Beyond Bald Patches: Exploring The Pathophysiology and Management of Alopecia Areata

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Abstract—**Alopecia Areata (AA) is a chronic, organ-specific autoimmune disorder characterized by non-scarring hair loss on the scalp and other body sites. It manifests as sudden, well-circumscribed patches of hair loss with an unpredictable course, often leading to significant psychological distress. The pathogenesis involves peribulbar lymphocytic infiltration, primarily CD8⁺ T cells, and the collapse of hair follicle immune privilege, influenced by genetic predisposition, environmental triggers, hormonal imbalances, and associations with other autoimmune disorders. Conventional treatments, including corticosteroids, minoxidil, immunotherapy, and JAK inhibitors, can be effective but are often limited by adverse effects, incomplete regrowth, and relapses.**

Growing interest in herbal and natural therapies has emerged as a safer, sustainable alternative. Medicinal plants such as Aloe vera, Amla, Onion, Garlic, Bhringraj, Tea polyphenols, Fenugreek, Coconut oil, Almond oil, and Tulsi have demonstrated potent anti-alopecia activity through mechanisms including DHT inhibition, antioxidant and anti-inflammatory effects, enhanced scalp circulation, follicular repair, and promotion of the anagen phase. Integrating herbal medications alongside conventional therapy offers promising outcomes, with minimal side effects, improved hair regrowth, and enhanced scalp health.

This review compiles current knowledge on the etiology , pathophysiology, clinical features, and treatment options for AA, highlighting the role of herbal interventions as effective adjuvants in the holistic management of this complex autoimmune disorder.

Index Terms—**Alopecia Areata, Autoimmune Hair Loss, CD8⁺ T cells, Minoxidil, JAK inhibitors, Herbal Therapy, DHT Inhibition, Scalp Health.**

I. INTRODUCTION

Alopecia Areata is chronic, organ-specific autoimmune disease that affects the hair follicles, leading to non-scarring hair loss on the scalp and other body sites. It is characterized by sudden onset of well-circumscribed, round, or oval patches of hair loss. The disease has an unpredictable course, with 80% of patients showing spontaneous regrowth early in the disease, though relapses are common.[1]

The global incidence ranges from 0.57%–3.8%, with a lifetime risk of about 2%. It affects both genders, slightly more in females, possibly due to greater concern for hair loss. The average age of diagnosis is around 33 years, and many females present during adolescence, sometimes with associated autoimmune or nail disorders.

AA negatively impacts quality of life, especially among females aged 20–50 years, those under stress, or with more than 25% hair loss. Several comorbidities are linked, including atopy (11–38%), thyroid disorders (17.7%), diabetes mellitus (11.1%), vitiligo, SLE, rheumatoid arthritis, and psoriasis. Genetic predisposition involves immune-regulating and MHC genes, while environmental factors such as stress, infection, and trauma also contribute. [2-13]

The hallmark feature of active AA is a peribulbar lymphocytic infiltration—often described as a "swarm of bees" appearance—composed mainly of CD8⁺ cytotoxic T cells around the hair bulb. These immune cells are attracted to the follicle through the expression of NKG2D ligands, such as cytomegalovirus UL16-binding protein, which triggers immune activation.
A major pathogenic mechanism is the loss of immune privilege in the hair follicle. Normally, the follicle is protected from immune attack, but in AA, this privilege collapses, exposing follicular antigens to the immune system.

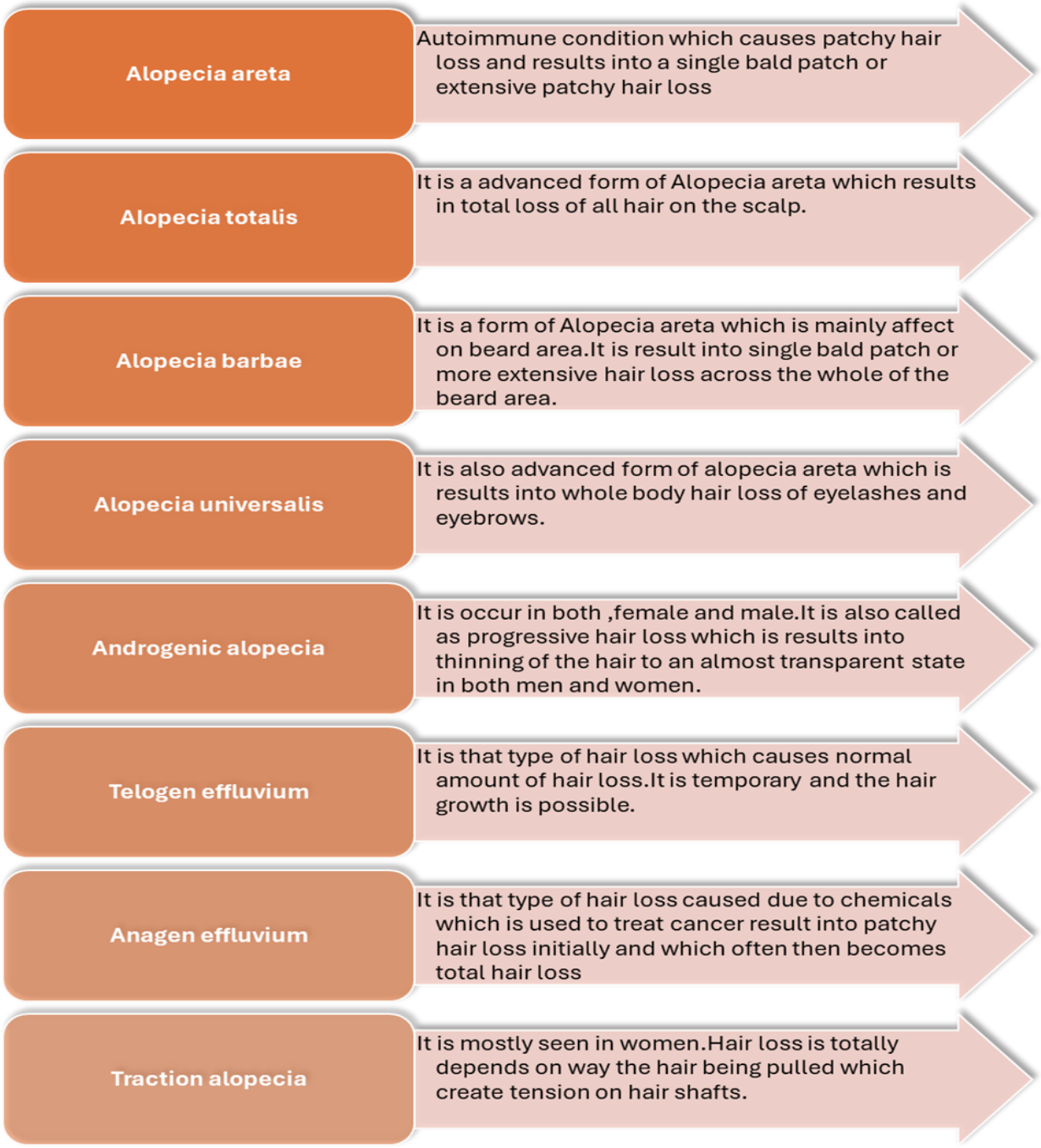
Experimental studies have provided direct evidence of T-cell–mediated involvement. In particular, transplantation experiments in severe combined immunodeficient (SCID) mice demonstrated that injecting NKG2D$^+$ activated T cells from healthy individuals could induce a hair loss pattern identical to human AA, confirming the central role of CD8$^+$ T cells and NKG2D ligand interactions in disease pathogenesis.[14-16]

