Alopecia: A Practical Approach to Diagnosis and Management

SUMMARY

Hair loss may not be as serious as cancer or heart disease, but when patients lose one of their most treasured possessions, the psychological trauma can be extremely severe. Although the pathogenesis of alopecia may seem complicated, if one understands the basic physiology and the normal hair growth cycle and learns how to perform a methodical examination and investigation, one can easily diagnose and treat most cases of alopecia seen in family practice. (Can Fam Physician 26:410-414, 1980.)

Human Hair Growth Cycle

The human hair growth cycle begins in the hair follicle with a new hair germinating from the hair bulb, progressing to the anagen or active growing phase which lasts approximately 900 days. As the period of growth ends, the lower transient part of the hair follicle degenerates. The hair then enters into the catagen or transitional atrophic phase which lasts about 14-21 days. Finally the atrophic hair goes into the telogen or resting phase which lasts about 90 days. The old resting hair is eventually pushed out of the follicle by the new growing hair or is shed as a result of ordinary wear and tear through washing and combing.

There are approximately 100,000 hair follicles on the average human scalp. About 90% of the hairs are in the anagen phase and ten percent in the telogen phase. The average hair loss for normal individuals is about 50-100 hairs per day. Human hair grows approximately 0.35 mm per day or one cm per month. Hair growth usually follows a cyclical and asynchronous pattern with increases in the summer and reductions in the winter.

Investigation of Hair Loss

When patients with alopecia are seen at the office, it is essential to obtain a detailed history and perform a methodical clinical examination, including Wood's light and microscopic analysis of hair. It is important to determine whether the hair is coming out with the roots attached or whether it is breaking off at some point distal to the roots. It is equally important to know the duration of the hair loss, drug history, mental status, physical development, past medical history, hair style, hair care, cosmetics and family history of hair problems. In the clinical examination, note the hair texture, length, style, pattern and distribution of hair loss. A "pull test" is useful in determining whether the hair is shedding excessively. This test is done by grasping eight to ten closely grouped hairs near the scalp with the thumb, index and middle fingers, and applying traction as the fingers are pulled firmly and slowly away from the scalp. Normally up to two hairs are pulled out by this method. If excessive shedding is present, four to six more hairs are easily and painlessly pulled out. Another useful test is to examine the hair tips against a contrasting background to see if they are tapered or broken. The scalp is assessed for the presence of scaling, inflammation, cradle cap, infection and scarring. Hair elsewhere on the body is examined to determine if there is too much or too little in other sites.

Light microscopic examination of a hair mount frequently provides helpful information. When tinea infection is suspected, besides examining the hair with Wood's light, a few short and
broken hairs are mounted in ten percent KOH solution which dissolves the keratin and allows easy visualization of fungal elements. Balsam, Premount or synthetic resin are used in examining hair shafts because in these media, the hair is seen sharply without surface light scatter.

Other laboratory investigations may be very helpful in excluding associated systemic diseases. These tests should include SMA-12, thyroid and hepatic function tests, serum vitamin A and B-12, folate, mercury, iron, magnesium, TSH, testosterone, 17-keto- and hydroxy steroids, VDRL and FTA-ABS, etc. Occasionally a scalp biopsy may be indicated.

Causes of Hair Loss

Common hair loss disorders may be divided into two major groups: hair coming out by the roots and shaft breakage.

The common causes of hair loss coming out by the roots can be remembered by the acronym STAMP: syphilitic alopecia, telogen effluvium, alopecia areata, male pattern or androgenetic alopecia and pill induced alopecia.

Syphilitic alopecia occurs in the secondary stage of syphilis. It presents primarily as a patchy ‘moth-eaten’ alopecia involving the scalp, eyebrows and beard, although it may also occur as diffuse shedding three to five months after the syphilitic infection. It usually recovers following adequate antiluetic antibiotic therapy.

Telogen effluvium occurs in association with many conditions such as hyperpyrexia, childbirth, severe infections, severe psychological stress, severe chronic illnesses, major surgery, hypothyroidism, crash diets,1 drugs such as heparin, coumarin, triparanol, thiourea, boric acid, indomethacin, carbamazepine, nitrofurantoin, lithium carbonate, asulfidine, etc. Here the hair loss represents increased shedding of the telogen hair. It is transient in nature and reversible in the majority of cases if the associated etiological factors are corrected.

Alopecia areata is characterized by sudden appearance of sharply defined, round or oval patches of complete hair loss. The scalp is commonly involved, but any hair-bearing skin may be affected. A positive family history is found in 20% of cases. The process may be limited to a few patches, or it may progress to alopecia totalis or to alopecia universalis. The clinical course is variable; in general, when the process is limited to two or three patches, the prognosis is good, but when it is extensive, the prognosis for complete recovery is poor. The etiology of alopecia areata is not clearly established, but it is often associated with certain autoimmune disorders2,4 such as Hashimoto’s thyroiditis, vitiligo, atopy, diabetes mellitus, Addison’s disease, pernicious anemia, collagen diseases, etc. Psychosomatic factors may play important roles in alopecia areata, although the etiological relationship remains controversial.5 Therapeutically, adequate explanation, reassurance, avoidance of emotional stress and guidance on proper scalp and hair care are very helpful. Various fluorinated topical steroids used with occlusive dressings or shower cap and intraslesional injections of triamcinolone acetonide or hexacetonide6 are often effective in limited alopecia areata with only a few patches involved.

