



Alopecia Areata

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Abstract

Alopecia Areata (AA) is a common non-scarring alopecia that usually presents as well circumscribed patches of sudden hair loss that affects 0.1-0.2% of the population. The aetiology is thought to be both genetic and autoimmune in nature. 139 single nucleotide polymorphisms have been identified in 8 regions of the genome and are found to be associated with T cells or the hair follicle. Furthermore, patients with AA have been found to have an increased frequency of hair follicle-specific auto-antibodies. The diagnosis of AA is usually made on clinical grounds, and further investigations are not usually indicated. Intralesional corticosteroids remain the treatment of choice. Systemic steroids are also highly effective; however side effects make them less desirable to both patients and physicians. Other treatment options available include anthralin, minoxidil, topical immunotherapy and these treatments will be discussed further in depth in this chapter. The morbidity of AA is largely psychological; therefore the successful treatment of AA should include focusing on the improvement of the psychological impact of this condition.

Keywords: Auto immunity; Alopecia areata; Hair loss; Non-scarring alopecia

Epidemiology

Alopecia Areata (AA) is a common non-scarring alopecia that affects 0.1-0.2% of the general population and accounts for 0.7-3% of all cases seen in dermatology practice [1,2]. This inflammatory disease manifests in the hair follicles and can also affect the nails in up to 66% of patients [3]. AA occurs in all races and both genders equally although it is thought that there may be a male preponderance in the adult population [4]. Prevalence rates peak between the ages of twenty to forty years of life. However, up to 60% of patients with AA will present with their first patch before the age of twenty years [5,6].

Aetiology

AA is most likely an organ-specific autoimmune disease. Gene association studies confirm a genetic predisposition. Environmental triggers have been postulated, but none have been confirmed.

Genetics

Family studies in AA have found that 28% of patients have at least one affected family member and the AA concordance rate in monozygotic twins ranges between 42 & 55% [7,8].

139 single nucleotide polymorphisms have been identified in 8 regions of the genome [9]. These polymorphisms (IL2/IL21, IL2RA, CTLA4, IK2F4, HLA, NK-activating ligands, ILBP6, ULBP6, STX17, PRDX5) were found to be associated with T cells or the hair follicle. In addition, there was a region of strong association on the cytomegalovirus UL16-Binding Protein Gene Cluster (ULBP) [9]. This gene cluster encodes the activating ligands of natural killer cell receptor NKG2D. ULBP3 expression was found to be unregulated in lesions of AA, which has been implicated in triggering autoimmunity.

Autoimmunity

Autoimmunity is believed to play a central role in the development of AA. Patients with AA have been found to have an increased frequency of hair follicle-specific auto-antibodies [10]. The inferior portion of the normal hair follicle is an "immune privileged" site which is protected from surveillance by T cells [11]. Major Histocompatibility Complex (MHC) class I and II, molecules that bind and present pathogens to the immune system, are not expressed in normal hair follicle epithelium.

In AA, however, this immune privileged site is compromised. There is an increased MHC I and II complexes and increased expression of adhesion molecules ICAM-2 and ELAM-1 surrounding the perivascular and peribulbar hair follicular epithelium [12].

Clinical Presentation

Clinically, AA presents as an area of well-circumscribed patch of sudden hair loss. Patches up to 2 cm in diameter may appear overnight and then extend circumferentially at a rate of around 1 cm per week. The initial loss is by hair breakage, close to or just below the surface of the skin. Hair remnants are visible as black dots on dermoscopy. While any hair-bearing area may be affected, 90% of lesions occur on the scalp [5,7]. The skin over the affected areas may have no overt epidermal changes except for some mild erythema and atrophy.

Exclamation mark hairs, which are dystrophic hairs with fractured tips, are commonly seen around the margins of the hair loss patch. Less commonly, AA can also present as diffuse hair loss. In this situation, other differential diagnoses such as female pattern hair loss, telogen effluvium and trichotillomania will need to be considered.

AA can be divided into a number of classifications based on the extent of hair loss or variations of presentations (Table 1).

Nail changes

Nail changes can occur in 10-66% of patients with AA [3]. Presentation may be limited to 1 or more nails. Nail changes occur more commonly in children (12%) compared to adults (3.3%) and in more severe forms of AA [13]. Although the presence of nail disease confers a poorer prognosis, the severity of nail disease does not correlate temporally with the severity of hair loss.

