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British Association of Dermatologists' guidelines for the management of alopecia areata 2012

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1.0 Purpose and scope

The guidelines have been revised and updated in accordance with a predetermined scope, based on that used in the 2003 guidelines. Recommendations in these guidelines supersede those in the 2003 guidelines. The objectives of the guidelines are to provide up-to-date recommendations for the management of alopecia areata in adults and children and a summary of the evidence base.

2.0 Stakeholder involvement

This guidance has been written by dermatologists and a patient representative. The draft guideline was made available for consultation and review by the British Association of Dermatologists' (BAD) membership, the Primary Care Dermatological Society (PCDS), the British Dermatological Nursing Group (BDNG) and the board of Alopecia UK, a patient support organization. The final document was peer-reviewed by the Clinical Standards Unit of the BAD (made up of the Therapy and Guidelines subcommittee) prior to publication.

3.0 Methodology

These guidelines have been developed using the BAD's recommendations[1] and also with reference to the AGREE (Appraisal of Guidelines Research and Evaluation) instrument.[2] PubMed, MEDLINE and EMBASE databases were searched from January 2002 to January 2012 and full relevant papers in the English language obtained. Additional, targeted searches were also carried out across these three databases, as well as a search on the Allied and Complementary Medicine Database (AMED); details of the search strategy are available as an Appendix online (see Supporting Information).

The recommendations made are those that are currently considered best practice. Where possible they are based on randomized controlled trials (RCTs). However, in view of the limited evidence from RCTs, guidance is also based on less rigorously controlled studies, uncontrolled studies, on clinical experience, and on patient experience. These recommendations will be modified at intervals in light of new evidence.

4.0 Limitations of the guidelines

This document has been prepared on behalf of the BAD and is based on the best data available when the document was prepared. It is recognized that under certain conditions it may be necessary to deviate from the guidelines, and that the results of future studies may require some of the recommendations herein to be changed. Failure to adhere to these guidelines should not necessarily be considered negligent, nor should adherence to these recommendations constitute a defence against a claim of negligence.

5.0 Plans for guideline revision

The proposed revision date for this set of recommendations is scheduled for 2017; where necessary, important interim changes will be updated on the BAD website.

6.0 Background

Alopecia areata is a chronic inflammatory disease that affects the hair follicle and sometimes the nail. The onset may be at any age and there is no known race or sex preponderance. Alopecia areata usually presents as patches of hair loss on the scalp but any hair-bearing skin can be involved. The affected skin may be slightly reddened but otherwise appears normal. Short broken hairs (exclamation mark hairs) are frequently seen around the margins of expanding patches of alopecia areata. The nails are involved in about 10% of patients referred for specialist advice.

6.1 Prognosis

Hair follicles are preserved in alopecia areata and the potential for recovery of hair growth is maintained, even in longstanding disease. One study from Japan reported that spontaneous remission within 1 year occurred in 80% of patients with a small number of circumscribed patches of hair loss.[3] Data from secondary and tertiary referral centres are less favourable indicating that 34–50% of patients will recover within 1 year. Almost all will experience more than one episode of the disease, and 14–25% progress to total loss of scalp hair (alopecia totalis, AT) or loss of the entire scalp and body hair (alopecia universalis, AU), from which full recovery is unusual (< 10%).[4,5] Disease severity at presentation is the strongest predictor of long-term outcome. In an Italian study, 191 patients with alopecia areata who presented to a university dermatology clinic between 1983 and 1990 were contacted by telephone in 2005 to give self-reports of their clinical status.[6] Patients with less severe disease at presentation were more likely to report being free of disease at follow-up (68% with less than 25% hair loss initially; 32% with 25–50% hair loss initially; 8% with more than 50% hair loss initially). Patients with more severe disease initially were also more likely to report worsening patterns of alopecia such as AT and AU.

