Hair loss in hospital medicine: a practical guide

Alopecia may indicate underlying systemic disease and is associated with significant impairment of quality of life. A thorough history and examination, including specialist techniques, can give vital clues to the aetiology. This article provides an overview of the common and important hair loss disorders for the busy clinician.

Hair loss disorders are important to recognize as they may signify serious underlying disease, but also commonly result in significant impairment of quality of life and marked psychological morbidity. This psychological impact was highlighted in a review of quantitative and qualitative studies of alopecia that concluded that hair loss can cause intense emotional suffering and may ultimately lead to personal, social and work-related problems. For example, 40% of women had marital problems as a consequence of their hair loss and 63% experience career-related issues (Hunt and McHale, 2005).

This article will help clinicians recognize and deal with common hair disorders encountered during the acute admission or outpatient clinic visit.

History

As with all aspects of medicine, diagnosing hair loss begins with a careful history, including duration and distribution of the current episode and any previous episodes (Olsen et al, 2004). Important features to ascertain include the onset, course and ongoing progression of the hair loss and whether increased hair shedding (hair fall on brush, pillow or with washing) or gradual thinning is experienced. Associated symptoms such as itch, pain, flaking or weeping from the scalp, loss of body hair or nail changes should be explored.

Excess facial hair, acne, seborrhoea and obesity in women may signify hormone dysregulation and should prompt a detailed review of the menstrual history (Blume-Peytavi et al, 2010). Drug history, including hormonal therapy, as well as a family history of alopecia or autoimmune disease may also be helpful in determining the final diagnosis.

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Clinical examination

The scalp should be carefully examined to determine the distribution of the alopecia (e.g. diffuse, 'patterned' or localized). Further, the scalp skin should be inspected for scarring and signs of inflammation such as erythema, scale, follicular plugging or pustules. Nails may show signs of pitting in alopecia areata, or longitudinal ridging in lichen planus. Body and facial hair may have abnormal distribution or density, as may eyelashes and eyebrows. Signs of thyroid disease and the pallor of anaemia may be present.

Specialist examination techniques also include the 'hair part width' (parting the hair in the mid-line to provide an indication of hair density), the 'hair pull test' (50–60 hairs firmly pulled away from the scalp to examine the number of hairs shed) and dermatoscopy (Hillman and Blume-Peytavi, 2009). Dermatoscopy can be performed at the bedside by an experienced individual using a hand-held, polarized light source with magnification. Features of specific dermatoses may become apparent, such as the 'yellow dots' and exclamation-mark hairs of alopecia areata, the broken hairs of trichotillomania and the loss of follicular ostia seen in cicatricial alopecias.

Investigations

Investigations need only be performed in selected cases but may include blood tests, microbiology and mycology cultures and/or scalp biopsy. Scalp biopsy is indicated in undiagnosed, non-scarring alopecia and all cases of scarring alopecia. Two 4 mm punch biopsies are taken from the scalp, one processed for horizontal sectioning and one for vertical sectioning. In conditions of scarring alopecia, biopsies should be taken from the area of active inflammation, usually at the edge of the alopecia, and not from the central scarred area as little additional information can be gleaned other than to confirm scarring. In cases of suspected cutaneous lupus erythematosus, direct immunofluorescence of the skin may demonstrate a 'lupus band' which may aid diagnosis.

Clinical photography is useful to document changes in severity and distribution of hair loss, although serial photographs must be performed under standardized conditions (i.e. lighting, distance) to permit realistic comparison.

Causes of alopecia

Although not an exhaustive list of all causes of alopecia, this article gives an overview of commonly encountered diagnoses and provides a pragmatic evidence-based approach to managing each condition. *Table 1* summarizes the conditions and their corresponding features.

Telogen effluvium

Scalp hair is continuously undergoing periods of growth, resting and shedding, termed 'the hair cycle'. This process is occurring independently from one hair to the next and is composed of three main stages: anagen, catagen and telogen. The majority of hairs (>85%) are in anagen, the active growth phase, the duration of which determines the final length of the hair. Catagen is a short transition phase from anagen to telogen lasting 2 weeks where the hair follicle stops growing and involutes. Telogen is the resting stage when follicles wait to be shed (mean duration = 3 months) before being replaced by a new growing anagen hair. Telogen effluvium occurs when synchronicity of the hair cycle results in increased numbers of hairs entering the telogen phase at the same time, with greater numbers of hair being shed a few months later.

