

Review Article

Alopecia Areata – A literature Review

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ABSTRACT

Alopecia areata (AA) is a disease marked by extreme variability in hair loss, not only at the time of initial onset of hair loss but in the duration, extent and pattern of hair loss during any given episode. This variable and unpredictable nature of spontaneous re-growth and lack of a uniform response to various therapies has made clinical trials in alopecia areata difficult to plan and implement. It is a type of alopecia that affects males and females equally. It occurs in both children and adults. The peak age of occurrence is 20 to 50 years. The most common clinical presentation is asymptomatic shedding of telogen hairs followed by patchy non scarring hair loss in association with nail pitting, Beau's line and nail dystrophy. The disease may progress from this limited presentation to total loss of all scalp hairs (Alopecia totalis) or all body hair (alopecia universalis) with significant onychodystrophy. Mostly it is characterised by reversible hair loss involving the scalp although others areas of head including eyelashes, eyebrows and beard may also be affected. Although, it is a mostly cosmetic problem but it often has devastating effects on quality of life and self-esteem. The paper aims at providing an overview of Alopecia areata.

Key words: Alopecia areata, totalis, universalis, onychodystrophy, telogen

Section – Dermatology

INTRODUCTION

Alopecia areata (AA) is a type of patterned hair loss marked by a focal inflammatory infiltrate of hair follicles by lymphocytes and sometimes changes in nails.^[1] Although AA is thought to be an autoimmune disorder but definite proof for this is lacking. AA affects men, women, and children. It is characterised by reversible hair loss most commonly involving the scalp although other parts of head, including eyelashes and beard, may also be affected. There may be a few small patches of hair loss or the whole scalp may be affected. Hair loss in some areas may coexist with re-growth in others. A few patients lose all the hair from

their heads (Alopecia totalis)^[2] or all body hairs (Alopecia universalis).^[3] AA is a non-scarring type of Alopecia.^[4] It may affect only the hair margins (Ophiasis). Clinically it has been observed in some studies that AA is sometimes characterised by asymptomatic nodules, located mostly on the vertex and the upper part of occipital area.^[5]

AETIOLOGY

There are various aetiological factors responsible for AA. Some of them are as follows;
Genetic factors; the importance of genetic factors in alopecia areata is indicated by high frequency of a positive family history in affected individuals.^[6,7] The life time risk of children of affected parents is approximately 6%.^[8] AA has been reported in identical twins. Jackow et al found 55% concordance rate in identical twins.^[9] A significant association with HLA class I (HLA-A,-B,-C) and class II (HLA -DR,-DQ,-DP) antigens have been studied in alopecia areata.^[10] A genome-wide scan performed on 20 families confirmed the linkage between alopecia areata and the MHC region on chromosome 6p.^[11]

Autoimmunity; Alopecia areata was at first thought to be an autoimmune disorder by Rothman following a paper presented by Van Scott.^[12] Alopecia areata have been found to be associated with other autoimmune diseases such as

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atopy, autoimmune thyroid disease and vitiligo.^[13] AA has been seen associated with type 1 Diabetes Mellitus in relatives of patients.^[14] Other associations are pernicious anaemia, lupus erythematosus, myasthenia gravis, rheumatoid arthritis, Polymyalgia rheumatica, ulcerative colitis, lichen planus, and Candida endocrinopathy syndromes.^[15] The association of Lichen planus was also reported by Brown *et al.*^[16]

Emotional stress; Stress may be a precipitating factor in some cases of AA.^[17] Alopecia patients often have a history of long standing emotional disturbances. This is not contradictory to an immunologic view of pathogenesis, as psychological stress can alter the immune function.^[18]

Infections; Sporadic reports of correlating alopecia areata with infective agents continue to appear.^[1] Viral infection has been suggested as a major inducer of hair loss in AA. Cytomegalovirus and Epstein-Barr virus has been reported to trigger AA, although there have been many controversial opinions. Recently in a case study AA was reported to be exacerbated by swine flu virus infection.^[19]

Pathogenesis;

T-lymphocytes mediate the perifollicular immunological milieu by triggering a cascade of events via cytokine production.