Its prevalence in the general population is about 0.1–0.2%, with a lifetime risk of 1.7%. Although males are reported to be more commonly affected, the disease leads to greater emotional distress in women and children due to cosmetic concerns.

In modern medicine, corticosteroids are the main treatment option, but their long-term use is limited by potential harmful side effects. Therefore, there is growing global interest in alternative medical systems. Among synthetic treatments, minoxidil, a potent vasodilator, is scientifically proven to promote hair growth and treat alopecia. However, the long-term use of synthetic drugs like minoxidil can lead to adverse side effects, making them less ideal for safe, sustainable therapy.

Therefore, there is a growing interest in natural and herbal alternatives that can effectively manage alopecia with fewer side effects. Many medicinal plants have demonstrated potent anti-alopecia activity through mechanisms such as DHT inhibition, improved scalp circulation, antioxidant effects, and nutritional support.[17-18]

Classification of Alopecia Areata:



Figure_no -1 Classification of Alopecia [19]

Pathophysiology of Alopecia Areata:

Alopecia areata is a chronic autoimmune disorder where the body's immune system mistakenly attacks its own hair follicles, leading to hair loss. The exact reason is (more than one cause).

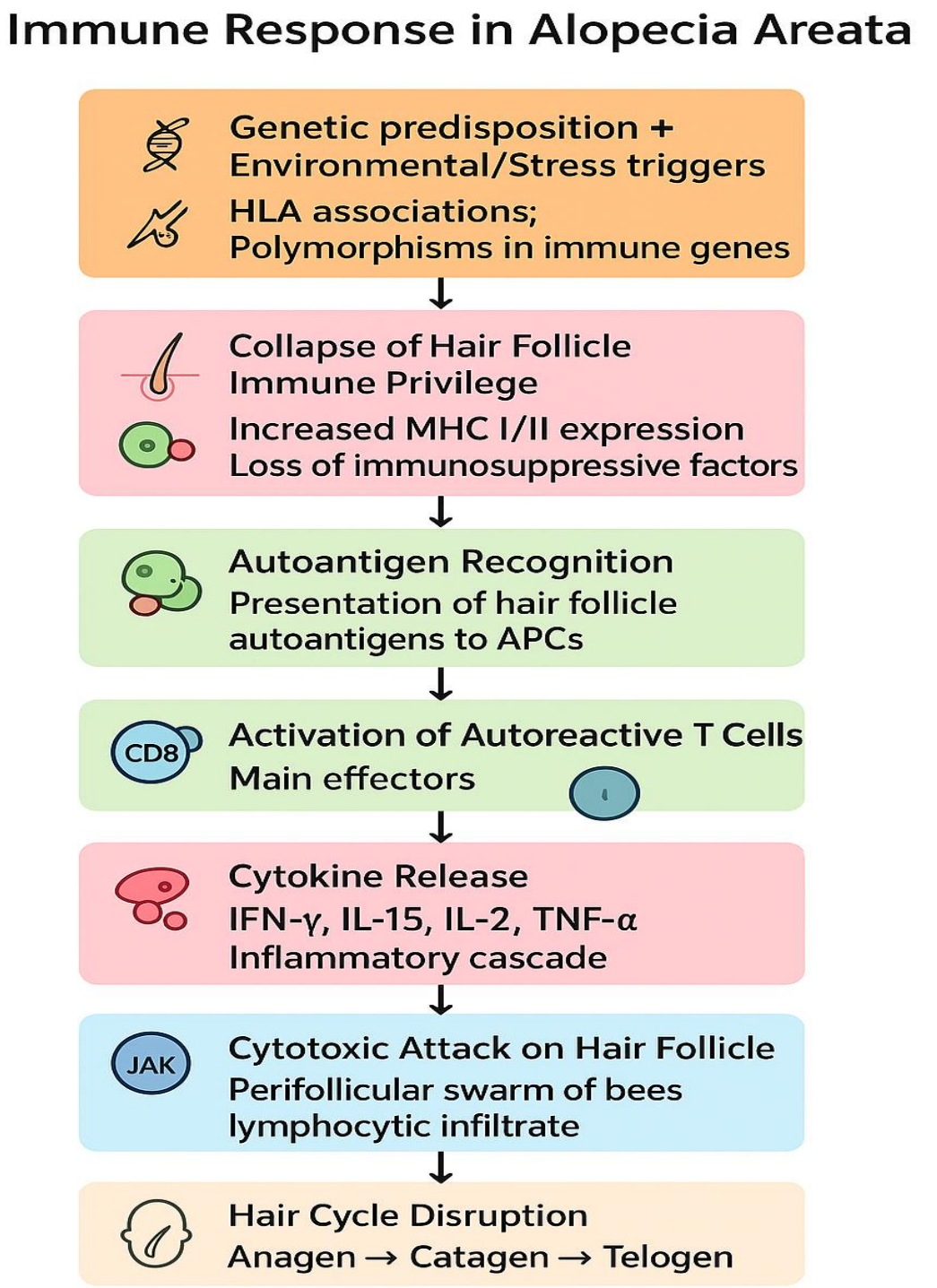
a. Autoimmune Reaction (Main Cause)

