg's syndrome)
c) Epidermolysis bullosae

Cicatricial Alopecia (Scarring Alopecia)

This develops as an end-stage condition, following a severe inflammatory process that causes permanent destruction of the hair follicles. It may occur in a localized area or be patchy and scattered throughout the scalp. There are many causes of cicatri-

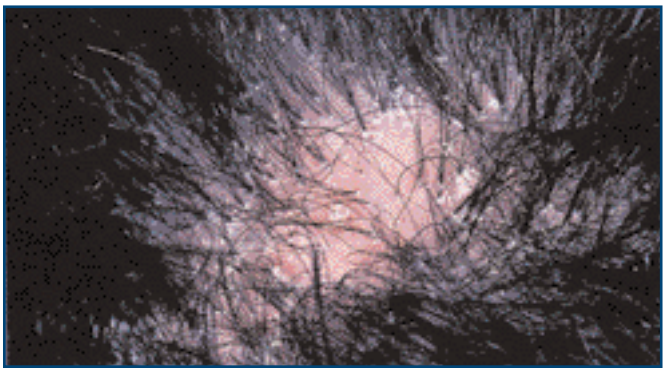


Figure 5. Lichen planopilaris. Photo courtesy of Merck Canada.

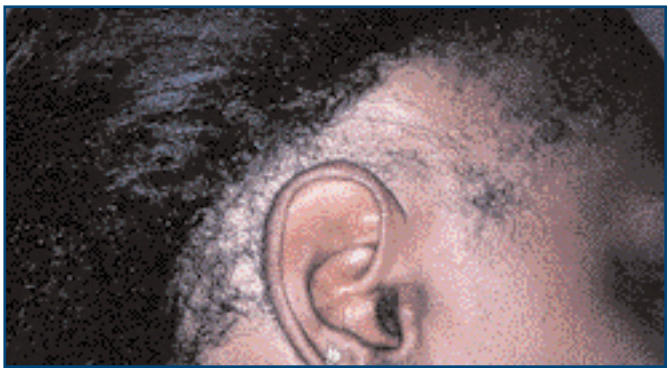


Figure 6. Traction alopecia. Photo courtesy of Merck Canada.

cial alopecia. It would be beyond the scope of this article to go into detail of all of these conditions and, as such, a classification of cicatricial alopecia is presented in Table 1.

Although this classification could be more extensive, the conditions listed represent the more commonly found conditions leading to cicatricial (scarring) alopecia.

Traumatic Alopecia

The most common causes of traumatic alopecia are trichotillomania and traction alopecia.

Tight "pony tail" hairstyles may lead to hair loss at the frontal hair margin.

Trichotillomania

This term describes alopecia produced by an individual who compulsively twists or pulls at their hair. This usually is a habit disorder and may be due to underlying psychologic or emotional factors. It occurs in a 2:1 ratio of females to males. Below six years of age, however, boys outnumber girls in a 3:2 ratio—the peak age of incidence in boys being two to six years. Trichotillomania occurs approximately seven times more frequently in children than in adults.

Clinically, the area of alopecia is mainly in one fronto-parietal area. It presents as an ill-defined patch, which shows hairs broken at varied lengths.

Trichotillomania also may be found in eyelashes, eyebrows, moustache and beard areas.

This habit disorder is usually self-limited, however, if the problem is severe, patients should be managed with psychotherapy.[7]

Traction alopecia (cosmetic alopecia)

This variety of alopecia occurs as a result of prolonged physical stresses imposed on the hair. There are two processes that account for the patterns of hair loss seen. In the first, hair weakened by chemicals may be broken by the application of tension or by friction (cosmetic alopecia). In traction alopecia, prolonged tension produces inflammatory changes in the follicles, leading eventually to scarring.[8]

Clinically, these conditions are seen mainly in the African-American population, as a result of the pulling and chemicals involved in straightening the hair or from tightly braiding the hair *(e.g.*, corn row hairstyles). The hair loss pattern tends to occur at the frontal and temporal hair margin, and also in a triangular area in front of, and above, the ears (Figure 6).

Tight "pony tail" hairstyles also may lead to hair loss at the frontal hair margin, due to the traction produced at the hair margins by the pulling on the hair to form the pony tail.

In the early stages, the changes of traction alopecia are reversible if the traction event is discontinued. Prolongation of the traction, however, will lead to permanent hair loss, with scarring of the affected follicles.

Miscellaneous Causes of Alopecia

Miscellaneous causes of alopecia may have several etiologies outlined by the following classification:

1. Hair shaft abnormalities: There are several hair shaft abnormalities that make the hair susceptible to minor trauma leading to breakage. These may be due to hereditary or acquired causes:
   a. Monilethrix—beaded hair
   b. Trichorhexis nodosa
   c. Trichoschisis
   d. Pili torti
   e. Trichothio dystrophy
2. Anagen effluvium: Loss of hair in anagen phase, usually secondary to the effect of chemotherapy.
3. Alopecia of endocrine origin: Many endocrine syndromes may cause hair loss, such as:
   a. Hypopituitary states
   b. Hypo- and hyperthyroidism
   c. Diabetes mellitus
4. Alopecia of nutritional origin: Hair is exquisitely sensitive to protein deficiency, as the fuel requirements of the follicle are very high. Thus, disorders of hair growth are seen in cases of protein-calorie malnutrition, such as kwashiorkor and marasmus.
5. Congenital disorders: There are several genetic conditions in which alopecia, sparse hair or poor hair growth occur. These conditions include:
   a. Ectodermal dysplasias
   b. Congenital alopecia
   c. Hereditary hypotrichosis
   d. Premature aging syndromes (*e.g.*, progeria)
   e. Netherton's syndrome

Conclusion

As demonstrated in this article, there are many causes of hair loss. The more common causes have been explained whilst the less common have been tabulated.

When a patient presents with a history of hair loss, it is always necessary to do a complete history and physical examination to determine the etiology of the loss and to determine the best therapeutic approach. Dx

Acknowledgment

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