Characteristically telogen effluvium presents with increased shedding of hairs, described as 'falling out by the roots', associated with variable degrees of visible hair thinning. A triggering event usually precedes the alopecia by 1–6 months. Common triggers include hormonal changes, such as childbirth or hormonal contraceptives, medication, surgery or major illness (*Table 2*). On examination, the scalp itself is normal without

Table 1. Common causes of alopecia: clinical findings

Diagnosis	History	Inspection	Special examination
Telogen effluvium	Trigger event, increased shedding or 'thinning', asymptomatic scalp	Diffuse frontal hair thinning, no scarring or inflammation, visible regrowth	Pull test positive (telogen) Dermoscopy: short regrowing hairs, empty ostia
Anagen effluvium	Widespread, sudden, recent chemotherapy or medication	Diffuse alopecia, no scarring or inflammation, loss of body hair	Pull test positive: anagen or broken hairs Dermoscopy: empty ostia
Alopecia areata	Patches or diffuse hair loss, sudden, asymptomatic or itch, nail changes (10%), history of auto-immunity	Circular patches, involved hair line, or diffuse. No scarring, smooth scalp, minimal erythema, loss of body hair	Pull test positive (acute phase: anagen) Dermoscopy: exclamation mark hairs, yellow or black dots
Pattern hair loss (female and male)	'Thinning', slow, increased fall of hair (early), family history possible, scalp asymptomatic	Frontal, vertex or diffuse thinning. No scarring or inflammation. No involvement of body hair. Widened hair parting	Pull test positive (early), negative (late) Dermoscopy: miniaturized follicles, hair diameter variability
Lichen planopilaris	Mainly females, vertex or frontal, itchy or sore, progressive or gradual onset. Rash at other sites	Patches of erythema, scale and scarring or alopecia in frontal band and loss of eyebrows or body hair	Pull test: anagen hairs (active phase) Dermoscopy: loss of ostia, erythema, white patches, peripilar casts (keratin)
Chronic cutaneous lupus erythematosus	Localized centrally, worse after sun. Itchy, painful, scaly with rash at other sites	Vertex or central scalp, scarring, erythematous, scaly, follicular plugging, pigmentation changes	Pull test: anagen hairs (active phase) Dermoscopy: follicular plugging, loss of ostia, red dots, white patches
Folliculitis decalvans	Mainly males, painful, localized, weeping	Any site, most commonly vertex, thickened, boggy scarring, pustules and scale. 'Tufting'	Pull test: anagen hairs (active phase) Dermoscopy: tufting, pustules or scale, loss of ostia
Dissecting cellulitis of the scalp	Mainly males, painful, localized, discharging. History of acne or hidradenitis suppurativa	Multiple fluctuant nodules and sinus tracts	Pull test: anagen hairs (active phase), telogen hairs (scarring). Dermoscopy: nodules, yellow or black dots
Acne keloidalis nuchae	Mainly young, Afro-Caribbean heritage, males, 'lumps' over scalp, worse when shaving hair	Occipital, skin-coloured, firm, nodules and keloid-like plaques and pustules	Dermoscopy: as folliculitis decalvans
Tinea capitis	Children, also adults, localized, 'Dry skin'	Usually vertex, scarring in later stages, scaly and broken hairs. Boggy, pustular, scaly if kerion	Dermoscopy: broken or comma hairs, scale Wood's light: possible fluorescence
Alopecia syphilitica	Gradual, diffuse or patchy alopecia with mucosal symptoms, ulceration or malaise	Diffuse, moth-eaten appearance, non-scarring, occiput and vertex, with lymphadenopathy	Dermoscopy: preserved ostia, reduced hair count
Traction alopecia	Usually female, Afro-Caribbean heritage, tight hair styles or straightens hair	Localized alopecia at sites of tension or hair part, non-scarring (early) or scarring (late). Inflammation rare	Pull test: negative Dermoscopy: broken hairs, and hairs in active traction
Trichotillomania	Feeling of tension, may report removing hair, possible history of obsessive compulsive disorder	Short, broken hairs in patches, spares anterior hairline, scalp normal, but 'bristly'	Pull test: negative Dermoscopy: broken hairs, absence of exclamation mark hairs

* dermoscopic features from Miteva and Tosti (2012)

evidence of inflammation or scarring. Hair loss is diffuse and generalized although hair thinning can give the illusion of a frontal preponderance. The hair pull test is positive (>10% shedding) when active, with increased numbers of telogen hairs being easily removed. On recovery, there may be visible signs of hair regrowth with short but morphologically normal hairs present throughout the scalp.

The condition is self-limiting and spontaneous regrowth will occur if all potential causes are investigated and treated (*Table 2*). In a minority of cases, shedding may persist beyond 6 months duration and become chronic telogen effluvium. The main differential diagnoses for telogen effluvium include androgenetic alopecia and the diffuse variant of alopecia areata.