The immune system mistakenly identifies hair follicle cells as foreign.

T-lymphocytes (immune cells) surround and attack the follicle bulb (where hair growth begins).

This causes inflammation and disrupts the hair growth cycle, especially the anagen phase (growth phase).

The follicle becomes "inactive," leading to sudden hair shedding. [19-20]



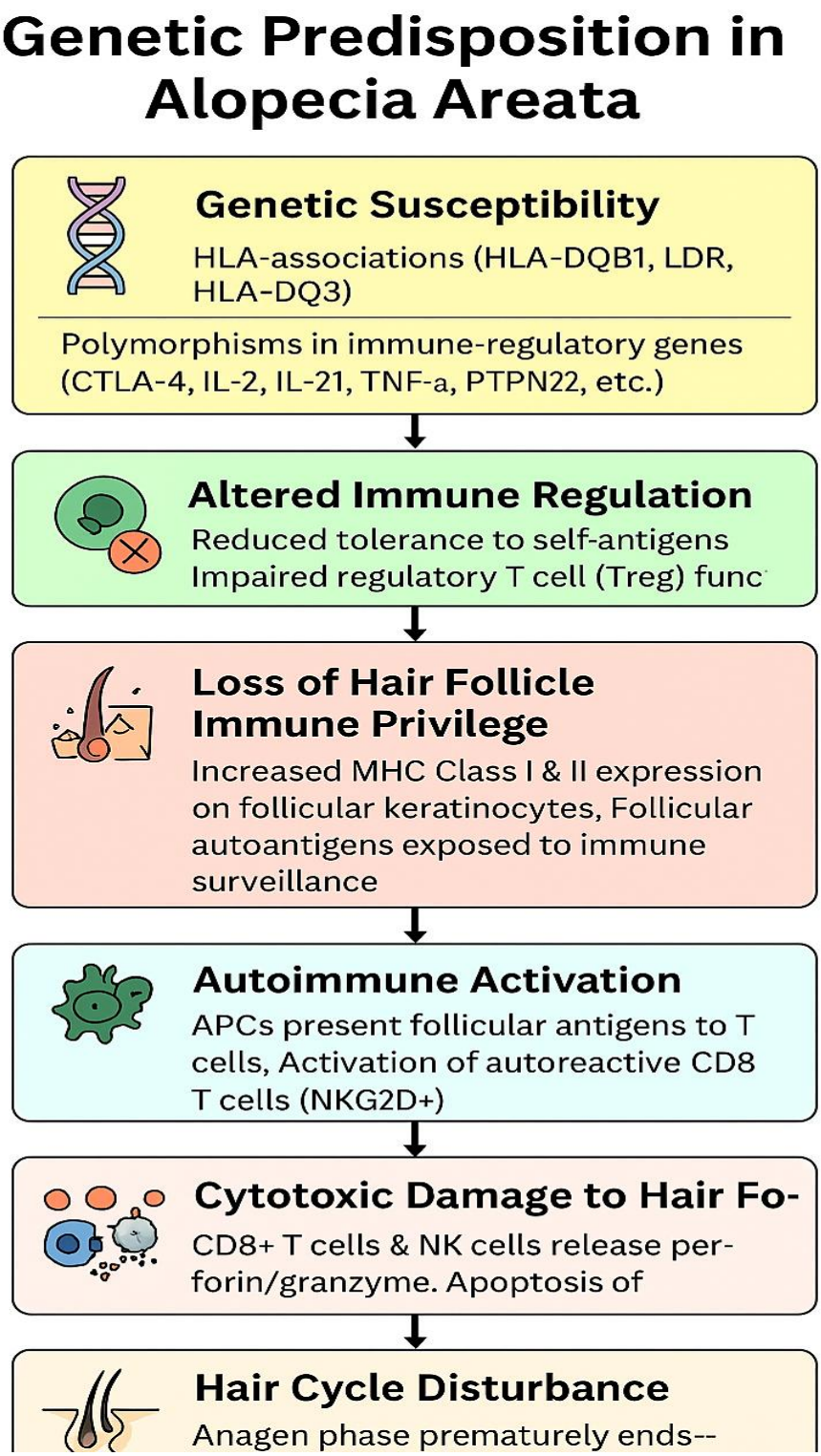
b. Genetic Predisposition

Alopecia areata often runs in families.

10–25% of patients report a family history.

Genes linked to immune regulation, especially HLA (Human Leukocyte Antigen) genes, play a role.

