An approach to the diagnosis and management of patchy, non-scarring hair loss

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Abstract

This article presents a clinical approach to patchy, non-scarring hair loss and includes conditions like alopecia areata, trichotillomania, dermatophyte infection of the scalp, syphilitic alopecia and traction folliculitis.

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Introduction

Shedding of hair is termed effluvium or defluvium, and the resulting condition is called alopecia (Greek: alópekia, [baldness]). The normal scalp is adorned with approximately 100 000 hairs. More than 90% are in the growing phase (anagen). Up to 100 hairs may normally be shed per day. Individuals are often aware of and very concerned about subtle thinning of their hair. Patients presenting with hair loss are commonly encountered in clinical practice. The causes of hair loss are outlined in Table I.

Table I: Causes of hair loss

Scarring hair loss
Primary
Lymphocyte associated
- Lichen planus
- Lupus erythematosus
- Pseudopelade of Brocq
- Follicular mucinosis

Neutrophil associated
- Folliculitis decalvans
- Diffecting folliculitis
- Folliculitis keloidalis nuchae

Secondary
- Trauma
- Radiotherapy
- Some dermatophyte infections
- Primary neoplasia
- Secondary neoplasia (metastasis)

Non-scarring hair loss
On an abnormal scalp
- Psoriasis
- Seborrhoeic dermatitis
- Some dermatophyte infections
- Traction alopecia

On a normal scalp
Diffuse
- Telogen hair loss
- Anagen hair loss

Patchy
- Alopecia areata
- Triangular alopecia
- Trichotillomania
- Secondary syphilis

With a distinct pattern
- Male pattern baldness
- Female pattern baldness

Other causes of hair loss
- Hair shaft abnormalities
- Congenital hypotrichosis
- Loose anagen syndrome

Table II: Non-scarring hair loss versus scarring hair loss

	Non-scarring hair loss	Scarring hair loss
Incidence	common	less common
Erythema/scaling/ pustulation	-/+	+
Atrophy	-	+
Loss of follicular openings	-	+
Tufted hair*	-	+
Course	regrowth is quite common	no regrowth
Prognosis	generally favourable	generally unfavourable

* Tufted hair - more than one hair shaft emerge from a single follicular opening as a result of abnormal follicular dynamics related to fibrosis.

Differences between scarring and non-scarring hair loss are summarised in Table II.

Five conditions frequently encountered in clinics and presenting with patchy, non-scarring hair loss, are alopecia areata, trichotillomania, dermatophyte infection of the scalp, secondary syphilis, and traction folliculitis/alopecia.

Alopecia areata

Alopecia areata (AA) is an autoimmune disease caused by interaction of T-lymphocytes and follicular epithelium resulting in progressive shrinkage of follicles and loss of hair. In case of fulminant AA, persons may experience "going grey overnight". Alopecia occurs in round or oval areas without any visible inflammation of the skin (Figure 1).

There may be one, or several, discrete or confluent patches. Follicular openings are present. No atrophy or scarring occurs. The most common presenting area is the scalp, but the eyebrows, eyelashes, pubic hair and beard may also be involved. Alopecia areata totalis (AAT) refers to total absence of terminal scalp hair (Figure 2) and alopecia areata universalis (AAU) to total loss of terminal body and scalp hair. Ophiasis is characterised by a bandlike pat-

Figure 1: Alopecia areata. The scalp appears normal. (Figure courtesy of Derm101.com)

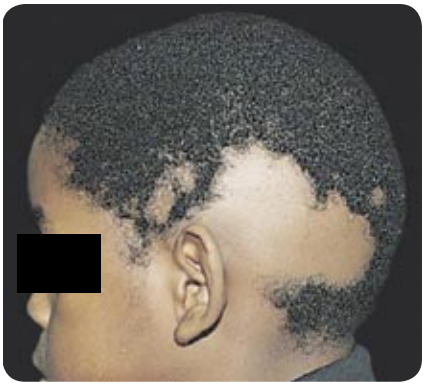
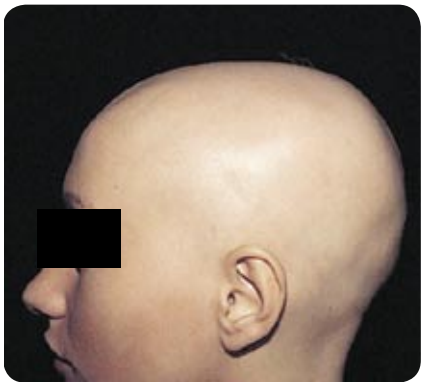