Anagen effluvium is rarely confused with telogen effluvium as it affects hairs in the anagen growth phase, and as such the hair loss is much more widespread. Anagen hairs stop growing and break off resulting in almost universal hair loss. Anagen effluvium is typically associated with the use of drugs, such as chemotherapy agents. In some chemotherapy regimens cold caps are worn to prevent hair loss. Psychological support and wig provision are the mainstay of treatment, but 2% topical minoxidil lotion significantly shortened the duration of baldness after breast cancer chemotherapy in one study (Duvic et al, 1996).

Table 2. Recognized triggers for telogen effluvium

No trigger identified (approx one third)	
Hormonal	Pregnancy
	Childbirth
	Miscarriage
	Hormonal contraceptives
Medications	Oral retinoids
	Beta blockers
	Non-steroidal anti-inflammatory drugs
	Antidepressants
	Anticoagulants
Major surgery	
Acute febrile illness	
Chronic disease	Thyroid disease
	Chronic kidney disease
	Liver disease
	Connective tissue disease
	Malignancy
	HIV
Malnutrition	Iron deficiency
	Dieting
	Vitamin D deficiency
	Low protein diet
Psychological stress	
Heavy metal poisoning	Arsenic
	Thallium
Inflammatory scalp disorders	
Seasonal hair moult (spring and autumn)	

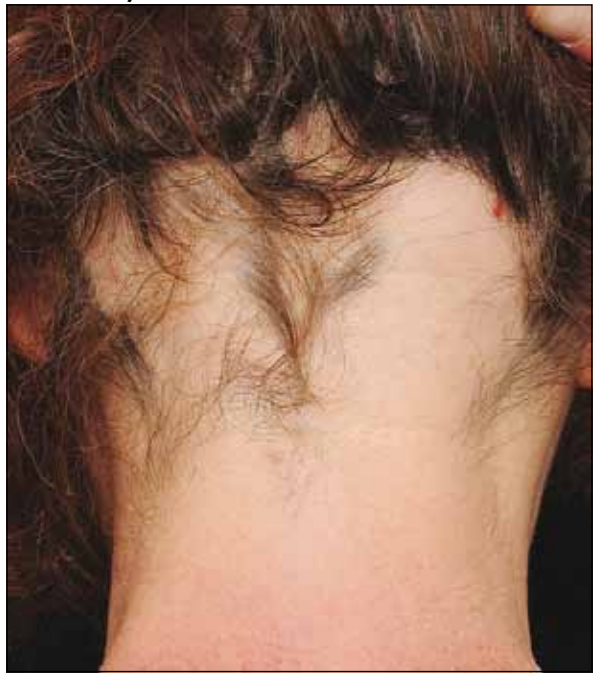
Triggers occur 1–6 months before onset of hair loss. Unpublished data, M Harries, 2012

Alopecia areata

Alopecia areata is an autoimmune disease of the hair follicle which commonly presents with discrete circular patches of hair loss. The condition can involve the hairline in isolation (ophiasis) (*Figure 1*), have a more diffuse pattern of hair loss (diffuse alopecia areata) or result in complete loss of all scalp hair (alopecia totalis) or all scalp and body hair (alopecia universalis). Although the scalp is the most common site, alopecia areata may affect any hair-bearing skin and can occur suddenly or may relapse and remit over time, sometimes with a history of a triggering event. There is no visible inflammation or scarring present, although mild erythema may be seen. Examination of the scalp with magnification may identify exclamation mark hairs (tapered, broken hairs pathognonomic of alopecia areata) (Messenger et al, 2012). Dermatoscopy may reveal 'yellow dots' (representing sebum or keratin plugs) and 'black dots' (i.e. broken hair shafts) and exclamation mark hairs (Miteva and Tosti, 2012). Nail dystrophy may be present in up to 10% of patients (*Figure 2*) (Harries et al, 2010).

Although alopecia areata may co-exist with other autoimmune conditions, routine screening is not neces-

Figure 1. Alopecia areata in a woman with ophiasis (involvement of the hairline).



sary because of the low yield of investigations in asymptomatic patients (Messenger et al, 2012). Investigations are rarely required but exclusion of other causes of alopecia may require fungal culture, antinuclear antibody, venereal diseases research laboratory (VDRL) serology and skin biopsy. Serum ferritin levels may identify iron deficiency but there is no conclusive evidence that iron replacement improves hair growth in such patients.

