Alopecia Areata (AA, Patchy Hair Loss) — Causes and Treatment

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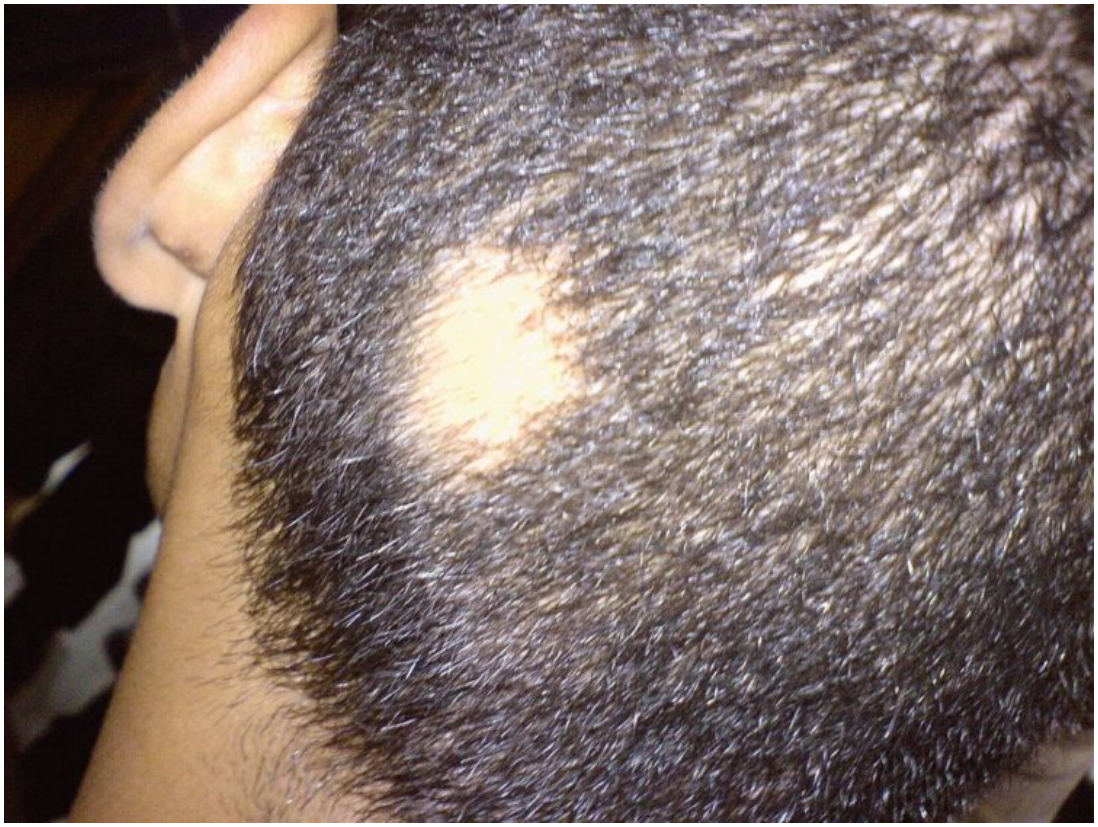
Alopecia areata is an autoimmune disease of unknown etiology characterized by recurrent non-scarring hair loss in hair-bearing areas, ranging from a small patch to complete loss of hair. It is a benign condition that usually occurs without symptoms. Burning sensation and pruritus in the affected area can be present in rare cases. Other autoimmune diseases may be associated with this condition, such as thyroid diseases and vitiligo. A scalp biopsy may be required for diagnosis in some cases. Treatment, which is based on the type of alopecia, includes topical minoxidil, corticosteroids (topical, intralesional, or oral), or anti-androgens.



Epidemiology of Alopecia Areata

Alopecia areata affects 1% of the population (although this figure varies from 0.1% to as much as 2%). There is no sex predisposition, but men seem to be more frequently affected by more severe forms of the disease than women. It starts more commonly in children and young adults and has a **sudden onset**. Interethnic variation is not significant.

Etiology of Alopecia Areata

The etiology of alopecia areata remains unclear, but it may be related to genetic, immunologic, and environmental factors.

Genetic factors

About 20% of patients with alopecia areata have at least one parent or sibling who is affected. The concordance among **monozygotic twins** is **55%**. Some **HLA class II genes** are shown to be associated with **alopecia areata**: DQ3, DQ7, DR11, DR4, DR5, and DPW4, with the former three predisposing to more severe forms. One study also reported that eight genomic loci are associated with this disease.

Immunological factors

Many patients with alopecia areata have other **autoimmune disorders,** such as **lupus erythematosus**, **scleroderma**, **thyroiditis**, **celiac disease**, etc. Also, the fact that steroids and **immunomodulators** are effective against this disease further suggests an autoimmune pathology

Environmental factors

Stress (physical or emotional), **infections** (e.g., cytomegalovirus), and **hormones** (e.g., corticotropin-releasing hormone) predispose individuals to autoimmunity.

Pathology and Pathophysiology of Alopecia Areata

Alopecia areata is essentially an **autoimmune disease** resulting from a genetic predisposition and/or environmental factors. The loss of immune privilege by the hair follicle (perhaps due to attack by **natural killer cells**) and the subsequent change in the **cytokine/chemokine profile** leads to the **infiltration of T lymphocytes** around the bulb.

In alopecia areata, due to various inflammatory and autoimmune reactions, hairs do not progress beyond the **anagen phase**. The hair enters the anagen phase, does not complete this phase properly, and then quickly enters the **telogen phase** (dying phase). This cycle continues in alopecia areata.

