Clinical Algorithms

Hair loss

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In the hair of the scalp slowing of growth, structural narrowing, and diminishing density are inevitable accompaniments of aging. Medical opinion is sought because of the accelerated appearance of bald areas or of diffuse hair loss manifested by obvious thinning or an increased daily fall.

In making a clinical assessment of hair loss, the hair, scalp, pattern of loss, nails, and teeth need to be examined and the possibility of underlying systemic disease considered. The condition of the hair can be assessed by holding a few stretched strands of hair with the thumb and forefinger of one hand and sliding the other thumb and forefinger towards the roots. If the hair resists this movement and ruckles up the outer cuticle has been damaged. It is also helpful to assess the ease of hair removal on traction; as the technique requires a firm tug applied to a group of hairs pinched between thumb and forefinger it often increases the dismay of patients concerned about their lack of hair. Rarely are more than three hairs extracted by this method, and the loss of six or more must be considered abnormal.

It is diagnostically helpful to consider patchy or localised loss separately from generalised loss. In either case the algorithm then requires decisions on whether the hair loss is due to hair fall or hair breakage and on the normality or otherwise of the scalp.

Generalised loss

The average head of hair contains 100 000 hairs, half of which may be lost before thinning becomes apparent. In the early stages of diffuse thinning the loss may therefore be difficult to confirm and even if the clinician is unconvinced about the presence of disease he should arrange a further review before making a definite conclusion. It is often useful to ask what drew the patient's attention to the loss. Was it, for example, the number of hairs on the pillow in the morning, an abnormal loss on washing, or the increasing numbers of hairs removed when brushing or combing? In assessing the response the clinician should remember that many patients are aware that a loss of 100 hairs a day is considered normal and may exaggerate the loss to be taken seriously. Although some obsessional people collect all discarded hair and live in terror of incipient balding despite having a normal daily hair loss, the only psychiatric condition commonly underlying a spurious complaint of alopecia is depression, and this diagnosis should be considered whenever a patient complains of diffuse loss for which there is little supporting evidence.

When generalised loss is present it is useful to assess the ratio of anagen (growing) hair to that of the telogen hair (resting hair with club shaped root). As with the tug technique, this should be assessed on unwashed hair and 50 or more hairs must be extracted for the assessed ratio to be considered representative. In the normal scalp

telogen hair forms about 10% of that extracted. If this rises significantly telogen effluvium is present. For some conditions microscopy is an essential part of examining the hair, but this, together with scalp biopsy and more advanced investigations, is best left to the specialist.

Telogen effluvium is one of the more dramatic yet often encountered hair fall phenomena. It may be precipitated by a wide variety of stressful events, many of which are associated with fever, by hormonal changes, or by the administration of some drugs. Changes in the hair cycle are induced, with hairs proceeding from anagen into telogen phase faster than normal. Some three to four months after this has occurred the increased loss will be noted as the telogen hair is shed. Only when an underlying disease such as thyroid disorder or iron deficiency anaemia exists is any treatment required as the normal hair cycle will eventually re-establish itself. Anagen effluvium, on the other hand, is the result of a severe sudden onslaught on the growing hair, the only two common causes being cytotoxic treatment and radiotherapy. Hair growth is abruptly stopped, and the hair is either shed immediately or breaks due to weakness induced in the hair shaft. The anagen: telogen ratio within the affected hair remains normal. Clearly, normal hair regrowth can be expected when the treatment is discontinued.

If hair breakage is widespread it is usually associated with fragility of the hair shaft. The increased ease of breakage can usually be confirmed at examination. Many chemicals and strong sunlight disrupt the disulphide bonds on which hair strength depends and lead to breakage. Structural weakness may also arise as part of congenital syndromes, which may induce diverse structural abnormalities; a few of the more common conditions which can be identified with the naked eye or hand lens include: short beaded hair—monilethrix; twisted hair—pili torti and Menkes' syndrome; banded hair—low sulphur hair syndrome; and local twisted hair—kinky hair syndrome. Trichorrhexis nodosa—hair of irregular length with multiple shaft fractures—can be seen in association with argininosuccinic aciduria and other congenital syndromes but is more commonly the result of trauma. It is often associated with split ends (trichoptilosis).

Ectodermal dysplasias (skin, nail, and teeth abnormalities) and other congenital and premature aging syndromes are often associated

TABLE 1—Drugs commonly causing alopecia

Drug	Condition
Cytotoxic agents	Anagen effluvium (telogen: anagen ratio normal in affected hair)
Antithyroid drugs	Hair dry and brittle; patient usually euthyroid
Anticoagulants	Heparin and coumarin can induce telogen effluvium; axillary and pubic hair may be affected
Oral contraceptives	Probably due to progesterone component
Withdrawal of oral contraceptives	Condition akin to telogen effluvium
Phenytoin	Often associated with hirsutism
Retinoids, Atromid, and hypervitaminosis A	All interfere with keratinisation
Androgens	Androgenic alopecia: several drugs used to treat menopausal symptoms contain androgens, such as Mixogen