Alopecia areata demonstrates a spectrum of severity and those with patchy scalp involvement (<50%) have a high chance of spontaneous regrowth, whereas in those with extensive alopecia areata (i.e. alopecia totalis or alopecia universalis), fewer than 10% will fully recover (MacDonald Hull et al, 2003) First-line treatment for alopecia areata may therefore include observation only or potent or ultra-potent topical corticosteroids as a 3–6-month trial. Other available treatment modalities in secondary dermatological care include intra-lesional corticosteroid injection for small areas of alopecia (i.e. <50%), contact immunotherapy with diphencyprone for larger surface areas (i.e. >50%), and other interventions with less robust evidence base include oral steroids, minoxidil and psoralen plus ultraviolet A (Messenger et al, 2012). The Cochrane review of interventions for alopecia areata concluded that there have been few treatments that have been well evaluated in clinical trials and further research is required to provide conclusive evidence (Delamere et al, 2008), although important half-head studies showing clear treatment responses were excluded from this review (Harries et al, 2010).

Female pattern hair loss

Female pattern hair loss, otherwise known as androgenetic alopecia in women, is a slow, progressive, non-scarring alopecia characterized by miniaturization of the hair follicle. The presentation of female pattern hair loss is usually distinct from that seen in male androgenetic alopecia (i.e. fronto-temporal and crown involvement). Female pattern hair loss displays three main patterns:

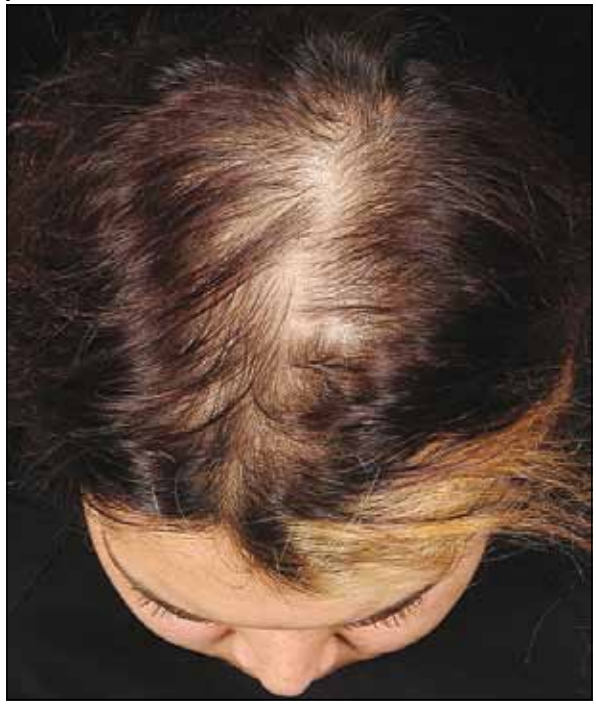
1. Diffuse thinning of the frontal and mid areas of the scalp with preservation of the frontal hairline
2. Widening of the midline hair part (*Figure 3*)
3. Thinning with bi-temporal recession (Blume-Peytavi et al, 2010).

Figure 2. Nail pitting in alopecia areata.

In all forms the occipital hair density is usually well preserved. The prevalence of female pattern hair loss increases with age, with two peaks in incidence at puberty and post-menopause (Blumeyer et al, 2011). A family history of androgenetic alopecia may be reported in up to 54% of male and of 21% female first-degree relatives (Smith and Wells, 1964).

Diagnosis of female pattern hair loss is based on clinical appearance and pattern of hair loss. Close inspection will reveal miniaturized hairs in the described distribution but otherwise the scalp should appear normal without inflammation or scarring. Interventions for female pattern hair loss have been the subject of a recent Cochrane review. Van Zuuren et al (2012) concluded that of all potential interventions studied, topical minoxidil was the only treatment that had been proven to be efficacious: 2% topical minoxidil lotion used twice daily and 5% minoxidil used once daily did not show any difference in physician-rated outcomes but some improvement in patient-reported outcomes with the higher strength, although 5% minoxidil was associated with a higher rate of adverse effects, including facial hypertrichosis and irritant dermatitis. The majority of included studies had relatively short follow-up periods of 6–12 months and so conclusions could not be drawn as to the sustainability of effects seen. Evidence-based guidelines published by Blumeyer et al (2011) suggest that 2% minoxidil should be applied for 6 months before assessing response and that patients should be counselled as to the increase in shedding of telogen hairs within the first 2 weeks of use, as minoxidil stimulates follicles to move into anagen. Treatment must be continued indefinitely to maintain efficacy.

Figure 3. Widening of the hair part – a clinical sign of female pattern hair loss.



The guidelines also failed to find conclusive evidence of the effect of finasteride (a 5-alpha reductase inhibitor licensed for male androgenetic alopecia), cyproterone acetate (often prescribed as Dianette), and spironalactone (100–200 mg per day). However, large-scale trials in those with and without hormone dysregulation are still required to determine their role (Blumeyer et al, 2011).