The prognosis is also less favourable when onset occurs during childhood[4,7–9] and in ophiasis.[9] The concurrence of atopic disease has been reported to be associated with a poor prognosis[3,9] but this has been disputed.[10]

6.2 Aetiology

About 20% of people with alopecia areata have a family history of the disease indicating a genetic predisposition.[11] Associations have been reported with a variety of genes, including major histocompatibility complex (MHC) and cytokine genes, suggesting that the genetic predisposition is multifactorial in nature. A genome-wide association study confirmed the link with the MHC genes and also identified associations with other genes involved in regulating immune and inflammatory responses, and with some genes expressed in the hair follicle.[12] The hair follicle lesion is probably mediated by T lymphocytes.[13] The association between alopecia areata and other autoimmune diseases suggests that alopecia areata is itself an autoimmune disease although this is unproven. It has been proposed that the hair follicle is an immunologically 'privileged tissue' which is sheltered from immune surveillance by autoreactive T cells, and that failure of such immune privilege plays a key role in the pathogenesis of alopecia areata.[14,15]

7.0 Diagnosis

The diagnosis of alopecia areata is usually straightforward although the following may cause diagnostic difficulties:

1 Trichotillomania – this condition probably causes most confusion and it is possible that it coexists with alopecia areata in some cases. The incomplete nature of the hair loss in trichotillomania and the fact that the broken hairs are firmly anchored in the scalp (i.e. they remain in the growing phase, anagen, unlike exclamation mark hairs) are distinguishing features.
2 Tinea capitis – the scalp is inflamed in tinea capitis but the signs may be subtle.
3 Early scarring alopecia.
4 Telogen effluvium.
5 Anagen effluvium (drug-induced) may mimic diffuse alopecia areata.
6 Systemic lupus erythematosus.
7 Secondary syphilis.

Dermoscopy can aid the diagnosis of alopecia areata. Regular round yellow dots are commonly seen in areas of hair loss and can indicate active disease progression. Dermoscopy also highlights common features seen in this condition such as dystrophic hairs with fractured tips (exclamation mark hairs) and hairs fractured before emergence from the scalp (cadaverized hairs). These findings are not present in triangular alopecia, trichotillomania or localized scarring conditions, which are sometimes considered within the differential of alopecia areata.[16] Occasionally, alopecia areata presents as diffuse hair loss which can be difficult to diagnose. The clinical course

often reveals the true diagnosis but a biopsy may be necessary in some cases.

8.0 Investigations

Investigations are unnecessary in most cases of alopecia areata. When the diagnosis is in doubt appropriate tests may include fungal culture, skin biopsy, serology for lupus erythematosus or serology for syphilis. The increased frequency of autoimmune disease in patients with alopecia areata is probably insufficient to justify routine screening.

One small case series suggested that iron deficiency is more common in women with alopecia areata than the population at large[17] but this was not confirmed in two subsequent studies,[18,19] and routine testing for iron status is not recommended. There are no published studies demonstrating a treatment response to iron replacement therapy.

9.0 Management

An overriding consideration in the management of alopecia areata is that, although the disease may have a serious psychological effect, it has no direct impact on general health that justifies the use of hazardous treatments, particularly of unproven efficacy. In addition, many patients, although by no means all, experience spontaneous regrowth of hair. However, the psychological effects of alopecia may impact on general health and depends on the individual's coping strategy when dealing with an altered body image, which can result in higher levels of anxiety and a greater risk of depression leading to social, work-related and personal problems.[20]

9.1 Counselling

An explanation of alopecia areata, including discussion of the nature and course of the disease and the available treatments, is essential. Some patients are profoundly upset by their alopecia and may require psychological support. Many find it difficult to disclose their alopecia to family members and friends and struggle to find the answers to their medical and many practical questions. Contact with other patient experts and patient support groups can help individuals cope with the changing aspects of alopecia and provide support to find a new level of self-acceptance of their altered body image.

Alopecia areata in children can be particularly difficult. If a parent feels there is a significant change in a child's needs (withdrawn, low self-esteem, failing to achieve at school, change in behaviour), referral to a paediatric clinical psychologist, educational psychologist or social worker may be needed.

It is important to consider both the positive and negative aspects of active treatment in this chronic condition. Some patients do respond well to treatment. However, treatment can be uncomfortable for the patient, time-consuming and can be associated with undesirable side-effects. It may also alter the patient's attitude to their hair loss. Some patients find it difficult to cope with relapse following or during initially successful treatment and they should be forewarned of this possibility. These considerations are particularly important in children where the social disruption and focusing of the child's attention on their hair loss, which may result from active treatment, have to be carefully weighed against the potential benefits. On the other hand, some patients are appreciative that something has been tried, even if it does not work.

