

# Alopecia: Pathophysiology, Diagnosis, and Treatment Approaches

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## Abstract

**Alopecia, often known as hair loss, is a complex illness that can afflict people of all genders and ages, with serious psychological and social consequences. This review paper examines the pathophysiology, clinical manifestation, and diagnostic techniques of the several forms of alopecia, such as androgenetic alopecia and alopecia areata. The study underscores the significance of obtaining an accurate diagnosis via clinical assessment and histological analysis, since they are crucial in ascertaining the suitable treatment approaches. With differing degrees of efficacy and adverse effects, modern therapy approaches include topical and intralesional corticosteroids as well as more sophisticated medicines such JAK inhibitors and immunomodulating drugs. The difficulties in treating severe types of alopecia are also covered in the report, emphasizing the necessity of continuing investigations into the genetic and molecular pathways behind these disorders. Novel therapeutic approaches, such as gene therapy and stem cell research, present encouraging prospects for future medical interventions. The overall goal of this review is to present a thorough understanding of alopecia, its effects on patients, and the changing field of treatment options, emphasizing the need of personalized care in the management of this complex illness.**

**Keywords: Alopecia, Hair loss, Androgenetic alopecia (AA), Alopecia areata (Aa), JAK inhibitors, Psychological impact, Stem cell therapy**

## Introduction

Alopecia, also referred to as hair loss, is a complex disorder that can impact people of any gender or age. Both physically and psychologically, a patient's quality of life may be greatly impacted by hair loss. Alopecia can manifest in a number of ways, including cicatricial forms like lichen planopilaris, frontal fibrosing alopecia, and discoid lupus erythematosus, as well as noncicatricial forms like androgenetic alopecia, alopecia areata, and telogen effluvium. A thorough strategy is necessary for the diagnosis and treatment of alopecia, including both clinical examinations and diagnostic tests [1]. A variety of invasive, semi-invasive, and noninvasive techniques are used in patient assessment; each one offers important information about the underlying causes and possible therapies. To determine the amount and pattern of hair loss, invasive procedures like scalp biopsies, semi-invasive instruments like the trichogram, and noninvasive techniques like hair counts, microscopic assessments, and trichoscopy are frequently used. Together with a comprehensive clinical history, these diagnostic techniques aid in identifying the kind and degree of alopecia, which helps to select the best course of treatment. To determine probable causes of hair loss, such as recent systemic disease, weight fluctuations, or drug use, a thorough clinical history is necessary. Gynecological history and hormonal examinations are especially important

in women, particularly when androgenetic alopecia is present. Additionally, because deficiencies in these areas can exacerbate hair loss, laboratory testing is frequently required to evaluate thyroid function, iron levels, and vitamin D status [2], [3].

Medicinal interventions such as finasteride and minoxidil constituted the mainstay of alopecia treatment. These treatments, particularly in androgenetic alopecia, were vital because they stimulated hair growth and slowed the development of hair loss. Furthermore, immunomodulatory medications like corticosteroids were crucial in the management of autoimmune-related hair loss conditions including alopecia areata. Despite the fact that many people responded well to these treatments, their short- and long-term effects encouraged researchers to look into more sophisticated treatments [7].

### **Types of Alopecia**

The most frequent type of hair loss is androgenetic alopecia, also referred to as male or female pattern baldness. It is primarily caused by hormonal causes, especially dihydrotestosterone (DHT), as well as hereditary susceptibility. This produces diffuse thinning across the scalp in women, while it recedes the hairline and thins the crown in men. Patchy hair loss from autoimmune conditions like alopecia areata, in which the body's immune system targets hair follicles, can develop into more severe forms like alopecia totalis, which affects the entire scalp, or alopecia universalis, which affects the entire body. When a significant number of hair follicles enter the resting phase, a condition known as telogen effluvium results in temporary hair loss. This condition is frequently brought on by sickness, hormonal fluctuations, or physical or emotional stress. Alopecia permanente, also known as cicatricial alopecia, is characterized by the irreversible loss of hair resulting from the permanent death of hair follicles by inflammation. Discoid lupus erythematosus and lichen planopilaris are two probable causes of this disorder. Last but not least, tight hairstyles can generate constant stress on the hair, known as traction alopecia. If this tension is left untreated for an extended period of time, it can result in irreversible hair loss [2].