A subset of women with female pattern hair loss will also show symptoms and signs of hormone dysregulation and these patients must be identified. Affected women may have menstrual cycle abnormalities, history of infertility, or signs of hyperandrogenism, such as facial hair growth, acne and seborrhoea. Documentation of the use of hormonal contraception or medications with antithyroid action is essential (Blume-Peytavi et al, 2010). Underlying endocrine pathologies may include polycystic ovarian syndrome but also rarely androgen-secreting tumours, congenital adrenal hyperplasia and Cushing's syndrome.

Consensus opinion from Blume-Peytavi et al (2010) suggests investigation of this sub-group by analysing the free androgen index, calculated from free serum testosterone and sex hormone-binding globulin, and prolactin. Further investigation may also require measurement of levels of circulating 17-OH-progesterone, follicle-stimulating hormone, oestradiol and serum cortisol. Correct identification of this sub-group allows treatment of the underlying disorder but also, anecdotally, may predict a more favourable response to therapies such as cyproterone acetate, spironalactone or finasteride.

Male pattern hair loss is considered physiological and consequently treatment for this condition is not available on the NHS. For this reason male pattern hair loss has been excluded from this article, although further information may be obtained from Blume-Peytavi et al (2010) and Blumeyer et al (2011).

Cicatricial alopecias

The term cicatricial alopecia is derived from the Latin word cicātrīx, meaning scar. These scarring condition can be divided into primary cicatricial alopecias, caused by an underlying primary pathology of the hair follicle, and secondary cicatricial alopecias, which may result from damage to hair-bearing skin by a distant process or local trauma (e.g. ionizing radiation, infection, burns, sarcoidosis, surgery).

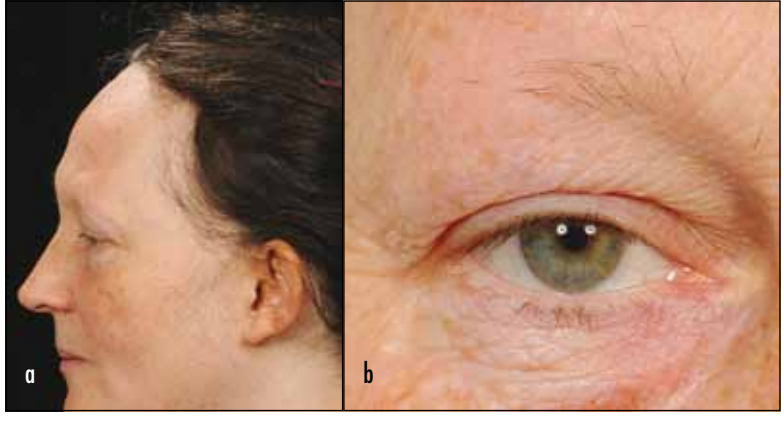
The primary cicatricial alopecias are characterized by scarring, identified by visible loss of the openings of the hair follicles ('follicular ostia') on magnification of the scalp. Scalp biopsy plays an essential role in the diagnosis by identifying the inflammatory infiltrate seen around the hair follicle as lymphocytic, neutrophilic or mixed. This division relies on adequate skin sampling from the area of active inflammation, which is usually identified by erythema and increased scale, additionally guided by patient symptoms (Harries et al, 2009).

The scarring of primary cicatricial alopecia is irreversible as a result of inflammation-induced damage to the epithelial hair follicle stem cells located at the 'bulge region', an area of hair follicle epithelium at the level of the insertion of the erector pili muscle (Harries and Paus, 2010). The aim of treatment in these conditions is to arrest progression and reduce symptoms.

Lichen planopilaris and variants are the most commonly encountered primary cicatricial alopecia. Lichen planopilaris is seen predominantly in women and is most likely to have a localized pattern on the central scalp, with perifollicular erythema and scale visible at the periphery of the alopecia patch. Patients may describe itch and tenderness from the scalp, and may also have evidence of lichen planus at another skin site, mucous membranes or nails. Frontal fibrosing alopecia is a variant of lichen planopilaris that typically affects post-menopausal women with a band-like recession of the frontal hairline associated with loss of eyebrows (*Figure 4*). Similar inflammation to classical lichen planopilaris can be seen at the advancing edge of the alopecia. Lichen planopilaris and variants may be treated in a similar manner after confirmation of the diagnosis by scalp biopsy. Potent topical steroids applied for 12 weeks initially is the most commonly prescribed first-line treatment, although clinical response is often variable (Harries et al, 2008). Other treatments include intralesional steroid injections, oral steroids, oral retinoids, tetracycline antibiotics and antimalarials, although for all treatments the evidence base is generally poor (Harries et al, 2008).