Important: One of the two hallmarks of alopecia areata is **peribulbar infiltration** of **CD4+** and **CD8+ cells** around anagen follicles. The other hallmark is the presence of **exclamation mark hair.**

Symptoms of Alopecia Areata

There is a **sudden onset** of **non-scarring** patch or patches of hair loss on **any hair-bearing area**, most commonly the scalp and also the beard area. Rarely, the eyebrows can also be affected.

Most patients have only a single patch, but two or more than two patches are not rare. The multiple patches may coalesce into a large area of alopecia. The patches themselves are **asymptomatic**; however, some patients may experience itching or burning.

Alopecia, although normally unifocal or multifocal, can come in various forms:

- **Alopecia totalis**: Complete loss of hair on the scalp
- **Alopecia universalis**: Complete loss of hair on the entire epidermis
- **Ophiasis**: A band of hair loss on the occipital scalp
- **Sisaipho** (almost the reverse spelling of ophiasis): The opposite of ophiasis, i.e., the band of hair loss is more frontal.
- **Diffuse alopecia areata** (also known as **alopecia areata incognita**): A very rare form characterized by the absence of typical patches of hair loss, but rapid progression to **alopecia totalis** or **universalis**; predominantly in young women.

The severity of the disease is classified as follows:

- **Mild**: Three or fewer patches or < 3 cm
- **Moderate**: More than 3 patches or at least one patch > 3 cm
- **Severe**: Alopecia totalis or universalis

Diagnosis of Alopecia Areata

The diagnosis of alopecia areata is mainly **clinical**; an **excisional biopsy** is rarely needed. Some of the clinical tests that can be performed are:

- **Pull test**: Considered positive if > 10 hairs are pulled out
- **Pluck test**: Examination of the root of the plucked-out hair
- **Trichoscopy**: Examination using a **dermoscope**. Findings include hyperkeratotic plugs, **exclamation mark hair** (considered to be pathognomonic of alopecia areata), and destroyed hairs.

Differential diagnoses

Other skin diseases can also cause patchy hair loss:

- **Tinea capitis:** Inflammation is present and **Wood light and KOH examination** can reveal the presence of the fungus.
- **Trichotillomania:** Incomplete hair loss with negative pull test (**epilation test**).
- **Cicatricial alopecia** (e.g., **lichen planopilaris**, **chronic discoid lupus erythematosus**): Patchy erythema and hyperkeratosis are present; excisional biopsy can help differentiate.
- **Androgenic alopecia:** Genetic predisposition and family history.

Diffuse alopecia areata must be differentiated from the following conditions:

- **Syphilis** – serological testing; other clinical signs and symptoms
- **Telogen and anagen effluvium** – lack of exclamation mark hairs; a history of intake of a causative drug
- **Loose anagen hair syndrome** – anagen hairs on **trichogram**; mainly young children with blond hair; autosomal dominant inheritance

Management of Alopecia Areata

At present, there is no cure for alopecia areata, nor is there any effective agent that universally induces remission. The treatment includes **topical**, **intralesional**, and/or **systemic agents**, and varies with patient age and disease severity.

Important: The first-line therapy usually is **high-potency topical corticosteroids** (e.g., betamethasone lotion or clobetasol gel) or **intralesional steroids** (e.g., multiple 1-mL injections of 2.5–10 mg/mL triamcinolone acetate) because of their anti-inflammatory effects.

Some prefer intralesional steroids over topical steroids, especially for small patches of alopecia. Redness and spots are possible at the site of injection. Systemic corticosteroids are not preferred because of high rates of side effects and high relapse rates; however, short-term therapy, such as **pulse methylprednisolone** or **oral mini-pulse therapy**, is reported to be effective.

Minoxidil (usually as 5% lotion for men and 2% lotion for women; twice daily, not more than 25 drops twice a day) is another agent that is used in the treatment of alopecia areata. It works by stimulating hair growth, especially follicular proliferation. It may be given alone or combined with **anthralin**. Skin irritation may occur, manifesting as itching, rash, spots, etc., at the site of application.

Short-contact or overnight treatment with **anthralin** (usually as 0.5% or 1% cream), an agent that has irritant properties as well as immunosuppressive and anti-inflammatory properties, has been reported to be an effective treatment. This type of treatment is recommended for use in children. The adverse effects of anthralin are **scaling** of the skin, **folliculitis**, and **lymphadenopathy**. Anthralin preparations can stain the clothes.

Topical immunomodulators (e.g., dinitrochlorobenzene, diphenylcyclopropenone, or squaric acid dibutyl ester) work by inducing **allergic contact dermatitis**. Another therapy with reported efficacy is **psoralen with ultraviolet A (PUVA)**; however, it has a high relapse rate. Other drugs include topical **tacrolimus**, topical **cyclosporine**, and **methotrexate**.

Important: Young patients (age < 10 years) are given topical corticosteroids or minoxidil or anthralin. Patients are older than 10 years of age with < 50% of scalp involvement are given topical or intralesional corticosteroids or minoxidil or anthralin. Patients in whom > 50% of the scalp is affected should be given **topical immunomodulators** as the first-line therapy; if the response is poor, however, topical corticosteroids or minoxidil should be tried.

Therapies of uncertain efficacy in the treatment of alopecia areata include **aromatherapy** (massage of certain essential oils every day for 2 minutes on the patches).

There is a lot of psychological distress among these patients. Counseling and psychotherapy may be advised, and wigs may be prescribed to such patients.

Progression and Prognosis of Alopecia Areata

Data regarding progression are limited. There is a high **spontaneous remission rate** for mild cases. Patients with moderate disease have lower rates of spontaneous remission, and those with severe disease can have a high relapse rate. The prognosis is especially poor in those with **alopecia totalis** or **universalis**.

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