Twin studies show higher chances in identical twins than in non-identical twins → strong genetic link.[21-28]



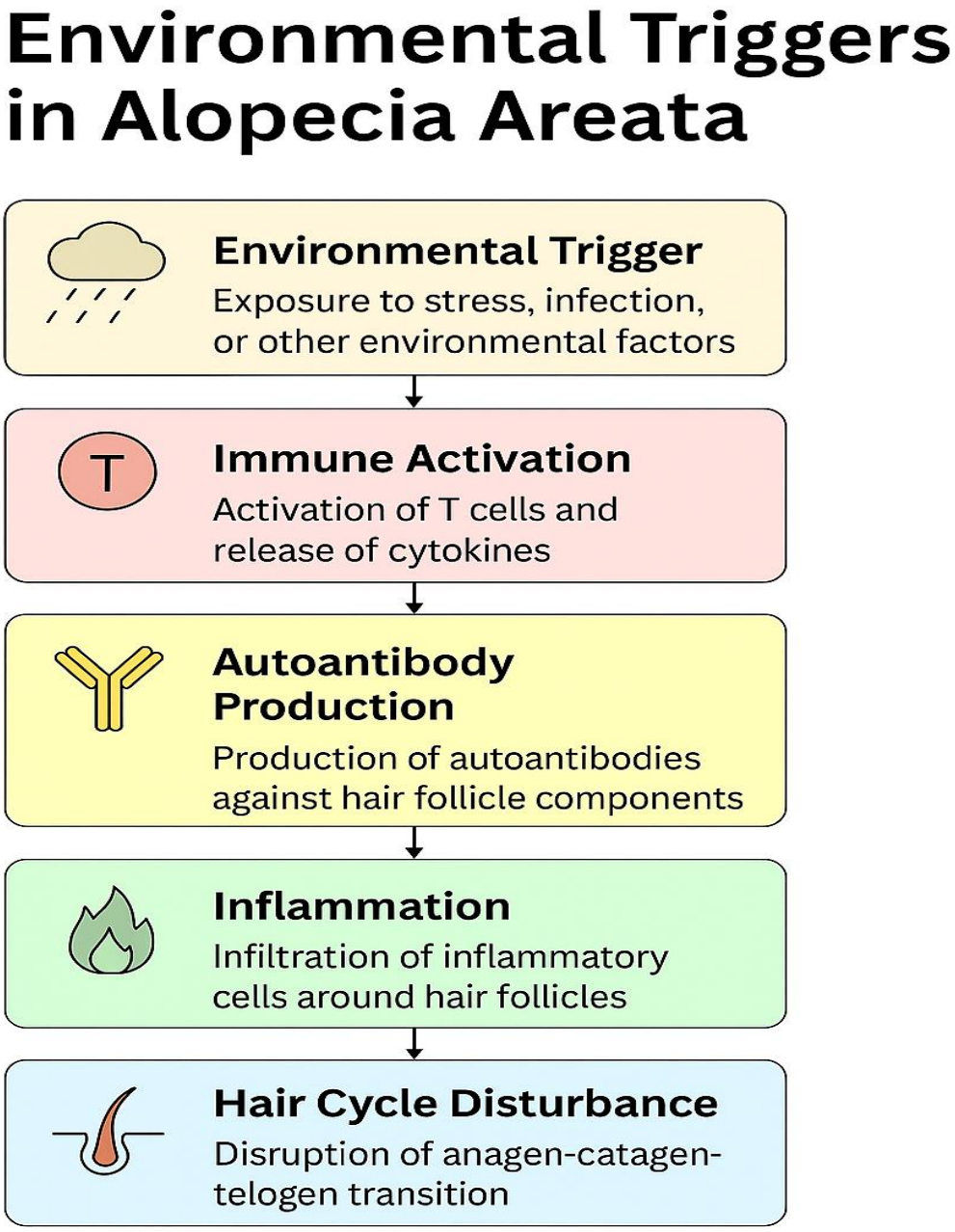
c. Environmental Trigger[29]

Certain outside factors can trigger or worsen the condition in genetically people:

Emotional or physical stress (exams, trauma, surgery).