### **Androgenetic Alopecia (AA)**

AA commonly referred to as male and female pattern baldness is a genetically predisposed kind of hair loss that impacts both sexes, in general however manifesting differently in each gender. In males, it has a distinct progression commencing with a receding hairline at the temples and thinning at the crown, ultimately resulting in baldness in these regions. This pattern is categorized by the Hamilton-Norwood scale, indicating a transition from minor hair loss to severe baldness, characterized by the presence of only a horseshoe-shaped band of hair. In women, androgenetic alopecia generally presents as widespread thinning throughout the scalp, especially at the parting line, without a prominent receding hairline, and is categorized by the Ludwig scale. Female pattern hair loss is typically less severe than in males and predominantly impacts the crown rather than the frontal area. The primary underlying reason in both sexes is the sensitivity of hair follicles to dihydrotestosterone (DHT), which abbreviates the hair development cycle and results in follicular shrinkage. Nonetheless, hormonal fluctuations, especially in women undergoing menopause, significantly contribute to the onset of this illness. Both men and women have gradual hair thinning; however, the intensity and pattern of hair loss vary considerably due to hormonal and genetic differences [3].

### **Alopecia Areata (Aa)**

Alopecia areata (Aa) is an autoimmune disorder marked by abrupt hair loss in distinct, localized patches, typically manifesting as circular or oval bald spots. The scalp is the most frequently afflicted location (90%), however other body hair, including eyebrows, eyelashes, beard, underarm, and pubic hair, may also be involved. More

severe manifestations can lead to alopecia universalis, affecting the entire body, or specific regions such as the posterior scalp, known as the ophiasis pattern. The impacted skin typically appears unremarkable, devoid of discernible scaling or irregularities. Hair loss results from a sudden transition from the anagen (growth) phase to the telogen (resting) phase, typically manifested as localized shedding. A characteristic of AA is the existence of "exclamation point hairs," which are short, fractured hairs that narrow towards the scalp and broaden at the distal end. Hair pull tests conducted at the lesion's periphery frequently suggest active disease when six or more hairs are effortlessly extracted. Over time, separate areas of alopecia may converge, and regrowth usually initiates with white or blonde hairs due to a deficiency of pigment. Nail involvement, including pitting, Beau's lines, and other abnormalities, is a prevalent characteristic of AA [4].

### **Telogen Effluvium (TE)**

Telogen effluvium (TE) is characterized by non-scarring, generalized alopecia of the scalp, usually manifesting approximately three months following a precipitating incident. This condition is typically self-resolving, with hair loss persisting for around six months and impacting less than 50% of the scalp hair. TE, first delineated by Kligman in 1961, is marked by the widespread loss of telogen hairs. Kligman posited that irrespective of the stimulus, hair follicles prematurely conclude the anagen (growth) phase, swiftly progress into catagen, and then enter the resting (telogen) phase. Nonetheless, the mere observation of heightened telogen shedding does not determine the etiology of hair loss. Establishing the cause of TE necessitates a comprehensive medical history and relevant laboratory tests to exclude endocrine, nutritional, and immunological problems. A range of triggers has been associated with the onset of TE; nevertheless, its actual incidence is ambiguous due to insufficient thorough data, particularly for subclinical instances [4], [5].

### **Scarring Alopecia**

Scarring alopecia, also known as cicatricial alopecia, is an uncommon and irreversible disorder characterized by inflammation that obliterates hair follicles, resulting in permanent hair loss and scarring of the scalp. This illness encompasses multiple subgroups, including Lichen Planopilaris (LPP), Frontal Fibrosing Alopecia (FFA), Discoid Lupus Erythematosus (DLE), Central Centrifugal Cicatricial Alopecia (CCCA), and Folliculitis Decalvans. The precise etiology remains ambiguous but is believed to encompass autoimmune reactions, genetic predispositions, and environmental stimuli. Symptoms fluctuate, frequently encompassing erythema, pruritus, and unease prior to discernible alopecia. Diagnosis typically necessitates a scalp biopsy, and although therapies such as corticosteroids, immunosuppressants, and minoxidil may impede progression, they are incapable of regrowing lost hair. Timely identification and intervention are essential for disease management; however, the prognosis is contingent upon the subtype and severity, with certain individuals exhibiting slow deterioration despite treatment [5], [6].