An individual's reaction to alopecia will vary depending on their own perceptions of body image, self-esteem, coping strategies, personality traits and their social support network. Commonly, people may feel self-conscious, conspicuous, angry, rejected, embarrassed or different and they may behave in a shy, cautious, aggressive, retreating, evasive or defensive (SCARED) manner.[21] It is important to mention self-acceptance particularly in those with long-standing, extensive and persistent alopecia areata.

9.2 Treatment

A number of treatments can induce hair growth in alopecia areata but none has been shown to alter the long-term course of the disease. The high rate of spontaneous remission makes it difficult to assess efficacy, particularly in mild forms of the disease. Some trials have been limited to patients with severe alopecia areata where spontaneous remission is unlikely. However, these patients tend to be resistant to all forms of treatment and the failure of a treatment in this setting does not exclude efficacy in mild alopecia areata. There are numerous case reports and uncontrolled case series claiming response of alopecia areata to diverse treatments. However, few treatments have been subjected to RCTs and, except for contact immunotherapy, there are few published data on long-term outcomes. A Cochrane review of 17 RCTs in alopecia areata concluded that only one trial (of topical steroid) gave evidence of short-term benefit and none showed long-term benefit.[22] However, the review did not consider contact immunotherapy or intralesional corticosteroid treatment due to the absence of RCTs for these modalities.

9.2.1 No treatment

Leaving alopecia areata untreated is a legitimate option for many patients. Spontaneous remission occurs in up to 80% of patients with limited patchy hair loss of short duration (< 1 year).[3] Such patients may be managed by reassurance alone, with advice that regrowth cannot be expected within 3 months of the development of any individual patch. The prognosis in longstanding extensive alopecia is poor and a wig may be a better option in such patients than indulging in treatments that are unlikely to be effective in this group.

9.2.2 Corticosteroids

For the definition of levels of evidence see Appendix 1.

Topical corticosteroids (level of evidence 2+) Very potent topical steroids are widely used to treat alopecia areata but the evidence for their effectiveness is limited.

In a RCT of 0·25% desoximetasone cream in 70 patients with patchy alopecia areata, more patients treated with the corticosteroid experienced at least minor improvement, compared with placebo, but the result failed to reach statistical significance.[23]

In a trial of 0·05% clobetasol propionate foam, 34 patients with moderate to severe alopecia areata were randomly assigned to treatment to one side of the scalp and vehicle to the other side. After 12 weeks of treatment, more sites treated with clobetasol had at least 50% regrowth of hair (seven of 34 vs. one of 34).[24]

Clobetasol propionate applied under an occlusive dressing may be effective in some patients. In a study of 28 patients who had AT/AU for a mean duration of 7 years, 0·05% clobetasol propionate ointment applied under an occlusive plastic film on six out of seven nights for 6 months resulted in long-term hair regrowth in five patients (18%).[25] The study initially had patients use the treatment on only one side of the scalp, and no hair regrowth occurred on the untreated side.

Folliculitis is a common side-effect of treatment with potent topical steroids.

Intralesional corticosteroids (level of evidence 3) Depot corticosteroid injected intralesionally stimulates hair regrowth at the site of injection in some patients. Porter and Burton[26] reported that tufts of hair grew in 33 out of 34 sites injected with triamcinolone hexacetonide in 11 patients with alopecia areata, and in 16 of 25 sites injected with triamcinolone acetonide in 17 patients. The effect lasted about 9 months. In a study from Saudi Arabia, 62% of patients achieved full regrowth with monthly injections of triamcinolone acetonide, the response being better in those with fewer than five patches of < 3 cm in diameter.[27] This method is most suitable for treating patchy hair loss of limited extent and for cosmetically sensitive sites such as the eyebrows. Hydrocortisone acetate (25 mg mL^{-1}) and triamcinolone acetonide (5–10 mg mL^{-1}) are commonly used. Corticosteroid is injected just beneath the dermis in the upper subcutis. An injection of 0·05–0·1 mL will produce a tuft of hair growth about 0·5 cm in diameter. Multiple injections may be given, the main limitation being patient discomfort. Intralesional corticosteroids may also be administered by a needleless device (e.g. Dermajet™; Dermajet UK, Crawley, U.K.). The device should be sterilized between patients. Abell and Munro[28] reported that 52 of 84 patients (62%) showed regrowth of hair at 12 weeks after three injections of triamcinolone acetonide using the Porto Jet needleless device compared with one of 15 (7%) control subjects injected with isotonic saline. The results were less favourable in alopecia totalis than in localized alopecia. Skin atrophy at the site of injection is a consistent side-effect of intralesional steroid therapy, particularly if triamcinolone is used, but this usually resolves after a few months. Repeated injection at the same site or the use of higher concentrations of triamcinolone should be avoided as this may cause prolonged skin atrophy. There is a risk of cataract and raised intraocular pressure if intralesional corticosteroids are used close to the eye, e.g. for treating eyebrows.[29] There are two case reports of anaphylaxis in patients receiving intralesional triamcinolone acetonide for treatment of alopecia areata.[30,31] Intralesional corticosteroids are not appropriate in rapidly progressive alopecia or in extensive disease.