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Type	Description
Patchy AA	This is the most common presentation of AA. Hair loss is localized and patchy
AA totalis	Total hair loss on the scalp
AA universalis	Complete hair loss of all hair-bearing areas
Acute diffuse and total alopecia (ADTA)	Diffuse hair shedding with a positive hair pull test that continues for weeks to months but with few or no discrete patches of hair loss. Chronic diffuse alopecia areata can mimic telogen effluvium
Diffuse AA	Diffuse hair shedding with a positive hair pull test that continues for weeks to months but with few or no discrete patches of hair loss. Chronic diffuse alopecia areata can mimic telogen effluvium
Ophiasis	Hair loss is localised to the sides of the scalp and the nape
Sisapho	Hair loss spares the sides of the scalp and the nape, and usually affects the central scalp. This presentation is rare
Reticular patches	Several patches of AA may coalesce to form a reticular pattern

Table 1: Classifications of Alopecia Areata.

Differential Diagnoses

There are multiple disease entities which resemble AA (Table 2). Tinea capitis and trichotillomania both produce hair breakage and patchy hair loss. In tinea capitis, the scalp skin may be scaly. In trichotillomania, the border of the alopecia is angular rather than circular. On the upper eyelashes these 2 conditions may be impossible to distinguish. The clinician will need to rely on the natural evolution of the alopecia to make a diagnosis. However, TTM does not affect the lower eyelashes whereas these can be affected by AA. Therefore, this is a useful distinguishing feature when the lower eyelashes are affected.

Nail presentations include superficial pitting (the commonest change), trachyonychia (sand paper nails), leukonychia and Beau's lines. In severe disease, red lunulae can be seen.

Physical Examination

The diagnosis of AA is made on clinical grounds and a good physical examination is imperative to distinguish between the differentials. Nail pitting or trachyonychia are commonly seen. Beau's lines half way down the nail can be observed in severe AA. Dermatoscopic examination can provide additional useful information including broken hairs, yellow dots and exclamation mark hairs.

Investigations

Routine tests are not indicated. If thyroid disease is suspected on clinical grounds then thyroid function should be measured. If there is uncertainty regarding the diagnosis a scalp biopsy may be performed. If there is a suspicion for other differentials, tests such as fungal culture, lupus serology and syphilis serology should be undertaken.

Histopathology

The histopathological features vary depending on the stage of the disease. The histopathological changes of AA can be divided into 4 stages: acute, subacute, and chronic and recovery (Table 3).

Management

Psychological support

The morbidity of alopecia areata is predominantly psychological rather than physical. Alopecia has profound effects on quality of life and social functioning. Often patients are physically healthy, but may suffer from considerable psychological sequelae. AA may affect the patient's self image including functioning, emotions, self-confidence and stigmatization. AA has been found to be associated with anxiety and depression which can in turn impact the patient's physical health [14]. It is important to take into account the different coping strategies each patient may have in order to assist the depth of psychological support needed to adjust to the illness.

Successful treatment of AA should include focusing on the improvement of the psychological impact. Clinicians should ensure that patients have adequate social support. A number of support services exist in the community and aim to educate and increase awareness of AA. The psychological impact of alopecia on adolescent boys in particular should not be underestimated. Even mild disease is difficult to conceal with the shorter hair styles popular today. School avoidance and social isolation may aggravate the situation and suicide is reported separately in this edition.

While spontaneous remission will occur in up to 50% of patients in 1 year and 80% after 2 years, most people seek active treatment [15]. Also the natural evolution of alopecia areata, with multi-ocular disease evolving over a period of months rather than occurring simultaneously, suggests that the presence of active disease could increase the risk of subsequent patches. While this contention is unproven, the authors favour active treatment.

Pharmacological management

High dose oral corticosteroids are highly effective in regrowing hair and preventing relapse, however the associated side effects make many patients unwilling to take them and many physician reluctant to prescribe them. To date there are no curative therapies available for the treatment of AA. Pharmacological treatments currently aim to arrest the progression of disease and regrowing bald patches rather than changing the course of disease or preventing new patches from appearing. Multiple topical, local and systemic treatment options exist, many of which are immunosuppressants or immune-modulators.