### **Traction Alopecia**

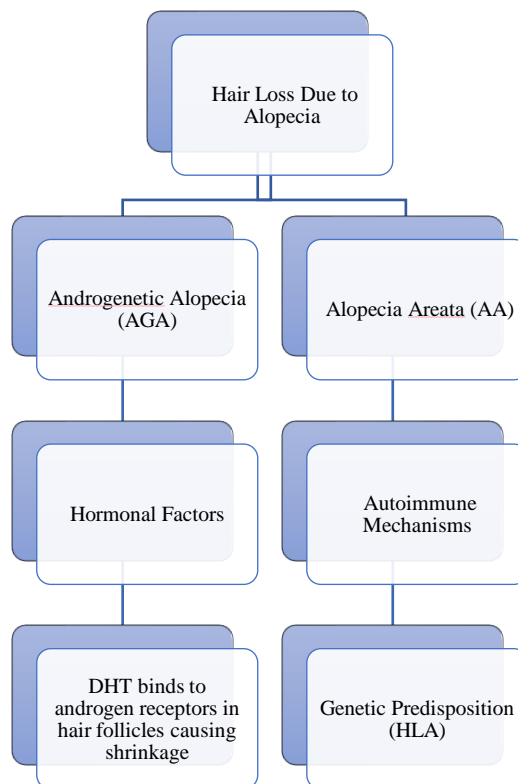
It is a type of hair loss caused by constant strain or pulling on the hair, often as a result of particular grooming routines or hairstyles. People with this illness are often those who wear tight hairstyles for extended periods of time, such as ponytails, cornrows, braids, or hair extensions. Although traction alopecia can affect people of any gender, women of African descent are more likely to experience it because to the popular practice of wearing tight weaves and braids. Hair thinning and loss, particularly at the hairline or temples, are the final consequences of the continuous tension on hair strands, which causes progressive damage and degeneration of hair follicles [7]. If the strain is released and the affected area is given time to heal, traction alopecia is reversible in its early stages.

However, in the event that the traction continues, the damage to the hair follicles can become irreparable, leaving scars and alopecia. The diagnosis is often based on observation and clinical history; first symptoms may include erythema, pain in the scalp, or small papules at the sites of hair traction. The focus of treatment is on reducing stress on the hair by avoiding chemical treatments that could worsen follicle damage and adopting looser hairstyles. In more severe cases, topical treatments like minoxidil or corticosteroids could be used to promote growth, while hair transplants are considered surgical procedures for permanent damage. Traction alopecia can cause irreversible hair loss, thus early detection and treatment are essential. In order to preserve healthy hair and scalp integrity, there is a greater emphasis on promoting safer hairstyling techniques as awareness of the disorder grows [7], [8].

**Pathophysiology**

Hair loss due to alopecia, including androgenetic alopecia (AGA) and alopecia areata (AA), is caused by a variety of biochemical, genetic, hormonal, and environmental causes. The understanding of these systems has significantly improved with recent research. The most common kind of hair loss, androgenetic alopecia (AGA), affects both sexes and is predominantly driven by hormones, specifically the androgen dihydrotestosterone (DHT). DHT binds to androgen receptors in hair follicles in genetically predisposed people, causing follicular shrinkage, a shortened hair growth cycle, and consequent thinning and loss of hair. The frontal and vertex areas of the scalp are where the hormonal effect is most noticeable.

Susceptibility to AGA is mostly influenced by genetic factors. Numerous genetic loci that increase hair follicle sensitivity to DHT have been identified by research, including those on chromosome 20p11 and the androgen receptor gene on the X chromosome. These results clarify the varying degrees of AGA severity across individuals and lay the groundwork for the development of customized treatments intended to alter the implicated hormonal and genetic pathways [9].