Systemic corticosteroids (level of evidence 3) Long-term daily treatment with oral corticosteroids will produce regrowth of hair in some patients. One small partly controlled study reported that 30–47% of patients treated with a 6-week tapering course of oral prednisolone (starting at 40 mg daily) showed more than 25% hair regrowth.[32] Unfortunately, in most patients, continued treatment is needed to maintain hair growth and the response is usually insufficient to justify the risks.[33] There are several published case series of high-dose pulsed corticosteroid treatment employing different oral and intravenous regimens (intravenous prednisolone 2 g,[34] intravenous methylprednisolone 250 mg twice daily for 3 days,[35,36] oral prednisolone 300 mg once monthly,[37] dexamethasone 5 mg twice weekly[38]). The differences in treatment protocols and patient selection make it difficult to compare these studies directly; overall, about 60% of patients with extensive patchy alopecia appeared to show a cosmetically worthwhile response to pulsed corticosteroids whereas fewer than 10% of those with ophiasiform disease and AT/AU responded. In the only controlled trial, 43 patients were treated with oral prednisolone 200 mg or placebo once weekly for 3 months.[39] Patients receiving prednisolone showed better hair regrowth at 6 months, but this was not statistically significant. There is little published information on long-term outcomes. In a small case series of 12 children with severe alopecia areata who were treated with bolus high-dose methylprednisolone, the long-term outcome (median follow-up 42 months) was poor despite a good or moderate response in 10 children at 1 month after treatment.[40]

Significant side-effects have not yet been reported with pulsed administration of systemic corticosteroids in alopecia areata. However, short- and long-term side-effects of systemic corticosteroids are well known and potentially severe, and in view of these dangers it is not possible to support their use until there is better evidence of efficacy.

9·2·3 Contact immunotherapy (*level of evidence 2++*)

Contact immunotherapy was introduced by Rosenberg and Drake in 1976.[41] The contact allergens that have been used in the treatment of alopecia areata include: 1-chloro,2,4,dinitrobenzene (DNCB); squaric acid dibutylester (SADBE); and 2,3-diphenylcyclopropenone (DPCP).

DNCB fell from favour when it was found to be mutagenic against *Salmonella typhimurium* in the Ames test.[42] Neither SADBE nor DPCP are mutagenic. One DPCP precursor is mutagenic,[43] and batches should be screened for contaminants by the supplier. DPCP is more stable in solution and is usually the agent of choice.

The protocol for contact immunotherapy using DPCP was described by Happle *et al.*[44] The patient is sensitized using a 2% solution of DPCP applied to a small area of the scalp. Two weeks later the scalp is painted with a weak solution of DPCP, starting at 0·001%, and this is repeated at weekly intervals. The concentration is increased at each treatment until a mild dermatitis reaction is obtained. Some clinicians treat one side of the scalp initially to distinguish between a treatment response and spontaneous recovery if hair regrowth occurs. Once hair regrowth is observed, both sides of the scalp are treated. In patients with severe longstanding alopecia, where spontaneous recovery is unusual, this precaution is unnecessary. Opinions are divided on whether patients should be allowed to treat themselves.

Once a maximum response is achieved most practitioners reduce the frequency of treatment. In patients where full regrowth of hair is obtained treatment can be discontinued. Subsequent relapses will usually respond to further contact immunotherapy although this cannot be guaranteed.