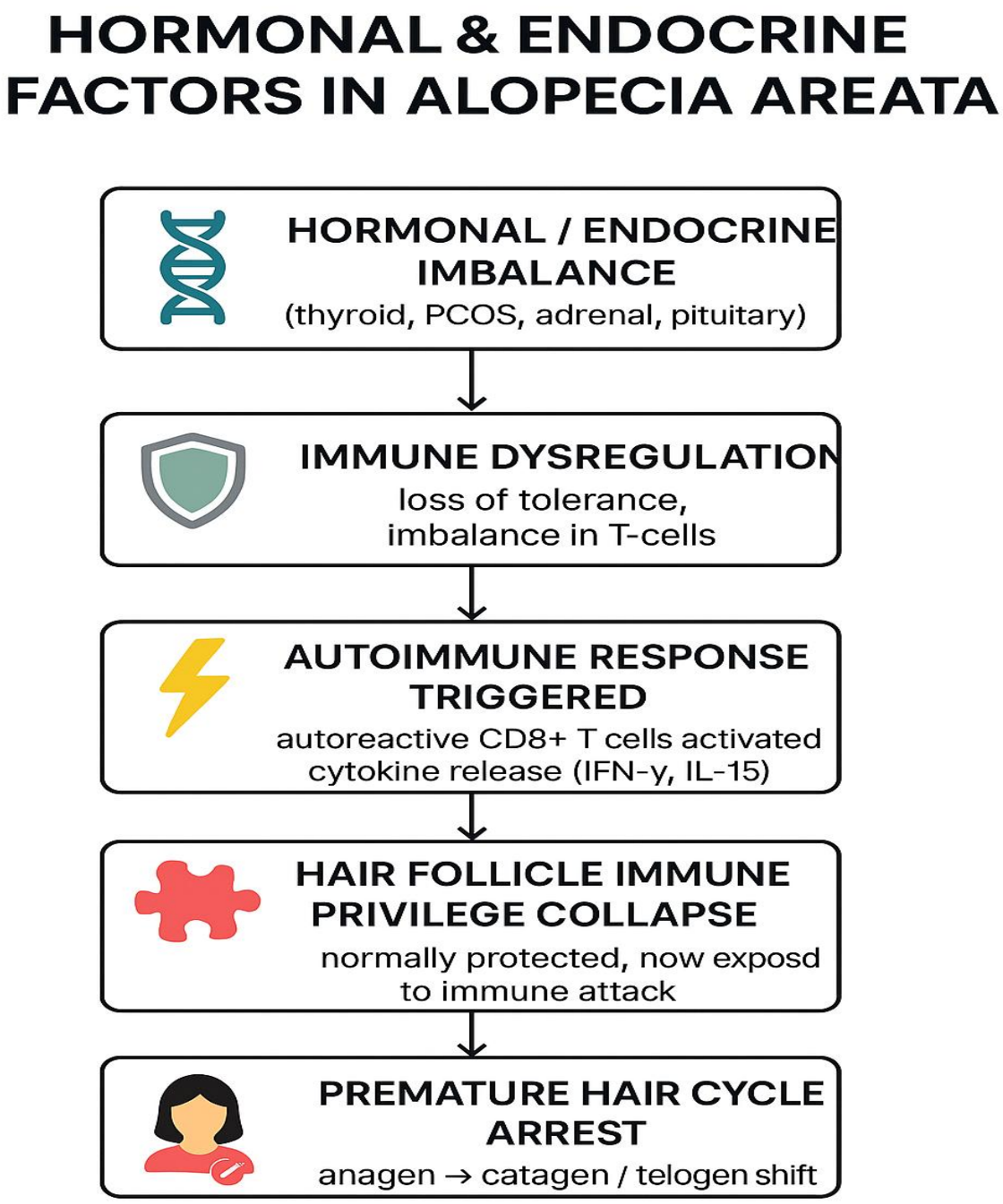
Infections (viral or bacterial), sometimes function as a trigger.

Seasonal changes or toxins may also stimulate abnormal immune activity.

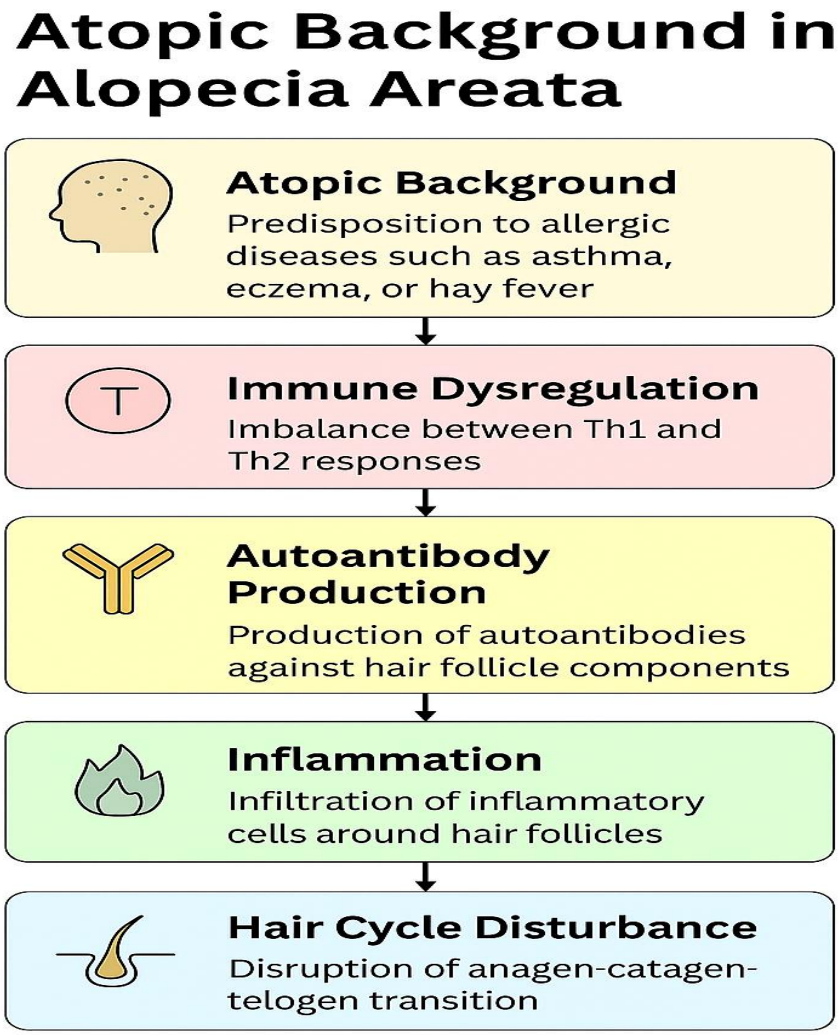


d. Hormonal & Endocrine Factors[30-32]
Hormones influence the immune system and hair growth.

Disorders like thyroid disease (Hashimoto's thyroiditis, Graves' disease) are strongly associated. Women with hormonal imbalance (e.g., PCOS)



e. Association with Other Autoimmune Disorders[33]
Alopecia areata is commonly seen alongside other autoimmune diseases:
Vitiligo (patchy skin depigmentation)
Type 1 diabetes mellitus
Rheumatoid arthritis
Systemic lupus erythematosus (SLE)
Psoriasis
This shows a shared immune dysfunction.
f. Atopic Background (Allergic Tendency)
People with eczema, asthma, or allergic rhinitis have increased risk.



g. Other Possible Factors (Still Under Study)[33]
Oxidative stress – imbalance of free radicals damaging follicles.
Vitamin D deficiency – affects immune regulation.
Gut microbiome imbalance – possible immune link.