Intralesional corticosteroids: Intralesional corticosteroids are the treatment of choice for patchy hair loss in AA. Triamcinolone acetonide (5-10 mg/mL) is our favoured intralesional corticosteroid. The authors' practice is to inject 0.1 mL of solution is injected into multiple sites 0.5 cm apart into the deep dermis using a 30 gauge needle. This process is repeated every 4-6 weeks. To minimize the risk of dermal atrophy, 5 mg/mL dosage is recommended as the initial concentration for the scalp and 2.5 mg/ml for areas on the face. No more than 20 ml should be injected for each visit. To minimize the discomfort of injections, a vibrating device placed around the area of treatment can be used as distraction, and the triamcinolone may be diluted with lignocaine. If there is no improvement over the course of 6 months, or significant atrophy develops, the intralesional corticosteroids should be ceased.

The most common side effect is atrophy of the skin which can be prevented by using smaller volumes or concentrations at each injection site. Other side effects include hypopigmentation, telangiectasia and atrophy. In addition, injections near the eyebrow carry a theoretical risk of cataracts and raised intracranial pressure. With the aim of preventing diffusion into the intraocular region, some injectors push the eyebrow superiorly against the bone above the supraorbital rim

Differential	Presentation and Distinguishing Features
Tinea capitis	The diagnosis of tinea capitis should be considered particularly in children. Scaling with or without scalp erythema, and broken hairs are often present on physical examination
Trichotillomania	Trichotillomania may co-exist with AA. The parieto-temporal scalp is usually affected in a bizarre or geometric-shaped pattern. Other features which can be noted include: - Broken hairs that may vary in length - The scalp surface may not appear smooth due to the variation of length in broken hairs - Affected patches may have a 'wire brush' feeling
Discoid Lupus Erythematosus (DLE)	DLE presents as type of scarring patchy hair loss associated with pruritus and burning. There may be follicular plugging (hyperkeratosis) within the patch. In later stages of the disease, there is usually scarring, atrophy and dyspigmentation.
Lichen planopilaris	Lichen planopilaris should be considered in women between the ages of 30 to 60. It typically starts on the central scalp and may be associated with pruritus, burning and pain. There may be peri follicular hyperkeratosis and erythema present.
Central centrifugal cicatricial alopecia	Cicatricial alopecia is characterised by patchy hair loss with loss of follicular orifices. Erythema, scaling, pustules and plugging may occur.
Congenital triangular alopecia	Often appears after 2 years of age and rarely occurs in adulthood. A scalp biopsy may be needed to distinguish between AA and triangular alopecia. Histology reveals a normal number of hair follicles which are vellus or indeterminate.
Telogen effluvium	Telogen effluvium (TE) is the main differential diagnosis for diffuse AA. TE is often associated with a precipitant including recent illness, surgery, weight loss or medications. TE can be difficult to differentiate from AA, but the history of a precipitating event 2-3 months prior may point towards this diagnosis. A scalp biopsy may be required to help with the diagnosis.
Systemic lupus erythematosus	Hair loss is not uncommon in SLE; however hair loss involving greater than 50% of the scalp area is rarely reported.
Loose anagen hair syndrome	Mainly occurs in young girls with blonde or light brown hair between the ages of 2-6. Hair often appears lustreless. The occiput is usually affected due to repeated friction of the patient's head against a pillow during sleep.
Secondary syphilis	Presents commonly as a moth eaten pattern but may also present as diffuse hair loss. May be present with mucosal or cutaneous lesions

Table 2: Differential diagnosis of Alopecia Areata.

Stage	Histological Appearance
Acute stage	Terminal hairs are surrounded by bulbar lymphocytes, which can take the classical (but not commonly observed) appearance of a 'swarm of bees' [3]
Subacute stage	There is an increase in catagen and telogen hairs and a decrease in anagen hairs
Chronic stage	There are decreased terminal and increased miniaturized hairs. Inflammation may be variable. Immunofluorescence studies have shown deposits of C3, IgG and IgM on the basement membrane of the inferior part of the hair follicle
Recovery stage	The number of terminal anagen hairs increase and there is a lack of inflammation

Table 3: Histological Stages of Alopecia Areata.

for eyebrow injections. Dermal atrophy usually recovers over 3 to 6 months, provided there is no further injection into the site.