Figure 2: Alopecia totalis. Eyebrow hair is also sparse. (Figure courtesy of Derm101.com)



tern of hair loss over the periphery of the scalp. Hair loss usually develops gradually over weeks to months. Patches of AA can be stable and often show spontaneous regrowth over a period of several months; new patches may appear while others resolve. AA is asymptomatic but individuals are usually very concerned about the hair loss and possible continued, progressive balding.

Occasionally diagnostic, broken-off, stubbly hairs called exclamation point hairs (distal ends are broader than proximal ends) are present. With the regrowth of hair, new hairs are fine, often white or grey. Microscopy of the skin shows peribulbar lymphocytes, likened to a "swarm of bees", and miniaturisation of hair follicles.

The nails may show fine pitting ("hammered brass") of the dorsal nail plate, mottled lunulae, trachyonychia (rough nails) and onychomadesis (separation of nail from matrix). Associated findings include Hashimoto's thyroiditis, vitiligo, myasthenia gravis and Addison's disease.

Differential diagnosis

In the differential diagnosis, secondary syphilis, grey-patch tinea capitis, trichotillomania, traction alopecia, early chronic cutaneous lupus erythematosus and androgenetic alopecia should be considered. The differences between trichotillomania and alopecia areata are outlined in Table III.

Table III: Differential diagnosis of trichotillotic hair loss and alopecia areata

	Alopecia areata	Trichotillotic hair loss
Mechanism Incidence	Autoimmune disorder Males = females, Peak: second and fifth decades	Compulsive psychosomatic disorder Females:males = 2:3 in children Females:males = 4:1 in adults Children:adults = 7:1 peak two to six years
History	Sudden onset, no history of hair pulling	Insidious onset, history of hair pulling
Clinical features	Circumscribed, smooth, totally bald patch; residual exclamation hairs; loose marginal hairs (in the active stage)	Ill-defined, uneven alopecia with broken hairs of different lengths: exclamation hairs and strong marginal hairs may be present
Histopathology	Increased telogen hairs, lymphocytic infiltrate around anagen follicles; miniaturised follicles; amelanotic hairs	Increased catagen hairs; no inflammatory infiltrate; traumatised hair bulbs with haemorrhage; trichomalacia (specific)
Nails	Nail pitting (dystrophic plate changes)	Nail biting (irregularly eaten distal nail margins)
Underlying psychology	Psychological abnormality found in 90%, with a triggering emotional stress common	Obsessive-compulsive disorder
Associations	Atopic and autoimmune diseases	Atopic diseases, anaemia
Prognosis	Good, except in certain groups (atopic, early onset, ophiatic and extensive forms)	Fair in children, poor in adults
Relapse rate	100%	Recurrent in adults
Treatment	Reassurance, steroids, topical immunotherapy	Reassurance, antidepressants, psychotherapy

Course

Spontaneous remission is common in patchy AA. Poor prognosis is associated with a number of factors (Table IV). If AA occurs after puberty, 80% of patients experience hair regrowth within a year. Recurrences of AA are, however, frequent.

Table IV: Factors indicative of poor prognosis

- Ophiasis (bandlike pattern)
- Alopecia areata totalis
- Alopecia areata universalis
- Onset < five years of age
- Duration > five years of age
- Atopy

Management

No cure is currently available. Remission may be induced but the course is not altered. Treatment for AA is generally unsatisfactory. In many cases, the most important factor in the management of the patient is psychological support from the dermatologist, family, and support groups. Individuals may prefer to wear a wig. Make-up applied to the eyebrows is helpful.

The first line of treatment is the application of a potent steroid cream twice daily for six weeks. If there is no improvement, this treatment should be terminated. Few and small spots of AA can be treated with an intralesional steroid. Tufted regrowth of hair commonly results.