A review of all the published studies of contact immunotherapy concluded that 50–60% of patients achieve a worthwhile response but the range of response rates was very wide (9–87%).[45] Patients with extensive hair loss are less likely to respond.[46,47] Other reported adverse prognostic features include the presence of nail changes, early onset and a positive family history.[45] In most studies, treatment has been discontinued after 6 months if no response is obtained. In a large case series from Canada, clinically significant regrowth occurred in about 30% of patients after 6 months of treatment but this increased to 78% after 32 months of treatment, suggesting that more prolonged treatment is worthwhile.[48] The response in patients with AT/AU was less favourable at 17% and this was not improved by treatment beyond 9 months. Relapses may occur following or during treatment. In the Canadian series, relapse following successful treatment occurred in 62% of patients.

Two case report series of contact immunotherapy in children with alopecia areata reported response rates of 33%[49] and 32%.[50] A third study found a similar short-term response in children with severe alopecia areata but < 10% experienced sustained benefit.[51]

Adverse effects Most patients will develop occipital and/or cervical lymphadenopathy during contact immunotherapy. This is usually temporary but may persist throughout the treatment period. Severe dermatitis is the most common adverse effect but the risk can be minimized by careful titration of the concentration. Uncommon adverse effects include urticaria,[52] which may be severe[53] and vitiligo.[54,55] Cosmetically disabling pigmentary complications, both hyper- and hypopigmentation (including vitiligo), may occur if contact immunotherapy is used in patients with pigmented skin. Such patients should be warned of this risk before embarking on treatment. Contact immunotherapy has been in use for 30 years and no long-term side-effects have been reported.

Precautions Contact immunotherapy is an unlicensed treatment that uses a nonpharmaceutical grade agent. Patients should (i) be fully informed about the nature of the treatment; (ii) be given an information sheet; and (iii) give signed consent. Great care must be taken to avoid contact with the allergen by handlers, including pharmacy, medical and nursing staff, and other members of the patient's family. Those applying the allergen should wear gloves and aprons. There are no data on the safety of contact immunotherapy during pregnancy and it should not be used in pregnant women nor in women intending to become pregnant.

DPCP is degraded by light. Solutions should be stored in the dark and patients should wear a hat or wig for 24 h following application.

9·2·4 Photochemotherapy: psoralen plus ultraviolet A (*level of evidence 3*)

There are several uncontrolled studies of psoralen plus ultraviolet A (PUVA) treatment for alopecia areata, using all types of PUVA (oral or topical psoralen, local or whole body UVA irradiation)[56–59] claiming success rates of up to 60–65%. Two retrospective reviews have reported low response rates[60] or suggested that the response was no better than the natural course of the disease,[61] although these observations were also uncontrolled. The relapse rate following treatment is high and continued treatment is usually needed to maintain hair growth, which may lead to an unacceptably high cumulative UVA dose.

9·2·5 Minoxidil (*level of evidence 2–*)

An early double-blind study reported a significantly greater frequency of hair regrowth in patchy alopecia areata in patients treated with topical 1% minoxidil compared with placebo.[62] Subsequent controlled trials in patients with extensive alopecia areata using 1% or 3% minoxidil failed to confirm these results.[63–65] Two of these studies reported a treatment response during an extended but uncontrolled part of the trial. In one study comparing 5% and 1% minoxidil in extensive alopecia areata, regrowth of hair occurred more frequently in those receiving 5% minoxidil but few subjects obtained a cosmetically worthwhile result.[66] Topical minoxidil is ineffective in AT/AU.

9·2·6 Dithranol (*level of evidence 3*)

There are a small number of case report series of dithranol (anthralin) or other irritants in the treatment of alopecia areata.[67–69] The lack of controls makes the response rates

difficult to evaluate but only a small proportion of patients seem to achieve cosmetically worthwhile results. In one open study, 18% of patients with extensive alopecia areata achieved cosmetically worthwhile hair regrowth.[67] The published data indicate that dithranol needs to be applied sufficiently frequently and in a high enough concentration to produce a brisk irritant reaction in order to be effective. Staining of hair limits its use in fair-haired individuals.