Systemic corticosteroids: Systemic corticosteroids are highly effective in the treatment of AA. Short-term use of up to 3 months is generally well tolerated. The chronic nature of the alopecia areata may encourage the chronic use of corticosteroids. Clinicians should strongly consider commencing vitamin D and calcium supplements in patients at risk of osteopenia and in those who require prolonged therapy. The use of long-term systemic corticosteroids is usually not justified due to side effects and the high relapse rate, except in severe cases. For treatment naïve patients a high starting dose is used and tapered by 30% every 2 to 3 weeks. Prednisolone may be given at a starting dosage of 0.5 mg/kg/day i.e., in most patients this would be approximately 37.5-50 mg/day. Higher doses might be associated with aseptic necrosis of the femoral head and this complication should be considered in a patient complaining of hip pain on high dose systemic corticosteroids. For patients who have previously responded to oral corticosteroids, lower starting doses are used for relapse episodes. A useful guide for dosing is to determine the relapse dose from a previous tapering course. For example, if a patient had previously regrown hair with prednisolone therapy initiated at 40 mg/day and tapering over the ensuing few weeks had relapsed once the dose was reduced below 15 mg/day, it would be appropriate to restart prednisolone therapy for a subsequent relapse at 20 mg/day.

Use of corticosteroids, particularly at high doses used over a long period of time, carries a risk of suppressing the hypothalamic-pituitary-adrenal axis which in turn may cause variations of blood pressure and may increase blood sugar. In addition, patients are at

risk of developing conditions like psychosis, cataract, glaucoma, peptic ulcer disease or osteoporosis. Pulse corticosteroid therapy has been trialled in an attempt to minimize the risk of steroid related side-effects and there have been reports of successful therapy with 250 mg IV methylprednisolone for 3 days in patchy AA [16,17].

Topical corticosteroids: Topical corticosteroids are commonly used to treat AA especially in children who are unable to tolerate the discomfort of injections. The authors do not routinely use topical corticosteroids to treat AA in adults due to their limited efficacy. The levels of efficacy vary depending on the strength of the preparation. Treatment with topical corticosteroids needs to be continued for at least 3 months before regrowth is seen. Common adverse reactions to topical steroids include atrophy of skin, telangiectasia and folliculitis. More success is seen in children than in adults. Children responders commonly develop hypertrichosis of the forehead and face.

Psoralen and ultraviolet A (PUVA): Psoralen and ultraviolet A light (PUVA) deplete inflammatory cell infiltrate and can produce satisfactory regrowth in patients with AA totalis and AA universalis with relatively low relapse rates (21% of participants studied over five years) [18]. The most common complaints are erythema and burning of skin.

Minoxidil: Minoxidil is an effective topical preparation that stimulates proliferation at the base of the bulb and differentiation above the dermal papilla [3]. A concentration of 2-5% minoxidil is used twice daily as a topical application to the affected area with good results in patient with mild to moderate AA. The authors use oral minoxidil at a dose of 5 mg daily and find this to be a useful adjunct treatment for more extensive AA [19]. Minoxidil can maintain affected hairs

in the anagen phase to prevent transition into the telogen shedding phase, and at the same time is also a good hair growth stimulant and can thicken up surrounding normal hairs to camouflage the sparse AA affected areas. This treatment is limited by a small risk of hypertrichosis or worsening of hirsutism.

Topical immunotherapy: Topical immunotherapy may be useful for severe and refractory cases of AA especially in children whom treatment options are more limited [20]. It is hypothesized that the allergic reaction created by the topical immunotherapy generates suppressor T cells that inhibits an autoimmune reaction against the hair follicle [20]. Initially patients are sensitized by applying 2% DPCP in acetone or paraffin to the inner arm or a small area on the scalp under waterproof occlusion for 48 hours. After a fortnight, initiation of treatment with a 0.001-0.005% solution is applied on the scalp and this concentration is increased up to a maximum of 2% concentration over weeks or a few months, aiming for a mild allergic contact dermatitis on the areas of application i.e. erythema, pruritus and mild scaling that lasts for 24-48 hours. Weeping and vesiculation indicated the concentration is too high and should be reduced. Postauricular lymphadenopathy may be painful but indicates a better prognosis for regrowth. This treatment has a success rate of only 50-60% and often has unpredictable results, therefore is not commonly used as first line. Treatment should be ceased after 6 months of unsuccessful therapy.

Anthralin: The exact mechanism of action of anthralin (dithranol) is used in some countries including Australia) is largely unknown however it is thought to have immunosuppressive and anti-inflammatory properties via the generation of free radicals [21]. It is also a skin irritant and may work in a method analogous to topical immunotherapy. 0.5-1% of anthralin cream is applied to the affected areas for a period of 20-30 minutes daily. Over the first 2 weeks of therapy exposure can be build up gradually until erythema and pruritis develop. Common side effects include scaly skin, folliculitis and regional lymphadenopathy which can be easily reversed by withholding the application for several days. This treatment is not commonly used due to limited evidence of efficacy and its unpleasant staining properties on linen and clothing.