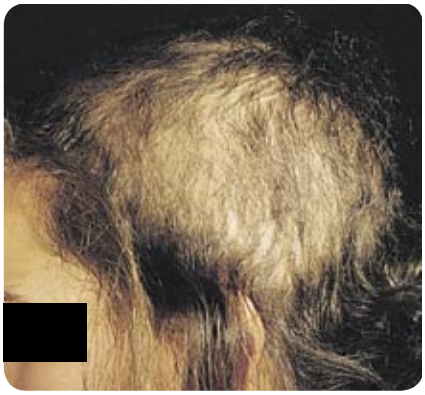
Systemic glucocorticoids usually induce regrowth, but AA recurs on discontinuation. The risks of long-term therapy therefore rule out their use. Systemic cyclosporin induces regrowth, but AA recurs when the drug is discontinued. Cyclosporin is costly and renal damage is troublesome. Induction of allergic contact dermatitis with nitrochlorobenzene, squaric acid dibutylester, or diphencyprone can be used successfully, but local discomfort due to allergic contact dermatitis, and the swelling of regional lymph nodes, poses a problem. Oral PUVA (photochemotherapy) is variably effective, as high as 30%, and worth a trial in patients who are very distressed by the problem. The entire body must be exposed since the therapy is believed to be a form of systemic immune suppression. PUVA treatment has many side effects, the most important being nausea, photosensitivity and the development of nonmelanoma skin cancer.

Trichotillomania

Trichotillomania (TTM), first described by Halopeau in 1889, is an obsessive-compulsive disorder, characterised by the irresistible urge to pull out hair. There is a sensation of relief after the pulling episodes. The psychological stimulus is satisfied by hair plucking, but the resulting unsightly alopecia fuels the anxiety of the patient and results in a vicious cycle.

Adult TTM is more frequently chronic and is associated with more profound psychopathy. Adult TTM is more common in females (females:males 15:1). Childhood TTM is more common and also more common in females (females:males 4:1). TTM usually involves the

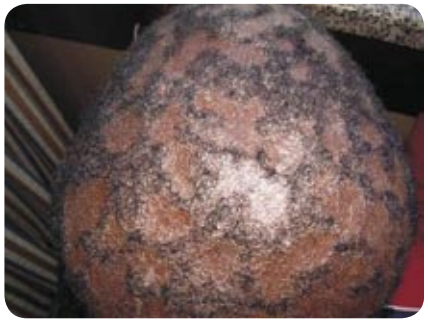
Figure 3: Trichotillomania. Hairs are thin and of different lengths. (Figure courtesy of Derm101.com)



scalp, but sometimes the eyebrows or eyelashes are plucked. Manual dominance is characteristic: left-sided lesions develop in right-handed patients and vice versa. Lesions are ill-defined and the scalp surface is normal (Figure 3).
The stubble of unevenly broken hairs is highly characteristic. The residual stubble hair has a normal colour and texture. Sometimes dystrophic "exclamation hairs" may also be found in TTM. The differences between AA and TTM are outlined in Table III.

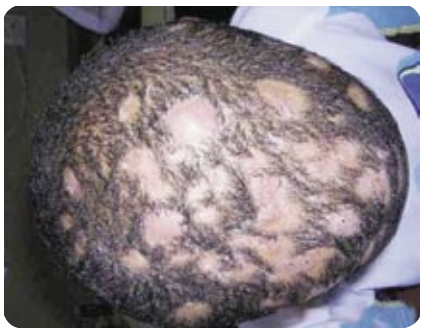
Dermatophyte infection of the scalp
Tinea capitis is most commonly caused by *Trichophyton violaceum* of the species trichophyton. The infection is usually spread from person to person. Infection by species of the genus microsporum is less common. Microsporum infection is usually acquired from cats or dogs.
The clinical presentation of this dermatophyte infection is variable, the commonest being grey-patch tinea capitis. The number of lesions is variable. A single lesion may be present, but more commonly one finds several lesions. Lesions show loss of hair and greyish, tightly packed, small scales (Figure 4).