9·2·7 Calcineurin inhibitors (*level of evidence 3*)

The dual properties of ciclosporin as an immunosuppressive drug and as a hypertrichotic agent make it a logical choice in treating alopecia areata and this is supported by animal studies. Although there are only a small number of published uncontrolled trials with low patient numbers the evidence that ciclosporin does stimulate hair regrowth in some patients with alopecia areata is convincing.[70] However, as ciclosporin has to be given orally (it is not active topically), side-effects are a major consideration and, in patients with severe alopecia areata, the cosmetically worthwhile response rate is probably too low to justify the risks.[71] No response to treatment was seen in a case series of 11 patients with moderate to severe alopecia areata treated with topical tacrolimus for 24 weeks.[72]

9·2·8 Eyelash alopecia and prostaglandin $F_2\alpha$ analogues (*level of evidence 2–*)

Eyelash hypertrichosis is a side-effect of topical treatment of open-angle glaucoma with the prostaglandin $F_2\alpha$ analogues, latanoprost and bimatoprost. In a study of 40 patients with AU, 45% achieved complete or moderate regrowth of eyelashes when treated with topical latanoprost for 2 years compared with no regrowth in a nonrandomized control group.[73] However, a 16-week controlled study in 11 patients with eyelash alopecia showed no significant response to either latanoprost or bimatoprost,[74] and partial regrowth was seen in only a single patient in a 16-week controlled study of latanoprost in 26 patients.[75] A larger, more prolonged RCT is needed to resolve these conflicting results.

9·2·9 Biologic drugs (*level of evidence 3*)

The response of alopecia areata to biologic drugs has so far proved disappointing. Evidence to date indicates that antitumour necrosis factor (TNF) biologic drugs are ineffective. There are several reports of alopecia areata occurring in patients receiving anti-TNF biologic drugs for other conditions,[76] and in an open-label study in 17 patients with moderate to severe alopecia areata there was no response to treatment with etanercept.[77] In a RCT in 45 patients with chronic severe alopecia areata, there was no significant response to alefacept, an anti-T-cell biologic, compared with placebo.[78]

9·2·10 Miscellaneous treatments

Partial evidence of efficacy, either from uncontrolled case series or from single controlled or partially controlled trials, exists for a number of treatments.

Sulfasalazine (level of evidence 3) Several uncontrolled case series have claimed response to sulfasalazine.[79–81] In an uncontrolled study of 26 patients with severe alopecia areata (> 40% hair loss), 22 of whom completed the treatment, six showed complete recovery and a further nine had partial regrowth of hair. Partial or complete relapse occurred in 10 of the 15 responders.[82]

Methotrexate (level of evidence 3) In a retrospective review of 22 patients with AT/AU treated with methotrexate 15–25 mg per week with or without prednisolone 10–20 mg daily, 14 achieved complete regrowth of hair, including three of six patients treated with methotrexate alone.[83]

Isoprinosine® (level of evidence 2–) Isoprinosine® (Newport Pharmaceuticals, Swords, Ireland) is an old drug that has immunostimulatory and antiviral properties. Early uncontrolled studies of its use in alopecia areata reported mixed positive[84] and negative results.[85] A more recent RCT in 32 patients with recalcitrant alopecia areata reported complete remission at 12 weeks in 50% of patients taking Isoprinosine compared with none in the placebo control group.[86]

Laser therapy (level of evidence 3) An infrared diode laser was used to treat patchy alopecia areata in 16 patients. Complete or partial regrowth was seen in 32 of 34 treated patches, whereas no growth occurred in patches left untreated.[87] In 18 adults, 42 patches of alopecia areata were treated twice weekly for 12 weeks with a 308-nm excimer laser. Regrowth was seen in 17 patches.[88] Similar results (60% response rate) were observed in a study of excimer laser treatment in nine children with alopecia areata. Patches left untreated failed to regrow hair.[89]

Aromatherapy (level of evidence 3) In a nonrandomized double-blind trial, 19 out of 43 (44%) patients receiving aromatherapy showed a treatment response at 7 months, compared with six out of 41 (15%) patients in the control group.[90]

Hypnotherapy (level of evidence 3) In a nonrandomized trial comparing 20 patients (most with severe alopecia areata) treated by hypnotherapy with 21 untreated control patients, the treated group showed a significant reduction in scores for depression and anxiety, but there was no difference between groups in terms of hair regrowth.[91] Despite the negative influence on hair growth this study highlights the role of nonpharmacological treatments in helping patients with alopecia areata.