Causes of Alopecia Areata**:**[34]
Hair loss is typically related to one or more of the following factors

1. Family History (Heredity): A hereditary disease that develops with ageing is the most frequent cause of hair loss. Male hormonal alopecia, male pattern baldness, and female pattern baldness are all terms used to describe this illness. It often occurs gradually and in predictable patterns, with women experiencing thinning hair above the head and males experiencing receding hairlines and bald patches.
2. Hormonal Changes and Medical Conditions: Permanent or temporary hair loss can result from a number of circumstances, including hormonal changes brought on by pregnancy, childbirth, menopause, and thyroid issues. Alopecia areata, which affects the immune system and results in patchy hair loss, ringworm infections of the scalp, and trichotillomania, a disorder characterised by compulsive hair pulling, are examples of medical illnesses
3. Medications and Supplements: Some drugs, including those for cancer, arthritis, depression,

heart issues, gout, and high blood pressure, can cause hair loss as adverse effects.

4. Radiation Therapy to the Head Hair may not regrow as quickly as it formerly did. Many people experience hair thinning months after a physical or mental shock, which is a highly upsetting occurrence. Temporary hair loss results from this kind.
5. Hairstyles and treatments: Traction alopecia is a kind of hair loss that is brought on by over-styling and pulling hairstyles like braids. Hair loss can also be brought on by curling and hot oil treatments. If scarring develops hair loss could be irreversible

Symptoms: [35-36]

Category	Symptom	Description / Notes	Frequency / Likelihood	Early/Late Sign	Visual Cue / Notes
Hair Loss Pattern	Patchy Hair Loss	Sudden round or oval bald patches; scalp appears smooth with no inflammation	Very common (~70-80%)	Early	Typically 1–3 cm, may coalesce into larger patches
	Diffuse Hair Thinning	Generalized thinning across scalp without defined patches	Less common (~10%)	Early	Often mistaken for stress-related hair shedding
	Complete Scalp Hair Loss (Alopecia Totalis)	Entire scalp affected; complete hair loss	Rare (~5-10%)	Late	Can develop over weeks to months; no scarring
	Complete Body Hair Loss (Alopecia Universalis)	Loss of all body hair including eyebrows, eyelashes, axilla, and pubic hair	Very rare (~1%)	Late	May cause eye irritation, sunburn on scalp/skin
Hair Changes	Exclamation Mark Hairs	Hairs taper near the scalp like “!”; fragile and break easily	Common	Early	Key diagnostic clue at patch edges
	Broken / Fragile Hairs	Hair shafts brittle and break with minimal traction	Moderate	Early	Short stubs visible under magnification
	Regrowing White / Gray Hairs	New hairs initially depigmented; may later regain color	Common	Late	Seen during remission/regrowth phase
	Altered Hair Texture	Regrown hair may be thinner, softer, or curly initially	Occasional	Late	Temporary; normal hair texture may return
Skin & Scalp Signs	Smooth, Bald Patches	Skin looks normal, no redness, scaling, or scarring	Almost always present	Early	Helps differentiate from fungal or scarring alopecias

	Mild Itching / Tingling	Pruritus or tingling sensation may precede hair loss	Occasional	Early	Often subtle; patients may not report
	Nail Changes	Nail pitting, ridging, Beau's lines, brittleness	10–20%	Early/Late	Common in chronic or severe cases
Progression Patterns	Rapid Onset	Hair loss can occur over days to weeks	Common	Early	Can be alarming to patients and caregivers
	Recurrent Episodes	Hair may regrow and fall out repeatedly; unpredictable	Common	Ongoing	Can affect scalp, eyebrows, eyelashes
	Symmetric / Asymmetric Involvement	Patches may appear on one or both sides	Variable	Early/Late	Sometimes bilateral but uneven distribution
Associated Symptoms	Psychological Distress	Anxiety, depression, low self-esteem due to visible hair loss	Very common	Early/Late	May require counseling or support groups
	Eye / Eyelash Loss	Partial or complete loss of eyelashes/eyebrows	Moderate (~50%)	Late	Can lead to eye irritation and cosmetic concerns
	Scalp Sensitivity	Tenderness, discomfort, or burning sensation in bald areas	Occasional	Early	Usually mild; resolves when hair regrows
	Trichodynia	Pain, itching, or tingling at hair roots	Occasional	Early	Often seen in active inflammation phase
Rare / Severe Features	Extensive Alopecia	Large confluent bald areas over scalp or body	Rare	Late	May indicate aggressive autoimmune involvement
	Nail Fragility	Severe nail brittleness leading to splitting	Rare	Late	Often accompanies widespread disease
	Eyebrow / Eyelash Sparsity	Partial loss of brows/eyelashes leading to cosmetic changes	Moderate	Late	Can affect facial expression and aesthetics

II. TREATMENT OF ALOPECIA AREATA

1. Camouflage: -

There are many different types of camouflage that can be offered and used by patients with AA. These include hair pieces (wigs, demiwigs, wiglets, toupees, cascades), hair thickening fibers, concealing powders. Semipermanent options include scalp micropigmentation which is a tattoo applied in a stippling pattern to mimic hair follicles. Realistic non-

traumatic sticker tattoos can be useful for eyebrow regions and are a painless option in those who do not want a permanent option [37-40]