Figure 4: Dermatophyte infection of the scalp. Multiple areas of hair loss with scaling.



Yellow-patch tinea capitis is a related condition. Lesions are covered with yellowish crusts caused by secondary pyoderma. Tinea capitis may also be seborrhoeic dermatitis-like and pustular. Black-dot tinea capitis is characterised by patches of alopecia studded with black dots which represent broken-off hair shafts packed with spores. Abscess-like lesions, e.g. kerion, and lesions displaying saucer-shaped scales (= scutulae) e.g. favus, are infrequent. Secondary pyoderma and cervical lymphadenopathy are often present. Pyoderma may lead to scarring (Figure 5). When pyoderma of the scalp is observed, tinea capitis, scabies and pediculosis should always be borne in mind.

Figure 5: Dermatophyte infection of the scalp. Multiple areas of hair loss with scarring.

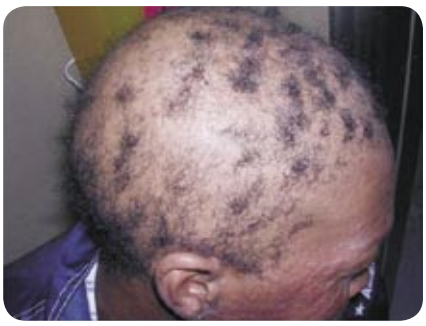


The diagnosis of tinea capitis can often be established clinically, and can be confirmed by microscopy of skin scrapings or hair plucked from involved areas. Such samples can also be despatched for culture on Sabouraud's dextrose agar. A therapeutic trial of antifungal treatment remains an acceptable alternative.

The treatment of choice is griseofulvin 10 - 20 mg/kg per day, with a meal, for 6 to 8 weeks. Patients should be followed up to ensure adequate healing of the infection. Regular use of a shampoo containing povidone-iodine decreases the shedding of spores.

Syphilitic alopecia (Secondary syphilis)
Secondary syphilis, the stage of spirochaetemia, is characterised by constitutional symptoms, variable organ involvement, generalised lymphadeno-pathy, skin eruptions, mucosal lesions, mucocutaneous lesions and hair loss. Skin eruptions include macular, papular, papulosquamous, nodular and pustular variants. The skin rash is usually asymptomatic and symmetrical. The palms and soles are commonly involved. In late stage secondary syphilis, lesions often show an arciform, annular or corymbiform pattern. Snail track ulcers are commonly observed on the oral mucosa and flat, glistening condylomata lata are present at moist mucocutaneous junctions. Syphilitic alopecia is characterised by patches of hair loss on the scalp. These patches show normal skin. The pattern is typically described as moth-eaten (Figure 6). No particular pattern is observed. A clinical diagnosis of secondary syphilis is confirmed by appropriate serological tests. A skin biopsy may be helpful. Treatment with penicillin is usually curative.

Figure 6: Syphilitic alopecia. The scalp has a moth-eaten appearance, eyebrow hair is absent and there is a rash on the cheek.



Traction folliculitis/alopecia
Hair and scalp diseases induced by traumatic hairstyling techniques, such as tightly drawn braids (Figure 7), are underappreciated.

Figure 7: Traction alopecia. Traction folliculitis is commonly associated.

A geometric pattern with the hair sectioned into squares or bands is highly characteristic. The hair in each square or band is pulled tightly to the centre and then secured with a hair band. Maximum traction is produced round the outer edges of the square or band with less traction in the centre. The first noticeable changes are faint erythema, some scaling and follicular mini pustules. Subsequently, in the areas of maximum traction, follicle-based papules and pustules associated with alopecia become evident. Hair loss and follicle-based papules and pustules along the frontal scalp margin are common. The pustules are sterile. This form of hair loss is also common in persons wearing a ponytail or bangs. Other hairstyles that can lead to traction folliculitis include hair twists worn by Sikh boys, chignons, hair weaving and hair extensions. Traction folliculitis may be associated with hair casts. These casts encircle the hair shaft, are yellowish white and are freely mobile. The units of pediculosis capitis are firmly fastened and immobile. Continuous traction causes loosening of the hair from the follicles resulting in

inflammation i.e. folliculitis. If the traction continues, chronic inflammation ensues, which may lead to follicular atrophy with thinner, shorter hair. Initially the process is reversible, but follicular destruction, scarring and permanent hair loss may follow if traction persists. An associated posterior cervical lymphadenopathy is not uncommon.