**Figure no 1: Pathophysiology of Alopecia**

In essence, alopecia areata (AA) is an autoimmune disease. The immune system attacks the hair follicles in alopecia areata, resulting in partial or complete hair loss. According to recent research, T lymphocytes—particularly CD8+ T cells act on the hair follicles, causing inflammation and upsetting the regular cycle of hair production. Alopecia areata (AA) genetic predispositions are closely linked to other autoimmune diseases, and the disease's course is greatly impacted by human leukocyte antigen (HLA) genes as depicted in figure no 1. Treatment for AA might become more difficult when environmental variables such as stress and infections exacerbate or trigger the autoimmune response [8], [10]. In both AGA and AA, the interaction between genetic predisposition and environmental variables is evident. In androgenetic alopecia (AGA), environmental factors such as stress, poor diet, and hair care practices can exacerbate hair thinning; however, in alopecia areata (AA), they can start or worsen autoimmune attacks on hair follicles. While the procedures in each case are different, they both highlight the significant effects of both hereditary and environmental factors [9].

### **Treatment**

It is treated with immunomodulating medications such as corticosteroids, anthralin cream (Psoriatec), and 5% minoxidil. Topical immunotherapeutic drugs are also employed, albeit there are difficult management regimes for these powerful agents. Examples of these agents are dinitrochlorobenzene, squaric acid dibutyl ester, and diphenylcyclopropenone.

There may be a need for a dermatology consultation or referral. While none of these substances stop hair loss, they all promote hair growth [11]. AGA therapy options are determined by a number of considerations, such as cost, hazards, practicality, and effectiveness. The intention is to stop the process of miniaturization and, if feasible, reverse it. Pharmacotherapy, surgery, and cosmetic devices are among the treatment options. Despite the high demand, there are only two FDA-approved drugs for the treatment of AGA: finasteride, an inhibitor of DHT synthesis, and minoxidil, a potassium channel opener.

Both drugs are useful in treating AGA when used consistently over an extended period of time. They may have negative effects, need lifelong care, and are expensive. Patients may opt for non-surgical treatment, which is commonly depicted in Table 1. Medical therapy is most effective when started in the early stages of hair loss. By preventing surrounding native hair from falling out, medicinal treatment can play a crucial role as an adjunct to surgical procedures, enhancing the overall aesthetic results [11], [12].

### **5 $\alpha$ -reductase inhibitors (5 $\alpha$ Ri)**

Male androgenetic alopecia (AGA) is largely caused by lack of the enzyme 5 $\alpha$ -reductase type 2 (5 $\alpha$ R2), which transforms testosterone into DHT; this is demonstrated by the absence of hair loss in these men. Of the two isoenzymes of 5 $\alpha$ -reductase, type II is found in hair follicles, the prostate, and the genitourinary tract, while type I is mostly found in the skin and liver. Treatment for AGA involves two drugs that target this enzyme: finasteride, which blocks 5 $\alpha$ R2, and dutasteride, which blocks both 5 $\alpha$ R1 and 5 $\alpha$ R2. Finasteride lowers DHT levels in the scalp, prostate, and serum by roughly 60–70%. It was authorized by the FDA in 1997 for male AGA. Topical finasteride has also been demonstrated to be beneficial in treating AGA in recent studies. Additionally, evidence indicates that finasteride with minoxidil is a more effective combination than either medication alone [13].

Treatment Type	Active Agents	Mechanism of Action	Usage	Common Side Effects
<b>Immunomodulating Medications</b>	Corticosteroids, Anthralin, Minoxidil	Suppresses immune response, enhances blood flow to hair follicles	Corticosteroids via injections/topical use, Minoxidil topically	Skin atrophy, contact dermatitis, possible systemic effects with oral steroids
<b>5<math>\alpha</math>-Reductase Inhibitors (5<math>\alpha</math>Ri)</b>	Finasteride, Dutasteride	Inhibits DHT production by blocking 5 $\alpha$ -reductase enzyme	Oral use, topical finasteride available in recent studies	Sexual dysfunction (reduced libido, erectile dysfunction)
<b>Minoxidil</b>	Minoxidil 2%/5% solution, 5% foam	Potassium channel opener that increases blood flow to hair follicles	Topically applied twice daily	Contact dermatitis, initial shedding (telogen effluvium)
<b>Combination Therapy</b>	Finasteride + Minoxidil	Combines DHT inhibition and increased follicle blood flow	Oral finasteride with topical minoxidil	Side effects related to individual medications
<b>Corticosteroids</b>	Triamcinolone acetonide, Prednisone	Anti-inflammatory, suppresses immune response	Intralesional injections, oral prednisone for severe cases	Skin atrophy, systemic side effects from oral prednisone