9·3 Wigs and prostheses (*level of evidence 4*)

Coping with the impact of alopecia areata depends on the individual's ability to deal with an altered body appearance and their

perceptions of themselves. When wearing prostheses, individuals often have an underlying fear of being discovered, particularly when discussions about hair arise from social conversation, as many do not feel at ease disclosing their condition.[92] Wigs, integrated systems, hairpieces, headscarves, hats, false eyelashes and semipermanent make-up can be used as effective ways to cope with alopecia areata. However, choosing to wear a hair prosthetic can be an overwhelming experience, due to the variety of different options and suppliers to choose from. Choice can be limited depending on an individual's financial circumstance as wigs range in price from £50 to £5000.

Synthetic acrylic wigs are the most affordable option. Monofilament acrylic wigs are constructed to give the appearance of hair growing from the scalp; they are light, look natural and come in a variety of colours, lengths and styles. However, all synthetic wigs become damaged near heat such as opening oven doors and patio heaters and, if worn daily, will need replacing every 3–4 months to maintain the appearance of the acrylic fibres in good condition and the illusion of hair.

Human hair wigs vary; quality depends on where the hair has been sourced and the construction of the cap, i.e. if the wig is presized or made to measure. Human hair looks very natural and will last longer if kept in good condition, typically 1–2 years. Manufacturing techniques in wig cap construction give some wigs the ability to stay in place while sleeping, exercising, swimming and showering. However, they are expensive and careful consideration is required when choosing a supplier, particularly when purchasing online or abroad. The U.K. National Health Service (NHS) policy on entitlement for a prescription for human hair wigs is only available to patients who are allergic to acrylic or who have a skin condition made worse by acrylic.

For individuals with patchy alopecia areata or thinning hair loss, there are options to integrate a weft of human hair or use a top piece that is either clipped into surrounding hair or braided into place.

Individuals with AT/AU find it particularly challenging when losing eyelashes and eyebrows, as a protective mechanism from foreign particles has been lost, as well as dealing with a dramatic altered appearance. False eyelashes can be synthetic or human hair and top lashes can easily be purchased. They can be tricky to apply, but with practice can become quick and easy to manage. Eyebrows can be drawn and voluntary organizations can help to teach how to apply and/or style an eyebrow shape. Eyebrow pencil ink has much improved and can be waterproof lasting up to 24 h, or 3 days depending on the manufacture. Also, fake eyebrows can be purchased that are self-adhesive and can stay on for up to 3 days. Alternatively, techniques in micropigmentation from paramedical cosmetic intervention for scar camouflage and areola reconstruction have led to the development of semipermanent tattooed eyebrows. The pigmentation longevity varies from person to person with a colour boost required at 12 and 24 months, but can last up to 3 years. Some pigmentation remains in the skin permanently but tends to fade over time.

NHS policy on prescribing of wigs and prescription charges is given in Appendix 2.

10·0 Recommended audit points

1 Record keeping. Records should include the age of onset, history of relapses, family history and other diseases; the severity and type of alopecia (patchy, total, universal or ophiasis) and the presence of nail changes should be recorded.
2 Outcome of consultation. What was the outcome of the consultation – was treatment instituted and information supplied to the patient?
3 Treatments. For specific treatments, including contact immunotherapy, PUVA and systemic steroids – what information was supplied, informed consent?

11·0 Summary

The details of evidence are given above. Alopecia areata is difficult to treat and few treatments have been assessed in RCTs. The tendency to spontaneous remission and the lack of adverse effects on general health are important considerations in management, and not treating is the best option in many cases. On the other hand, alopecia areata may cause considerable psychological and social disability and in some cases, particularly those seen in secondary care, it may be a chronic and persistent disease causing extensive or universal hair loss. In those cases where treatment is appropriate there is reasonable evidence to support the following (strength of recommendations are defined in Appendix 1):

Limited patchy hair loss
- Potent topical steroid (*strength of recommendation C*)
- Intralesional corticosteroid (*strength of recommendation C*)

Treatment with potent topical corticosteroids probably advances regrowth of hair in some patients with mild to moderate disease but there are no data on long-term outcomes.