Chronic cutaneous lupus erythematosus of the scalp appears as erythematous and scaly plaques with evidence of follicular plugging and variable dyspigmentation. The focus of inflammation is in the centre of the localized area of alopecia, which is typically on the vertex or central scalp. Treatment options include potent topical steroids, intralesional steroids, antimalarials, topical immunomodulators (e.g. tacrolimus, pimecrolimus), oral retinoids and thalidomide (Harries et al, 2008). Patients should also be warned to protect

Figure 4. Frontal fibrosing alopecia with (a) band-like scarring alopecia of the frontal hairline and (b) involvement of the eyebrows.



themselves against ultraviolet radiation with the use of a hat and sunblock.

A Cochrane review of drugs for chronic cutaneous lupus erythematosus found only two trials that met inclusion criteria and concluded that a more potent topical steroid (i.e. fluocinonide cream) was more effective than hydrocortisone in this condition and that hydroxychloroquine and acitretin appear to be of equal efficacy, although adverse effects were more frequent in the acitretin group. Jessop et al (2009) concluded that there was not enough reliable evidence about other drug treatments for chronic cutaneous lupus erythematosus.

The neutrophilic primary cicatricial alopecias include folliculitis decalvans and dissecting cellulitis of the scalp. Folliculitis decalvans usually presents with a history of painful localized patches of alopecia, which may affect any hair-bearing site but most commonly the vertex of the scalp (*Figure 5*). Clinical signs include thickened (hypertrophic) and occasionally boggy scarring, tufting of the hair (i.e. many hairs leaving scalp through same ostia giving a 'doll's hair' appearance), pustules and scale. Bacterial swabs may culture *Staphylococcus aureus* and mycology samples will be negative (Harries et al, 2009).

Folliculitis decalvans may respond to eradication of *Staph. aureus* with the use of a single sensitive antibiotic, but symptoms may quickly recur. Thus, a 10-week course of rifampicin 300 mg twice daily and clindamycin 300 mg twice daily in combination is often suggested as this may be beneficial, potentially resulting in a sustained remission. Although less robust evidence is available, anecdotally there is a role for dapsone (Paquet and Pierard, 2004) and oral retinoids (Pujol et al, 1994) in some patients.

Dissecting cellulitis of the scalp presents with numerous fluctuant nodules on the scalp with associated sinus tract formation. It is more common in males, and is associated with acne conglobata and hidradenitis suppurativa as a result of a common observation of follicular occlusion in these conditions. Treatment with oral isotretinoin for 6–12 months has been beneficial in case reports and other treatments may include oral antibiotics, intralesional triamcinolone injections and dapsone (Harries et al, 2008).

A mixed neutrophilic and lymphocytic infiltrate may be seen in acne keloidalis nuchae, which is more commonly seen in young, Afro-Caribbean males. The distribution of hair loss is typically over the occipital scalp with skin-coloured, firm nodules and keloid-like plaques which may be induced or aggravated by mechanical trauma, such as shaving. Acne keloidalis nuchae leads to permanent scarring alopecia but the inflammation can be improved with the use of potent topical or intralesional steroids, with or without oral antibiotics; excision surgery is an option for those who do not respond to medical treatment (Harries et al, 2008).

Infective causes of alopecia

Tinea capitis

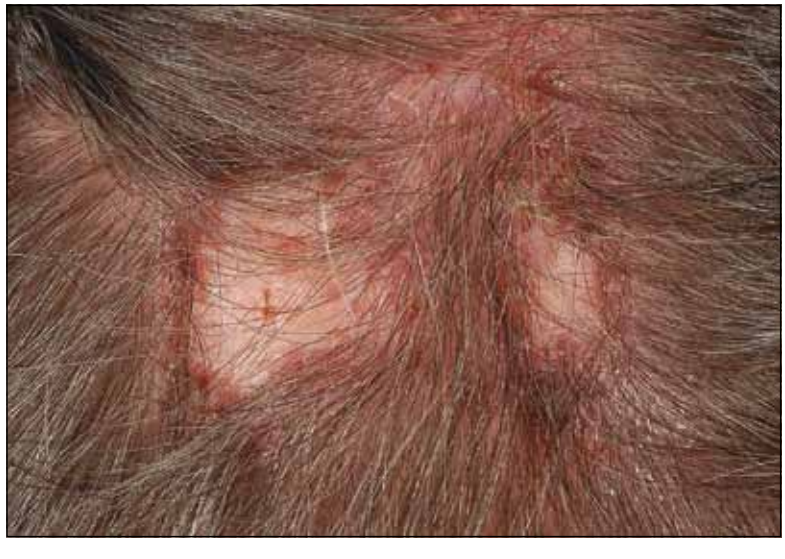
Fungal infections are a common cause of a patch of hair loss in children and should always be suspected especially if inflammation is present (e.g. increased scale, bogginess, pustules). Samples should be taken for fungal microscopy and culture. Clinical features may range from patchy hair loss with small amounts of scale and broken hairs to a large, painful inflammatory mass, termed a 'kerion', which may then lead to an area of scarring alopecia.