2. Micronutrient Supplementation: -

Many patients and providers advocate for supplementation of micronutrients in the treatment of AA. Although data sup ports an association between lower levels of serum vitamin D, zinc, and folate levels in patients with AA as compared to controls, there is a lack of data evaluating the benefit of sup plementation of these. [41]

3. Corticosteroids: -

A large range of different topical corticosteroids are used in practice in var ideregimens, and often in combination with other topical or systemic treatments.The most commonly used steroid for this purpose is triam cinolone acetonide

- TOPICAL CORTICOSTEROIDS: - Corticosteroids are absorbed just in a minimum dose on a normal scalp. The occlusion by means of plastic bandage is an effective method to help increment up to 10 times the absorption level. In the skin absorption process, a topical steroid is metabolised in the form of inactive compounds before entering the circulatory system, thus making systemic side effects rare, but possible. Recent experiences with high potency topical steroids in occlusion in severe AA forms have shown regrowth in a considerable proportion of patients. 104 Topical steroid side effects are usually reversible and include: folliculitis, atrophy, telangiectasia, hypertrichosis.
- SYSTEMIC CORTICOSTEROIDS: -The use of systemic cortisone to treat AA was introduced in 1952. Steroids usually induce regrowth of AA, but relapses are common after suspension.It is also sometimes possible to saw “rebound" effect, with the reoccurrence of the disease in a more severe form and resistant to therapy.Chronic administration is therefore often needed to maintain hair regrowth, and it is always associated with severe long term side effects, such as osteoporosis, weight gain, diabetes, adrenal insufficiency and some irreversible effects such as cataract, glaucoma, aseptic necrosis of the femoral head.[42]

4. Antiallergic drugs:-

The use of antihistamines in patients affected by pollinosis in order to avoid relapses triggered by al-allergic crisis has already been preconized. There are some favourable indications with the use of fexofenadine 106 and of m Ebastine for at least 4 months, These antihistamines have immunomodulatory properties such as the inhibition of T lymphocytes activation, the inhibition of several molecules induced by interferon gamma, and the inhibition of the production of substance P. Ebastine proved its superiority compared to an anxiolytic, with a therapeutic effectiveness in half of the patients, independently from the presence of allergies, and in a more evident way in subjects in which Alopecia was triggered by stress. Fexofenadine seems to ameliotate clinical response and tolerability to topical immunotherapy at least in the atopic patients. It is important to remember that hystamine could inhibit Treg lymphocytes through H1 receptors [43-44]

5. Antibiotics: -

The hypothesis that AA could be caused by infections has led in the past to the use and abuse of antibiotics. Recently, there were reports of possible therapeutic benefit also in the concomitant AA of the antibiotics used in patients with helicobacter pylori gastritis (HP). 115 On the other hand epidemiological studies have shown that a relation between HP and AA is not supported due essentially to the high prevalence of HP in the control populations. [45-46]

6. Anthralin: -

Both short-contact and overnight treatments have been used. Anthralin concentrations varied from 0.2-1%. Minoxidil [47]

7. Minoxidil: -

Appears to be effective in the treatment of extensive disease (50-99% hair loss) but is of little benefit in alopecia totalis or alopecia universalis. The 5% solution appears to be more effective. No more than 25 drops are applied twice per day regardless of the extent of the affected area. Initial regrowth can be seen within 12 weeks, but continued application is needed to achieve cosmetically acceptable regrowth.[48]

8. Hydroxychloroquine: -
Hydroxychloroquine is a systemic antimalarial known to affect the immune system via interference with Toll-like receptors and prevents activation of interferon- 1. Adverse effects included abdominal pain and headache.[49]

9. Apremilast: -
Apremilast inhibits phosphodiesterase 4, resulting in a decrease in the production of multiple pro-inflammatory cytokines. Its lack of immunosuppressive effects but favorable side effect profile when compared to traditional immunosuppressants.[50]

10. HMG-coa Reductase Inhibitors: -
HMG- coa Reductase Inhibitors (statins) have been demon started to decrease proinflammatory cytokines, including IFN- g, TNF- a, IL- 1b, and IL- 6, which contribute to AA disease.134 A combination of simvastatin and ezetimibe in a series of patients with AA affecting 40 to 70% of the scalp reported regrowth in 14 of 19 patients who completed 24 weeks of treatment.[51-53]

11. Psoralen plus UV: -
Both systemic and topical PUVA therapies have been used 20-40 treatments usually are sufficient in most cases Most patients relapse within a few months (mean, 4-8 months) after treatment is stopped.