**Table no 1: Common Non-Surgical Treatments for Alopecia**

Trials spanning up to ten years have established the therapeutic efficacy of finasteride; nevertheless, hair loss recurs when therapy is stopped because DHT levels revert to their initial levels. Although most people tolerate the medication well, there have been isolated instances of sexual side effects such decreased libido, erectile dysfunction, and problems ejaculating. According to a study, finasteride has no effect on sperm production when used for 48 weeks. While most of the time these adverse effects go away with continuous therapy or when the medicine is stopped, some men experience chronic sexual dysfunction when they stop taking finasteride; this is now known as 5 $\alpha$ R inhibitor Withdrawal Syndrome [14].

### **Minoxidil**

When minoxidil was first created, it was intended to treat high blood pressure. However, when patients started experiencing excessive hair growth, or hypertrichosis, minoxidil's potential for hair growth came to light. A topical treatment for pattern hair loss was created as a result of this observation. By converting minoxidil to minoxidil sulfate when administered topically, it activates potassium channels, relaxing blood vessels and boosting blood flow to hair follicles. Research on hair growth has demonstrated that minoxidil preserves the health of hair follicles and inhibits their degeneration, resulting in stronger, thicker hair. Instead of greatly increasing hair count, its main effect is to enhance the diameter of already existing hairs [1], [7].

2% and 5% solutions of minoxidil, as well as a 5% foam, are commonly accessible over-the-counter. Although initial shedding (telogen effluvium) frequently happens within the first eight weeks of medication, results are typically assessed after six months of use. Patient education should be provided. Relatively typical side effects include contact dermatitis, which can be avoided by using the foam formulation, which doesn't contain propylene glycol, or by reducing the concentration. It is imperative to apply minoxidil twice a day in order to sustain hair growth, as stopping treatment causes the results to reverse [1], [8], [11].

### **Corticosteroids**

The most common treatment for alopecia areata is corticosteroids, namely intralesional injections of triamcinolone acetonide (Kenalog). The usual dosage is up to 3 mL of a 5 mg/mL solution injected into the mid-dermis using a 30-gauge needle at many locations separated by 1 cm. The process can be repeated every four to six weeks, with hair regrowth typically becoming evident after four weeks. Injections into the mid-dermis should be carefully delivered to prevent localized skin atrophy. Topical corticosteroids can be applied twice daily; for best effects, combine them with anthralin or minoxidil. However, they are not as effective as injections. Although more severe types of alopecia areata can be effectively treated with oral corticosteroids like prednisone, their usage is decreasing because of possible adverse effects. Adults over 60 kg who have severe instances may begin taking 40 mg of prednisone daily and progressively reduce the dosage over a period of six weeks. A lower starting dose of 20 mg every day or every other day is advised for milder types, and after the disease stabilizes, a gentle tapering schedule should be followed [15].

### **Managing Alopecia Treatment in the Pharmacy Setting**

In a pharmacy context, managing alopecia entails teaching patients about the disorder and walking them through the various treatment options. When it comes to helping patients comprehend the chronic nature of alopecia, particularly alopecia areata (AA) and androgenetic alopecia (AGA), pharmacists are crucial. For a long time, minoxidil and finasteride have been known to be effective treatments for AGA. After being discovered to encourage hair growth, minoxidil—which was once created as a high blood pressure medication—became a crucial over-the-counter treatment for AGA. It strengthens already-existing hair by boosting blood flow to the hair follicles. Finasteride is a prescription medication that lowers DHT levels and has been successfully used to halt male pattern hair loss. Pharmacists should give patients thorough instructions on how to administer these therapies correctly, manage adverse effects, and make sure they understand that continued use is necessary to sustain outcomes. Additionally, patients need to be made aware of the possibility of early shedding when using minoxidil and the potential for sexual adverse effects while using finasteride [6].