A meta-analysis by Tey et al (2011) suggests that oral terbinafine is more efficacious than griseofulvin in treating tinea capitis caused by *Trichophyton* spp. and griseofulvin is more efficacious than terbinafine in treating tinea capitis caused by *Microsporum* spp. Therefore an attempt should be made to identify the pathogenic fungus before initiation of treatment, as a targeted approach will lead to more effective use of antifungal agents. *Trichophyton* spp. are the most common cause of kerion and the incidence of trichophyton infections is increasing in recent years, in particular in urban areas within the USA and UK (Hay and Ashbee, 2010).

Alopecia syphilitica

Annual diagnoses of infectious syphilis increased twelvefold between 1997 and 2007 and alopecia syphilitica may occur as the first presentation of this infection in up to 12% of those with secondary syphilis (Bi et al, 2009; Health Protection Agency, 2009). Alopecia syphilitica is a non-scarring alopecia and is moth-eaten in appearance (*Figure 6*) but a diffuse variant is also described. Alopecia syphilitica may affect any hair-bearing skin, but the most common site is the scalp. Once the suspicion of secondary syphilis is raised, associated features such as mucocutaneous involvement and lymphadenopathy should be sought. Diagnosis is confirmed by VDRL serology and co-existent HIV infec-

Figure 5. Folliculitis decalvans with localized scarring alopecia and pustules.



tion must be considered. Alopecia syphilitica should resolve gradually over several months following treatment with appropriate antibiotics.

Mechanical causes of alopecia

Traction alopecia

Traction alopecia is caused by hair care and styling practices, usually when the hair is braided or worn in a tight style, and is most common in women of Afro-Caribbean descent. The initial alopecia is non-scarring and may show broken hairs occurring at sites of most tension. However, with time the chronicity of the trauma may induce a scarring alopecia. Key to treatment of traction alopecia is reduction of tension on the hair by adopting loose hairstyles, sometime supplemented with topical or intralesional steroids and topical minoxidil lotion (de Berker et al, 2004). Hair transplantation may be considered in longstanding cases that have shown no evidence of further re-growth with these measures.

Figure 6. Alopecia syphilitica with moth-eaten appearance of hair loss.

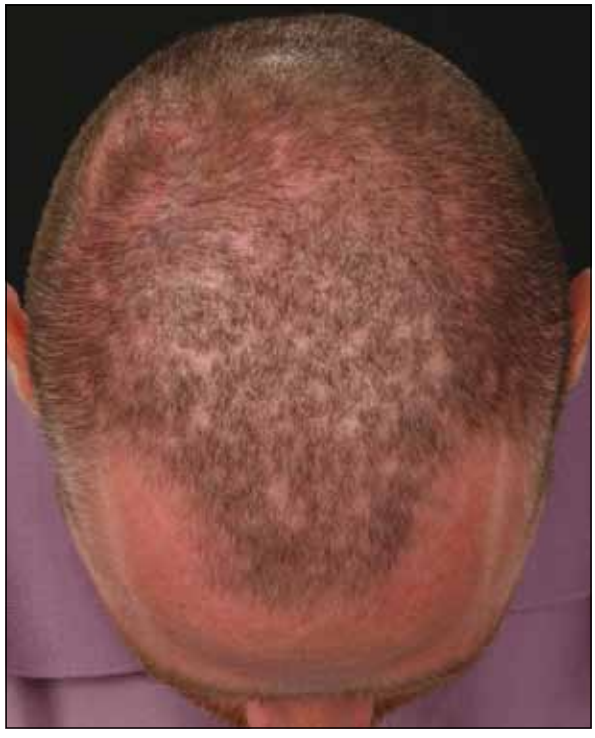
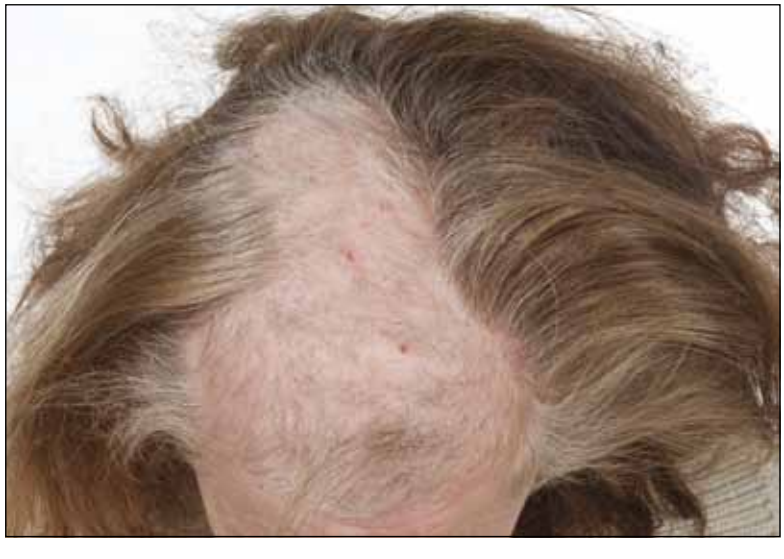


Figure 7. Trichotillomania demonstrating relative sparing of the frontal hairline and broken hairs.