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Hair loss 1

Hair loss 2

TABLE 11—Active inflammatory changes in scalp

Abnormality	Diagnosis	Associated features	Treatment
Plaques	Erysipelas	Acute, hot, red, usually raised temperature	Antibiotics
	Follicular mucinosis	May present as perifollicular papules or scaling erythema	Exclude associated neoplasia (20%); no known treatment; most cases will resolve in two years
Nodules	Infective	Fluctuant, usually pyogenic, rarely ringworm	Drainage and antibiotics
	Rare causes	Wide variety of infective, inflammatory, or neoplastic infiltrations	Biopsy usually required; treatment in the light of findings
Vesicle/bulla	Herpes zoster	Acute onset dermatome distribution	Systemic steroids, antibacterial dressings
	Cicatricial pemphigoid	Often associated with mucosal changes	Steroids
	Dermatitis herpetiformis	Usually typical skin lesions on buttocks, shoulders, and elbows	Dapsone and gluten free diet
	Insect bites	Usually confirmed by history	No treatment necessary unless infection supervenes
Papules	Lichen planus	In acute stages violaceous papules or perifollicular scale may be present. Do not forget to look in mouth	Steroids topically or intralesionally
	Discoid lupus erythematosus	Acute; erythema and perifollicular adherent scale (plugging), subsequently telangiectasia and scarring	Steroids, chloroquine derivatives, local cautery, thalidomide as indicated

When active inflammatory changes in scalp are present, always examine the skin for diagnostic changes elsewhere. Biopsy is necessary in many cases to secure a diagnosis.

with hypotrichosis. Sometimes diagnostic hair shaft abnormalities exist—for example, Netherton's (bamboo) hairs. They are an extremely diverse group of disorders which require specialist evaluation and investigation.

In trichotillomania the hair is usually normal. Often the patient is strangely unconcerned by the loss. In children, the parents are often aware of their child's habit of pulling at its hair but may resist the suggestion that trauma is the cause of the loss, and in these cases it is worth asking them to look for the pulled hair hidden among the child's playthings. In trichotillomania there are two often observed patterns to the hair loss. Firstly, in a right handed person, particularly a child, the loss is often from the left parietal region and in a left handed person from the right. Secondly, hair in the occiput is rarely damaged, possibly because of the greater sensitivity to pain in this area.

A common cause of hair fall seen in dermatology clinics is drug induced loss. The common drugs are included in table I, though others may also rarely cause hair loss.

Patchy loss

In assessing localised loss the clinicians should consider first alopecia areata, in which breakage and hair fall coexist. The condition accounts for some 2% of all new dermatological referrals. The activity can best be assessed by establishing the extent to which hair is lost by tugging at the advancing edge of the patch. If the fall is severe exclamation mark hairs (short stubby hairs) should be identifiable. In less active cases diagnosis may be difficult, but under these circumstances loss is rarely rapid and if no other cause suggests itself review can be arranged and further examination undertaken. The commonest type of localised loss in men is undoubtedly androgenic (male type) balding, but this pattern of baldness is so widespread that medical attention is not sought unless it advances rapidly or occurs in children or young adults. There is usually a family history of androgenic baldness, and appreciable loss is often present in the late teens and early 20s. Thinning, usually more of the diffuse vertex type rather than the bitemporal and frontovertical loss of the man, also occurs in postmenopausal women. Again this type of baldness often runs in families, but the common form of constitutional thinning, while perhaps reflecting to some extent androgenic loss, arises primarily from the slowing of hair growth of involutional alopecia. In assessing androgenic loss narrowing (miniaturisation) of the hair can usually be confirmed by rolling the hair between the thumb and forefinger and comparing it with a sample selected from an unaffected area in the parietal region or occiput.

The androgenic pattern occurring in young women may be drug induced, but if drugs have been excluded further investigations are needed to exclude virilisation, and assessment of hormonal function is commonly required (usually estimations of sex hormone binding globulin, testosterone, dehydroepiandrosterone sulphate, and androstenedione).

A diffuse loss may reveal a pre-existing localised loss, so if, for example, alopecia with a typical androgenic pattern appears quickly or in an unusual age group it is important to consider not only hormonal causes but also the possibility of generalised loss producing a misleading appearance.

Cicatricial or scarring alopecias are a heterogeneous group of disorders in which hair loss is secondary to follicular damage. They are diagnosed by the lack of or damage to hair follicles, as only in the later stages of some conditions does obvious scarring develop, which may be associated with telangiectasia and atrophy. Sometimes the condition appears in isolated patches without any evidence of preceding inflammatory disease. Obviously, diagnosis may be aided by gleaming from the history the time course of the condition or establishing any precipitating or aggravating factors. The presence of coexisting skin disease may be helpful. In such a diverse group of disorders, many of which are clinically indistinguishable in their later stages and in which a clinical diagnosis may be impossible, all known causes must be considered and biopsy and specialist evaluation are usually required.

Once scarring is established treatment is rarely, if ever, successful, and, although grafting of some localised areas can be undertaken in cases where progression is not expected, in most cases treatment will be the supply of a hair piece. Although the progression of some scarring alopecias may be slowed or halted with systemic treatment such as retinoids, penicillamine, or steroids, it is unwise to use these potentially toxic compounds for scalp disease alone as the process can be successfully disguised without risking the side effects of potent therapy.

References

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How common is rubber allergy?

Many chemicals may be added to rubber during processing. Of the several groups of compounds used, the accelerators and antioxidants incorporated into the rubber are the principal causes of allergic contact dermatitis. The rubber hydrocarbon itself does not cause allergic contact dermatitis, though there are a few reported cases of allergic contact urticaria caused by gloves made from natural latex. Everyone has daily contact with rubber, be it in household gloves, shoe soles, car components, or elasticated garments. As a rule, the accelerator in soles is mercaptobenzothiazole, in gloves a thiuram, and in heavy duty rubbers antioxidants based on derivatives of paraphenylenediamine. All patients with eczema who are investigated by patch testing are routinely tested with a series of rubber chemicals known to cause allergic contact dermatitis. Up to 10% of patients with eczema may be allergic to one or more of the 16 chemicals in the standard series of allergens. Their sensitivity may be of current or historical importance. The prevalence of rubber sensitivity in the general population is not known.—IAN R WHITE, consultant dermatologist, London.