Antigen presentation of responsible epitopes helps drive the condition. The exact epitope may be in the follicular keratinocytes, melanocytes, or dermal papilla.^[18]

Pathology;

Four stages have been noted in the histopathology of AA;

1. Acute hair loss
2. Persistent alopecia
3. Partial telogen to anagen conversion
4. Recovery

A peri-bulbar lymphocytic inflammation (“swarm of bees”) with no scarring is characteristic of the diagnosis of all 4 stages of AA. The inflammatory cellular infiltrate is composed chiefly of activated T lymphocytes together with macrophages and Langerhans cells. In acute phase of hair loss, matrix cell and metrical melanocyte failure with formation of dysplastic hair shafts is noted. A decrease in anagen to telogen ratio resulting in marked increase in telogen and catagen hair occurs, which can be observed on horizontal section of scalp biopsy specimens.^[3,20,21]

In patient with long standing history of alopecia, the involved hair follicles arrest in the end stage of telogen phase. In patients with complete recovery, normal hair follicles with little or no peribulbar lymphocytic infiltration and no decrease in hair density are noted.^[15] Eosinophils are also detectable in all stages of AA.

CLINICAL FEATURES;

The onset of AA may be at any age and peaks between the second and fourth decades of life. Both males and females are equally affected. The prognosis for AA is defined by the age at disease onset, duration, nail signs, the extent of hair loss and the presence of atopic dermatitis. The characteristic initial lesion is a circumscribed, totally bald

and smooth patch. The skin within the bald patch appears normal or slightly reddened. During active phases of diseases short easily extractable broken hairs, known as exclamation mark hairs, are often seen at the margins of the bald patches. They are broken off about 4 mm from the scalp, and are narrowed and less pigmented proximally. Patches are most common in the scalp and beard but other areas, especially the eyelashes and eyebrows can also be affected. An uncommon diffuse pattern is also recognized, with exclamation-mark hairs scattered widely over a diffusely thinned scalp.^[22] Many cases have been reported to have the nodules which are often asymptomatic and located mainly on the vertex and the upper part of the occipital area and their number doesn't exceed two. The lesions are like one or two small islands surrounded by calm sea as the surrounding scalp is normal unlike in cellulitis of scalp.^[5,22]



Clinical presentation of AA is subcategorized according to pattern or extent of hair loss.

According to pattern;

1. Patchy AA: round or oval patches of hair loss (most common)
2. Reticular AA: reticulated pattern of patchy hair loss.
3. Ophiasis: band like AA, hair loss in the parieto-temporo-occipital hair margin
4. Ophiasis inversus (saisapho): a rare band like pattern of hair loss on the frontal parieto-temporal scalp (the exact opposite of ophiasis)

5. Diffuse AA: a diffuse decrease in hair density over the entire scalp.

According to extent of involvement;

1. Alopecia areata; partial loss of scalp hair
2. Alopecia totalis; 100% loss of scalp hair
3. Alopecia universalis; 100% loss of hair on the scalp and body.
4. Alopecia areata/Alopecia universalis; AT with variable amounts of body hair loss.

DISCUSSION

White Hair in Alopecia Areata;

The sudden diffuse onset of AA may result in the sudden shedding of pigmented hairs only, leaving behind the white hairs. It has been postulated that hair related pigmented cell may be a target for the immune response in alopecia areata. There is also evidence to suggest that the immune response is directed at cortical keratinocytes, resulting in a defect in melanin transfer.^[18]

Nail changes;