Intralesional injections of corticosteroids, especially triamcinolone acetonide, have proven to be a highly efficacious treatment for people suffering from alopecia areata. Although topical corticosteroid treatments are less effective than injections, pharmacists can nonetheless provide advice on them. Important facets of pharmacy management include instructing patients on how to take these drugs, keeping an eye out for adverse effects like skin shrinkage, and sending them to dermatologists for more sophisticated therapy as needed. In order to assist patients in coping with the psychological effects of hair loss, pharmacists might also prescribe cosmetic options like volumizing creams and hair fibers. Effective patient care in a pharmacy context requires these tactics in addition to an emphasis on encouraging long-term adherence to treatment.

### **Limitation of current Treatment and future directions**

The long-term management of patients with alopecia is complicated by the limitations of the pharmaceutical

treatments for alopecia, especially androgenetic alopecia (AGA) and alopecia areata (AA). Results with AGA treatments such as finasteride and minoxidil vary; some people show minor improvement, while others see significant hair regrowth. Rather of creating new hair follicles, minoxidil primarily increases the width of existing hair, which limits its effectiveness in more severe cases. It also needs to be applied continuously, twice a day, and stopping treatment will reverse any progress made. Finasteride can result in side effects like decreased libido, erectile dysfunction, and, in rare instances, more chronic problems like post-finasteride syndrome, even if it is successful in lowering DHT levels and decreasing hair loss. Patients are typically discouraged from long-term use—especially younger men—by these negative effects [11], [13]. Topical or intralesional corticosteroids continue to be a common treatment for alopecia areata. Oral corticosteroids, while useful in managing severe instances, have significant systemic adverse effects that limit their long-term usage. Nevertheless, they frequently fail to prevent relapses. Current treatments are frequently insufficient for people with more severe forms of AA, such as complete hair loss (alopecia totalis or universalis). The creation of more specialized and efficient treatments has been hampered by a significant knowledge vacuum on the genetic and molecular causes of alopecia [13].

JAK inhibitor research has showed promise in treating alopecia areata by focusing on the immune response that causes hair loss in patients taking drugs like tofacitinib and ruxolitinib. Patients with more severe forms of AA have responded well to these therapy, providing optimism for further improvements in treatment. Furthermore, research on stem cells and gene therapy has surfaced as viable methods for regenerating or restoring hair follicles. Although these medicines are still in the experimental stage, they signify a move toward more individualized and maybe curative treatments; nonetheless, obstacles like costly development and the requirement for lengthy clinical trials may cause a delay in their general availability [1], [6].

## Conclusion

Alopecia is a complex medical and psychological condition that affects individuals across diverse demographic groups, requiring a nuanced understanding of its various forms and impacts. The condition not only causes physical changes but also carries a significant psychological burden, particularly in women, due to societal pressures and beauty standards. The importance of early and accurate diagnosis using tools such as trichoscopy and scalp biopsies has become central to tailoring effective treatment plans. Conventional therapies like minoxidil and corticosteroids remain vital in managing hair loss, though their success often depends on the specific type and severity of alopecia. Newer treatments, such as JAK inhibitors, offer promising hope for more individualized and effective therapies, especially for those with severe or treatment-resistant forms of the condition.

Advances in understanding the biology and genetics of alopecia are driving research toward more innovative solutions, including gene therapy and stem cell treatments that target the underlying causes of hair loss. However, challenges remain in managing chronic and severe cases, highlighting the need for continued research to explore the genetic and molecular mechanisms involved. A multidisciplinary approach that integrates medical treatment with psychological support is essential to improving the overall well-being of those affected by alopecia. As the field continues to evolve, collaboration across medical and psychological disciplines will be key in developing new strategies that can improve both the physical and emotional outcomes for patients.

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