Trichotillomania

Trichotillomania is the compulsion to remove one's own hair and is observed as bizarre and irregularly shaped alopecia patches with short, broken hairs (*Figure 7*). An increased emotional feeling of tension before the hair is pulled and a sense of relief once the hair is removed is documented (Millard and Cotterill, 2004). This condition most commonly affects the scalp but may affect the eyelashes or eyebrows in isolation in a minority of patients. It is described in both adults and children; in younger children this may just represent a habit that often settles, whereas in teenagers and adults it may represent a more significant underlying psychological disorder. The differential diagnosis includes localized alopecia areata, but when the short hairs are examined they are structurally normal but short and broken, rather than the tapered dystrophic exclamation mark hairs typical of alopecia areata.

Habit reversal techniques, selective serotonin re-uptake inhibitors and tricyclic antidepressants have been used with some success. Van Ameringen et al (2010) studied the use of olanzapine for the treatment of trichotillomania and found the agent to be superior to placebo when measuring the Yale-Brown Obsessive Compulsive scale of trichotillomania. The treatment and follow-up period was short (12 weeks) but results appear to support that trichotillomania may lie on the obsessive-compulsive spectrum of disorders, characterized by compulsive urges and ritualized behaviours.

Wigs, support and surgery

With alopecia of any cause it is important to explain the condition as well as the aim of treatment so the patient has reasonable expectations of outcomes and long-term prognosis. In many cases, for example cicatricial alopecia, the aim is to halt progression as de novo hair growth is unrealistic. It is often helpful for patients to discuss their condition with other sufferers. Contact with patient support groups should be encouraged (see contact details below). Support groups can also offer useful practical advice regarding the use of hair pieces, false eyelashes and semi-permanent tattooing.

Hair pieces or wigs are available on prescription in England and Wales with each patient being entitled to two NHS wig prescriptions per year and these prescriptions are provided by secondary care dermatologists.

Surgical techniques, such as follicular unit hair transplantation, may be of help to those with sufficient donor

hair, usually in the occipital scalp (Blumeyer et al, 2011). Conditions most likely to benefit are 'burnt out' scarring alopecia with no ongoing inflammation, secondary cicatricial alopecia and female or male pattern hair loss despite continued use of topical minoxidil.

Conclusions

This article has presented an overview of commonly encountered hair loss disorders and proposed a practical approach to diagnosis and treatment. Further quality clinical and basic science research is required in many areas of this subspeciality to provide a robust body of evidence on which to better our understanding of these conditions and make informed therapeutic recommendations in the future. **BJHM**

Figure 6 *was kindly supplied by Dr SN Shah.*

Conflict of interest: none.

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Patient support groups and information

Alopecia UK (www.alopeciaonline.org.uk)

Cicatricial Alopecia Research Foundation (www.carfintl.org)

Further patient information leaflets are available from the British Association of Dermatologists (www.bad.org.uk)

KEY POINTS

- In hair loss disorders, a careful history and examination will most often reveal the underlying diagnosis without need for extensive investigation.
- Simple bed-side tests, such as a hair part width and hair pull test, can provide additional clues to aetiology of alopecia within the clinic setting.
- Scalp biopsy is always indicated in scarring alopecia, where two punch biopsies should be taken from an area of active inflammation and redness, usually in the periphery of the patch of alopecia, to identify the predominant cell type of the inflammatory infiltrate.
- Telogen effluvium may be precipitated by one of many triggers up to 6 months before onset of alopecia and underlying systemic disease should be sought.
- Spontaneous resolution may occur in alopecia areata of limited extent (<50% surface area) and 'observation only' is an accepted treatment option in this group, but more extensive disease may require topical or systemic treatment.
- In female pattern hair loss, caution must be exercised to identify the sub-group with additional features of hormone dysregulation such as acne, hirsuitism, menstrual irregularities and infertility, as these patients require further investigation and treatment.
- The aim of treatment in scarring alopecia is to halt progression, as de novo hair growth is unrealistic and a key component of treatment is the management of expectations and linking with patient support groups.
- Two NHS wig prescriptions are available per annum from secondary care for patients with significant alopecia via dermatology clinics.