Nail signs are associated with 7-66% patients of AA. The nail plate is pitted and these pits are regularly arranged in horizontal or vertical rows or both (scotch plaid appearance). Pits are larger and less deep than on psoriasis. Onychodystrophy has been reported in number of cases. Changes may be seen in one, multiple, or all of the nails. The dystrophy may precede or follow AA. Pitting with irregular pattern or in organised transverse or longitudinal rows, trachyonychia, Beau's lines, onychorrhexis, thinning or thickening (pseudomycotic), onychomadesis, koilonychia, punctuate or transverse leukonychia and red spotted lunula may be associated with AA.^[18]

Ocular abnormalities;

The association of cataract with AA is a rarity unlike the occurrence of cataracts as a complication of certain dermatological entities or syndromes which are well known. There are many reports of cataract in association with alopecia totalis.^[18]

Diagnosis;

AA is usually diagnosed on the basis of history and physical finding.

Clinical diagnosis;

1. Circumscribed patch of alopecia without scarring.
2. Exclamation mark hair at the periphery.
3. Patches are most common in the scalp and beard but other areas, especially the eyelashes and eyebrows, can also be affected. An uncommon diffuse pattern is recognized, with exclamation mark hairs scattered widely over a diffusely thinned scalp.
4. Up to 50% of patients show fine pitting of the nails.

An association of patchy hair loss with autoimmune disorders, as well as with atopic dermatitis (in 39% of

cases) further points to the correct diagnosis.^[13] If the diagnosis is not clear after a clinical evaluation, as can be the case with the diffuse variant of alopecia areata, skin biopsy is usually diagnostic. In acute alopecia areata, histological examination reveals a characteristic "bee swarm pattern" of dense, perifollicular lymphocytic infiltrates around anagen hair follicles,^[3,20,21] but in patients with chronic disease, this pattern may be absent.^[21]

Course and prognosis;

The course of AA is difficult to predict. Patients usually present with several episodes of hair loss and hair regrowth during their life time. The recovery from the hair loss may be complete, partial or none. In 50% of patients, hair will re-grow entirely within one year without treatment. However, 7% to 10% can eventually develop the severe chronic form of the AA. Recurrence is a rule. The prognosis is good if hair loss confined to one region of scalp.^[18]

The prognosis is poor if

1. Rapid progression
2. Nail changes
3. extensive hair loss
4. Atopy
5. The presence of other immune diseases.
6. Family history of AA.
7. Patients with ophiasis
8. Early onset (onset under age of 10 years)

Treatment of alopecia areata;

Topical treatment

- Topical corticosteroids
- Intralesional corticosteroids^[7]
- Anthralin
- Contact allergens.^[18,21]
- Dinitrochlorobenzene
- Diphenecyprone
- Photo chemotherapy (PUVA)^[7]
- Topical Minoxidil
- Inosiplex
- Cyclosporine
- Tacrolimus

Systemic treatment;

Oral mini pulse (OMP) therapy with betamethasone is used in the treatment of AA especially recurrent cases.

An oral monthly pulse of prednisolone 300mg is effective and safe in the management of AA.

Protocol for the Treatment of Adult Alopecia Areata.^[23]

Patients over 10 years of age with less than 50% hair loss

1. Do nothing.
2. Intralesional triamcinolone acetonide injections.
3. Minoxidil 5% solution.
4. Combination of Minoxidil 5% solution and super potent topical corticosteroid.
5. Combination of Minoxidil 5% solution and Anthralin.
6. Topical immunotherapy if the above do not work.

Patients with more than 50% hair loss

1. Topical immunotherapy with Diphencyprone.
2. Minoxidil 5% solution and super-potent topical corticosteroids
3. Combination of Minoxidil 5% solution and Anthralin.
4. PUVA
5. Systemic corticosteroid therapy (rarely).

For children, under the age of 10 years, treatment options include Minoxidil 5% solution with or without topical mid-potent corticosteroids or short contact Anthralin therapy.^[21]

CONCLUSION

From the above results, the authors concluded that hepatic and renal involvement occurs in patients suffering from dengue fever. Hence, multidisciplinary approach should be carried out while treating such patients.

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