Intralesional corticosteroids stimulate hair regrowth at the site of injection. The effect is temporary, lasting a few months, and it is unknown whether the long-term outcome is influenced.

Extensive patchy hair loss
- Contact immunotherapy (*strength of recommendation C*)
- Wig/hairpiece (*strength of recommendation D*)

Alopecia totalis/universalis
- Contact immunotherapy (*strength of recommendation C*)
- Wig (*strength of recommendation D*)

Contact immunotherapy is the best-documented treatment in severe alopecia areata but it is not widely available, involves multiple visits to hospital over several months and stimulates cosmetically worthwhile hair regrowth in < 50% of patients. It is the only treatment likely to be effective in AT/AU, although the response rate is low. It may cause trouble-

some temporary local inflammation but serious side-effects are rare.

Dithranol (anthralin) and minoxidil lotion are widely prescribed by dermatologists for limited patchy alopecia areata, and are safe, but there is no convincing evidence that they are effective.

Continuous or pulsed systemic corticosteroids and PUVA have also been used to treat alopecia areata. However, in view of the potentially serious side-effects and inadequate evidence of efficacy, none can be recommended at this time.

Children may be treated in a similar fashion to adults. However, intralesional steroids are often poorly tolerated and many clinicians are reluctant to use aggressive treatments such as contact immunotherapy in children.

12·0 Patient support

Information on patient support organizations can be found in the BAD Patient Information Leaflet on alopecia areata (http://www.bad.org.uk).

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Appendix 1

Strength of recommendation

Level of evidence	Type of evidence
1++	High-quality meta-analyses, systematic reviews of RCTs, or RCTs with a very low risk of bias
1+	Well-conducted meta-analyses, systematic reviews of RCTs, or RCTs with a low risk of bias
1–	Meta-analyses, systematic reviews of RCTs, or RCTs with a high risk of bias[a]
2++	High-quality systematic reviews of case–control or cohort studies High-quality case–control or cohort studies with a very low risk of confounding, bias or chance and a high probability that the relationship is causal
2+	Well-conducted case–control or cohort studies with a low risk of confounding, bias or chance and a moderate probability that the relationship is causal
2–	Case–control or cohort studies with a high risk of confounding, bias or chance and a significant risk that the relationship is not causal[a]
3	Nonanalytical studies (for example, case reports, case series)
4	Expert opinion, formal consensus

[a]Studies with a level of evidence '–' should not be used as a basis for making a recommendation. RCT, randomized controlled trial.

Levels of evidence

Class	Evidence
A	• At least one meta-analysis, systematic review, or RCT rated as 1++, and directly applicable to the target population, **or** • A systematic review of RCTs or a body of evidence consisting principally of studies rated as 1+, directly applicable to the target population and demonstrating overall consistency of results • Evidence drawn from a NICE technology appraisal
B	• A body of evidence including studies rated as 2++, directly applicable to the target population and demonstrating overall consistency of results, **or** • Extrapolated evidence from studies rated as 1++ or 1+
C	• A body of evidence including studies rated as 2+, directly applicable to the target population and demonstrating overall consistency of results, **or** • Extrapolated evidence from studies rated as 2++
D	• Evidence level 3 or 4, **or** • Extrapolated evidence from studies rated as 2+, **or** • Formal consensus
D (GPP)	• A good practice point (GPP) is a recommendation for best practice based on the experience of the guideline development group

RCT, randomized controlled trial; NICE, National Institute for Health and Clinical Excellence.

Appendix 2

NHS wig policy

The NHS can provide a limited entitlement to wigs. Information on prescription charges in England and entitlement to free wigs is provided in leaflet HC12, available at: http://www.nhs.uk/nhsengland/Healthcosts/pages/Wigsandfabricsupports.aspx. In Wales, Scotland and Northern Ireland wig prescriptions are free.

According to NHS policy, real hair wigs are only available to patients who are allergic to acrylic or who have a skin condition made worse by acrylic (http://www.nhs.uk/conditions/hair-loss/pages/treatment.aspx).

Supporting information

Additional supporting information is available in the online version of this article.

Appendix S1 Literature search strategies.

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