Male pattern or androgenetic alopecia represents increased shedding of apparently normal telogen hairs. The loss from scalp of telogen hairs of many different diameters and the presence of many short, thin, relatively unpigmented vellus-like hairs are pathognomonic. In males, the hair loss involves primarily the frontocentral and frontoparietal regions of the scalp.7 In females, it manifests as diffuse thinning of scalp hair. There is no successful therapy available although previous generations of physicians have tried various therapeutic agents such as topical or intraslesional steroids, estrogen and testosterone8— with no success. Hair transplantation may be considered in certain individuals. The most practical approach to this problem is a wig.

Birth control pill-induced alopecia9 occurs in some women either as a result of taking or after stopping oral contraceptives. Alopecia occurring while taking birth control pills usually involves those women who are genetically predisposed and are on the progestosterone-dominant type of pills. The alopecia is reversible upon switching over to the estrogen-dominant type of pills such as Enovid, Ovulen and Demulen. Those women with alopecia occurring after stopping the pills usually recover fully two to six months afterward without specific therapy.

The common causes of hair loss due to hair-shaft breakage can be remembered by the acronym STATIC: structural hair-shaft anomalies (trichorhexis nodosa, pili torti, monilethrix, bamboo hair), tinea capitis, anagen arrest, trichotillomania, improper hair care, and cosmetic misuse.

The structural hair-shaft anomalies10 are an important group of disorders which may provide a clue to

Fig. 1 Syphilitic alopecia

Fig. 2 Telogen effluvium

Fig. 3 Alopecia areata
In respiratory infections

**VIBRAMYCIN**

because...efficient tissue penetration floods infection sites with powerful antimicrobial activity.

![Lung tissue](4.10 \mu g/g)

![Tonsillar tissue](1.56 - 4.30 \mu g/g)

![Sinus secretions](0.50 - 7.50 \mu g/mL)

† Substantially greater than levels required to exceed the Minimum Inhibitory Concentrations for most common respiratory pathogens.

widespread pathology such as an inborn error of metabolism or genodermatosis. Because such anomalies have a highly distinctive morphology, the diagnosis is easily made in the office with a microscopic hair-mount examination.

*Trichorrhexis nodosa* is the most common of all hair-shaft anomalies. It occurs primarily in the scalp or in the genitoinguinocrural area. The affected hairs show one or more light colored nodular swellings irregularly spaced along the hair shaft. The broken hairs have a 'brush-border' distal tip, with breakage occurring at nodes. Trichorrhexis nodosa is usually due to mechanical injury to the hair shaft, as from rubbing or twisting. *Trichorrhexis congenita* and *Trichorrhexis nodosa* associated with argininosuccinic-aciduria are uncommon. Treatment consists of identifying and eliminating exogenous mechanical injuries. Associated pruritic skin disorders such as seborrheic dermatitis, neurodermatitis, tinea cruris or candidiasis must be eliminated to prevent mechanical injury from excessive scratching and rubbing. The prognosis is good if patients treat their hair gently and use protein hair conditioners and cream rinses.

*Pili torti* is a hair-shaft defect in which the fragile hair is flattened and twisted on its own axis. This rare and usually hereditary disorder chiefly affects the scalp, but eyebrows, eyelashes, beard, axillary and pubic hair may also be involved. The breakage occurs at the twisted segments, producing partial alopecia. The reflection from the fractured twists gives the hair a glittering or 'spangled' appearance. The hair loss is usually seen at birth or shortly thereafter, but may begin at puberty. Usually one or more siblings and a parent are affected, suggesting variable inheritance. It may be associated with keratosis pilaris, dental abnormalities, dystrophic nails, corneal opacity and cochlear type of sensorineural deafness. It may also be associated with copper deficiency, a part of a clinical syndrome known as Menke's Disease which affects infant males and is characterized by pili torti, seizures, psychomotor retardation, growth failure and death by age four. Treatment of pili torti is nonspecific and is the same as that of damaged hair.

**Fig. 4**

Trichotillomania
from mechanical injury as in trichorrhexis nodosa. In view of its hereditary nature, genetic counseling is advisable.