Sulfasalazine: Sulfasalazine via the inhibition of inflammatory cells chemotaxis and cytokine antibody production has been reported to produce satisfactory results [22]. Hair regrowth rates were found to be 27.3% of complete hair regrowth and 40.9% of partial hair regrowth in AA patients [23]. Recommended dosages are 0.5 grams twice daily for the first month, then 1 gram twice daily for the second month, 1.5 grams for another three months. Its side effect profile includes gastrointestinal distress, headaches, fevers, rash and haematological abnormalities. In the authors experience this agent is universally disappointing.

Cyclosporine: Cyclosporine can be used alone or in combination with systemic corticosteroids. Its mechanism of action is via inhibition of T cell activation and via depletion of perifollicular lymphocytic infiltrates [24]. Treatment responses have been inconsistent with results ranging from from 25-88% [25]. Additionally, AA has been found to paradoxically occur in transplant patients using high doses of cyclosporine [26]. Cyclosporin also prolongs the anagen phase of the hair growth cycle causing hypertrichosis as a cutaneous side effect. Other side effects include gingival hyperplasia, nephrotoxicity, hepatotoxicity, headaches and hyperlipidemia.

Calcineurin inhibitors: Calcineurin inhibitors work by inhibiting the transcription of cytokines and tumour necrosis factor. There have been conflicting results surrounding the efficacy of topical calcineurin inhibitors. One study found that tacrolimus was effective in inducing

hair growth in mice while another revealed that no patients responded to tacrolimus over the course of 24 weeks [27,28].

Biologic drugs: There are a number of studies examining biologic therapies; however, to date overall results are disappointing. A study using 50 mg of etanercept twice a week for 24 weeks showed no significant hair regrowth [29]. In addition, a case was reported to have occurred in a patient treated with etanercept [30]. A randomised clinical trial examining 45 patients with chronic severe alopecia showed no significant response to alfacept compared to the placebo [31]. Abetacept, a fusion protein that is comprised of the Fc region of the immunoglobulin IgG1 and fused to the extracellular domain of CTLA-4, has shown promise in murine models of alopecia areata and is currently under investigation in the treatment of human alopecia areata.

Treating eyelash alopecia

Topical corticosteroids are rarely used around the eye for fear of cataract and glaucoma. Prostaglandin F_{2α} analogues such as latanoprost and bimatoprost have recently been FDA-approved for use to improve the appearance of lashes by lengthening and increasing the volume of eyelash hairs. These treatments are typically used for open-angle glaucoma however eyelash hypertrichosis was noted as a pleasing side effect. Response rates have been variable with one study showing 45% complete or moderate regrowth of eyelashes over 2 years using latanoprost whilst another study showed no significant response to latanoprost or bimatoprost over 16 weeks [32,33]. Clinicians should recommend protective glasses to patients with extensive and complete loss of eyelashes to protect the eyes from dust, grit and debris.

Wigs and prosthesis

The prognosis of extensive and long standing AA is poor and wigs or prosthesis can be an effective method of coping with the condition. Monofilament acrylic wigs can be constructed to give the appearance of hair growing from the scalp. Synthetic wigs need replacing every 6-12 months. Human hair wigs, in contrast, can last 3-5 years if maintained properly.

Prognosis

The prognosis for most patients with limited AA is good given the tendency for spontaneous remission. Patients with long standing extensive AA have a poorer prognosis. In 14-25% of cases AA will progress to alopecia totalis or alopecia universalis [34]. Progression to this stage rarely results in full recovery.

The greatest predictor of long-term outcome in AA is the severity of disease at presentation. Other poor prognostic factors include a positive family history, a long clinical duration of alopecia, and presence of nail disease, presence of other autoimmune diseases and a young age of onset. Disease prognosis is also poor in ophiasis and cases where AA develops during childhood.

Summary

In summary AA is a common non-scarring alopecia which can affect patients of all ages and races. Although the cause of AA is poorly understood there is a large body of evidence to suggest that genetics and autoimmunity underpin the pathogenesis. The role of immune mediated molecules surrounding the hair follicle has been examined in various mice and rat models and its significance continues to be investigated as a target of existing and novel treatments.

The most reliable treatment is corticosteroids either orally,

intralesionally or topically. For patients who fail to respond to corticosteroids, or who are intolerant of or unwilling to take corticosteroids the treatments options are many and varied, but the response rates are low.

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