See CPD Questionnaire, page 34

Ⓟ This article has been peer reviewed

References
1. Shelleh HH, Khan SA, Al-Hatiti HS. Trichotillomania or alopecia areata? Int J Dermatol 2006;45:1196-8.
2. Fox GN, Stausmire JM, Mehregan DR. Traction folliculitis: an underreported entity. Cutis 2007;79:26-30.

ADVANCE trial: Treating Hypertension vs. Lowering Blood Pressure: extending the benefit?

Results from the largest ever study in type 2 diabetes, the ADVANCE study, will have important clinical implications for patients. Diabetes mellitus is emerging as one of the greatest threats to the health of populations worldwide. Epidemiological data from the International Diabetes Federation puts the current number of patients with diabetes at 7,1 million in Africa. This number is estimated to rise to 15 million in 2025. The current prevalence of diabetes in South Africa varies from 5-30% depending on region, age and ethnicity, and it is well-known that these patients are at increased risk for cardiovascular complications. Most diabetic patients are also hypertensive, and the presence of hypertension further increases the cardiovascular risk of diabetic patients.

"People with diabetes have a two to four-fold greater risk of experiencing a cardiovascular event compared to non-diabetics. However, despite this high risk, there is surprising little recent evidence from randomised clinical trials on the role of blood pressure lowering in the prevention of diabetic vascular disease", points out the ADVANCE study chairman John Chalmers from the George Institute.

Previous trials in type 2 diabetic patients clearly demonstrated the benefit of lowering blood pressure in these patients. However, the Blood Pressure target of 130/89 mmHg was never reached in these trials, and blood pressure treatment seen as intensive was in fact not intensive by today's standards. Therefore, the blood pressure lowering arm of ADVANCE, with the use of Coversyl Plus® (perindopril 4mg and indapamide 1,25mg), aimed to begin lowering BP where previous trials have ended by including type 2 diabetic patients whatever their Blood Pressure level (average starting blood pressure 145/81). These patients were included in order to demonstrate the benefit of blood pressure lowering, as opposed to only treating hypertension. Coversyl Plus® was chosen specifically for ADVANCE, since perindopril has demonstrated 24 hour blood pressure control, and both perindopril and indapamide are glucose and lipid neutral. Added to this is the evidence that perindopril has proven benefits beyond blood pressure lowering.

In ADVANCE, Coversyl Plus® was added to all existing therapy including antihypertensives, lipid-lowering therapy and antiplatelet therapy.

For the physician, ADVANCE will show whether additional blood pressure lowering with Coversyl Plus®, will reduce mortality and the risk of the type 2 diabetic developing macro - and microvascular complications. These results should ensure that blood pressure lowering in this patient group, with an appropriately selected combination of agents, gets the attention that it deserves.

FOR MORE INFORMATION AND RESULTS VISIT: www.advance-trial.com

References:
1. International Diabetes Federation. Executive Summary of the Diabetes Atlas. www.idf.org
2. SEMDSA website. www.semdsa.org.za
3. Mancia G et al. Systolic and Diastolic Blood Pressure Control in Antihypertensive Drug Trials. *J Hyper* 2002; 20:1461-1464
4. Chalmers J, "Treating Hypertension" or "Lowering Blood Pressure" Extending the Concept. *Blood Pressure* 2002; 11: 68-70
5. The ADVANCE Collaborative Group. Rationale and Design of the ADVANCE Study: A Randomised Trial of Blood Pressure Lowering and Intensive Glucose Control in High-Risk Individuals with Type 2 Diabetes Mellitus. *J Hyper* 2001; 19(suppl 4):S21-S28.
6. The EUROPA Study. *Lancet*, 2003; 362:782-788
7. Myers et al *Journal of Hypertension* 2000 vol 18: 317-325