Monilethrix is a distinctive, rare hair-shaft defect inherited as a simple dominant trait, and named after the resemblance of the affected hair-shaft to a string of beads. In infancy, the hair breaks, leaving the scalp covered with broken hair 0.5-2.5 cm long. A hair-mount shows the distinctive 'nodes' which have a diameter of normal hair and are medullated, alternating rhythmically with the 'internodes', which are constricted and non-medullated, and are the sites of fractures. Keratosis pilaris is always associated with monilethrix and usually occurs on the upper back and shoulders. Occasionally, there are also other ectodermal defects such as brittle nails, decreased visual fields, juvenile cataracts and dental lesions. The etiology is unknown. There is no specific treatment, but the condition may improve at puberty, with pregnancy or with oral contraceptives.

Bamboo hair or trichorrhexis invaginata is a distinctive hair-shaft defect caused by intussusception of the hair shaft at the zone of beginning keratinization. The bamboo hair has nodose ball-and-socket deformities, with the socket forming the proximal and the ball part forming the distal portion of the node along the hair shaft. Bamboo hair is associated with Netherton's Syndrome, a rare ectodermal genodermatosis, which combines ichthyosiform skin changes, bamboo hair, and...
frequently the atopic state. The majority of patients so affected are female. The bamboo hair may be present not only on the scalp, but also on the eyebrows, eyelashes and, rarely, in other hairy sites. Bamboo hairs may disappear within a few years. Present evidence suggests an autosomal recessive inheritance. Potent topical steroid creams help the skin lesions temporarily, as does the Goeckerman regime.

*Timea capitis* is a fungus infection which mainly affects children. Hair infected with Microsporum show a bluish green fluorescence under the Wood's light, but Trichophyton infections do not fluoresce. There are two clinical types:

1. The grey patch type, manifested by non-inflammatory patches of fine, grey scaling with short broken hairs.
2. The inflammatory type is manifested by one or few raised, inflammatory patches covered with short broken hairs, scales, crusts and vesicles. Kerion and cervical adenitis are common. Systemic fulvicin U/F and topical fungicides such as miconazole, clotrimazole or tolnaftate are therapeutically very effective. It is important to continue treatment for at least four to six weeks; the criteria for cure are two negative cultures and a negative Wood's light examination.

Anagen arrest or anagen defluvium is hair loss caused by temporary arrest of mitotic activity in the matrix region of growing or anagen hair. This results in constriction of the hair shaft and subsequent hair breakage. The commonest causes are cancer therapeutic drugs such as Methotrexate, 6-mercaptopurine, 5-fluorouracil, nitrogen mustard, leukeran, cytoxan, thiopeta, actinomycin-D, vinblastine, etc. The alopecia begins about one to three weeks after systemic administration of these drugs is started and over 80% of the hairs are affected. Anagen arrest can also be caused by heavy metals such as lead and arsenic, and other drugs including trimethadione, thalium, heparin, coumarin, triparanol, thiourea, carbamazepine and lithium. Treatment is usually unnecessary because the hair regrows when the offending drug is discontinued.

Trichotillomania is hair-pulling or hair-pulling tic is the unconscious pulling out of hair by the patient and may be patterned or diffuse. Scalp hair, and occasionally eyelashes and eyebrows, are the areas attacked. Trichotillomania can be associated with trichocryptomania (hiding of hairs so removed) or trichophagy (swallowing of the hairs). If trichotillomania is stopped soon enough, no permanent hair loss occurs. However, prolonged trichotillomania may result in permanent alopecia. In children under ten years of age, the phenomenon is usually simply a nervous tic of pulling or twisting the hair about the fingers. Such children tend to be nervous and under emotional stress. Generally, bringing the habit to the child's attention and reassuring him or her is enough to stop the tic, especially if some of the aggravating stress is removed. In older children, especially girls, trichotillomania is associated with neurosis and requires psychiatric help.

Improper hair care and certain hair styles may cause traction alopecia if excessive tension is applied to the hair for a long period of time. Tight ponytails and braids are classic examples of such hair styles. Relieving the tension on the hair reverses the problem in most cases, but occasionally when traction has been prolonged, alopecia may be permanent.

Cosmetic misuse often leads to hair breakage and alopecia. Generally, there is very little damage from normal hair dyeing, bleaching, waving and straightening. However, some patients will experience some hair loss from improper use of hair cosmetics. The damage is usually caused by improper techniques such as too much tension during waving, waving solutions left on too long and improperly neutralized bleach applied to already bleached hair, or waving and dyeing of hair on the same day. The patients should be advised to stop the cosmetic procedures until the hair is fully regrown and to use mild protein shampoo with weekly application of protein hair conditioners and cream rinses.

**Conclusion**

If one understands the basic physiology of hair and the normal hair growth cycle, remembers STAMP and STATIC as the various causes of two major groups of hair loss, learns how to take a detailed case history and perform a methodical examination and investigation, one can establish an accurate diagnosis and then provide patients with appropriate therapy. For those patients with correctable alopecia, you become an instant hero. For those difficult and untreatable cases of hair loss, your concern, interest, sympathetic attitude, reassurance and guidance concerning proper hair care can be extremely helpful.

**References**


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