12. Immunotherapy and JAK Inhibitors [54-55]
Patients with extensive disease, often characterized by more than 50% hair loss on the scalp, may explore treatment alternatives such as topical immunotherapy or oral JAK inhibitors to reduce the necessity of numerous injections associated with intralesional corticosteroids.
Moreover, a retrospective study revealed the superior efficacy of topical immunotherapy compared to intralesional corticosteroids in patients with hair loss patches exceeding 50 cm^2.
The choice between topical immunotherapy and JAK inhibitors depends on patient preference and availability. JAK inhibitors are easier to apply but carry the risk of systemic adverse effects. On the other hand, topical immunotherapy requires careful application and may induce discomfort and cutaneous adverse effects.
A potent contact allergen, such as diphenylcyclopropenone (DPCP) or squaric acid dibutyl ester (SADBE), can be applied weekly to the scalp to stimulate hair regrowth. Clinicians obtain both these allergens from compounding pharmacies.
Treatment begins by applying a small patch at full concentration to sensitize the patient. After 1 to 2 weeks, complete treatment begins with diluted solution concentrations, gradually increasing the concentration weekly. The dose that causes mild dermatitis is the dose used for future doses. Patients may titrate up to a maximum dose of 2%.
Following treatment initiation, clinicians typically assess hair growth approximately 3 months later. A recent meta-analysis examining clinical outcomes of contact immunotherapy for alopecia areata indicates that 74.6% of patients with patchy alopecia experience hair regrowth, compared to 54.4% of patients with alopecia totalis and alopecia universalis
Among patients who receive maintenance treatment, the recurrence rate is 38.2%, contrasting with 49% among those who do not receive maintenance treatment.
Oral baricitinib, a selective and reversible JAK1 and JAK2 inhibitor, treats alopecia areata by suppressing the activation of T lymphocytes. Patients typically require ongoing therapy to sustain the benefits achieved. The United States Food and Drug Administration (FDA) has issued a boxed warning for JAK inhibitors, cautioning about the risk of severe infections, mortality, malignancy, major adverse cardiovascular events, and thrombosis. Initial dosing is 2 mg/d with an increase to 4 mg/d if the response is inadequate after 3 months.
Patients with severe symptoms may start at the 4 mg/d dose. Upon achieving an adequate response, clinicians reduce the dose to 2 mg. Therapy is discontinued if no improvement is observed after 6 months.
Oral ritlecitinib, an inhibitor of JAK3 and the tyrosine kinase expressed in the hepatocellular carcinoma kinase family, is approved by the FDA for patients aged 12 and older with alopecia areata. Similar to baricitinib, continued use is likely necessary to maintain the effects.
The initial dose is 50 mg/d. Potential adverse effects are increased risk for severe infection, death, malignancy, lymphoproliferative disorders, major adverse cardiovascular events, thromboembolic

events, and hematologic, hepatic, and creatine phosphokinase laboratory abnormalities

III. HERBAL MEDICATIONS IN THE MANAGEMENT OF ALOPECIA AREATA: [55-60]

Alopecia, a multifactorial autoimmune disorder characterized by progressive hair loss, has shown increasing prevalence in urban populations due to environmental stressors, lifestyle modifications, and exposure to synthetic drugs. Conventional allopathic treatments, such as those involving chemotherapeutic or hormonal agents, often induce adverse effects and limited efficacy, prompting the exploration of herbal alternatives for safer and sustained management of alopecia.

Several herbal agents possess pharmacologically active constituents that promote hair growth through distinct mechanisms of action:

- Aloe barbadensis (Aloe vera)-

It contains barbaloin, aloe-emodin, and aloenin, along with vitamins A, C, and E that enhance cellular regeneration and follicular repair. Its folic acid and vitamin B12 content contribute to reduced hair fall through 5-α-reductase inhibition.

- Phyllanthus emblica (Amla)-

It rich in ascorbic acid, gallic acid, and ellagic acid, exhibits potent antioxidant and antifungal properties. It improves scalp health, reduces dandruff, and inhibits 5-α-reductase, thus preventing androgenic alopecia.

- Allium cepa (Onion) and Allium sativum (Garlic)

It contain sulphur compounds such as allicin, diallyl disulphide, and allyl propyl disulphide that enhance collagen synthesis, exhibit antimicrobial activity, and stimulate blood circulation in the scalp, promoting follicular growth.

- Eclipta alba (Bhringraj)

The possesses wedelolactone and daucosterol, which induce the anagen phase in dormant hair follicles and nourish the scalp, enhancing hair strength and density.

- Thea sinensis (Tea), particularly its polyphenols (catechin, theaflavin, and EGCG), improves scalp microcirculation and inhibits hormonal pathways responsible for hair loss.
- Trigonella foenum-graecum (Fenugreek)

It contains saponins and flavonoids that inhibit dihydrotestosterone (DHT) binding to hair follicles, thereby reducing follicular miniaturization.

- Cocos nucifera (Coconut)

oil is abundant in lauric acid and triglycerides, which penetrate the hair shaft, reinforce protein binding, and protect against oxidative and environmental damage.

- Prunus amygdalus (Almond)

oil provides essential fatty acids, proteins, and biotin that enhance keratin synthesis and improve scalp microcirculation.

- Ocimum sanctum (Tulsi) is rich in eugenol and essential oils, conferring antimicrobial, antioxidant, and anti-inflammatory effects that strengthen hair roots and rejuvenate hair follicles.

IV. CONCLUSION

Alopecia Areata (AA) is a complex, multifactorial autoimmune disorder marked by non-scarring hair loss, with significant psychosocial impact, particularly in women and children. Its pathogenesis involves a combination of autoimmune attack, genetic predisposition, environmental triggers, hormonal imbalances, and associations with other autoimmune or atopic conditions, leading to disruption of normal hair follicle cycles. Clinical manifestations vary from patchy hair loss to complete scalp or body hair loss, often accompanied by nail changes and psychological distress.

Conventional treatments, including corticosteroids, minoxidil, immunotherapy, JAK inhibitors, and other pharmacologic agents, demonstrate variable efficacy but are frequently limited by side effects, relapse, and incomplete hair regrowth. This has driven growing interest in herbal and natural therapies, which offer safer, sustainable, and holistic approaches to hair restoration. Medicinal plants such as Aloe vera, Amla, Onion, Garlic, Bhringraj, Tea polyphenols, Fenugreek, Coconut oil, Almond oil, and Tulsi have demonstrated efficacy in promoting hair growth through antioxidant, anti-inflammatory, antimicrobial, and follicle-stimulating mechanisms, as well as improved scalp circulation and DHT inhibition.

Overall, integrating herbal interventions with conventional therapies may provide a synergistic, patient-friendly approach for managing AA, reducing the reliance on long-term pharmacologic treatment,

minimizing adverse effects, and improving both clinical outcomes and quality of life. Continued research into the mechanisms and efficacy of herbal agents is warranted to establish standardized, evidence-based protocols for the safe and effective management of alopecia areata.

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