

# REVIEW ON ALOPECIA: DISEASE AND CHARACTERISTICS, CLINICAL EVALUATION, PSYCHOLOGICAL IMPACT & AVAILABLE TREATMENT OPTIONS

## Abstract:

Alopecia Areata (AA) is a frequent recurrent dermatological autoimmune illness that causes patchy regions of hair loss on the scalp and/or body. It can afflict people of any age or skin type, and it affects both men and women. It is necessary to develop new diagnostic procedures. Scalp dermatoscopy is a potential new technique for diagnosing scalp and hair diseases that may be difficult to diagnose otherwise. In adults, high-potency topical corticosteroids (TCs) such as 0.05 percent clobetasol propionate in different formulations or 0.25 percent desoximetasone cream are often used, with response rates of around 47 percent and 60%, respectively. Individuals with stable conditions can benefit from CI therapy provided at home, which can be just as safe and effective as CI therapy administered in a clinic setting.

Keywords: Alopecia Areata, dermatoscopy, clobetasol propionate, CI therapy

## Background

Scalp alopecias are a group of dermatologic illnesses that encompass a wide range of different conditions. They are among the most prevalent dermatologic conditions. A comprehensive physical examination and a detailed review of the patient's history are typically sufficient to confirm the right diagnosis. The need for a scalp biopsy may be essential in some circumstances, such as those of cicatricial alopecia. Histopathologic traits, on the other hand, are not necessarily indicative of a diagnosis. As a result, it is necessary to develop new diagnostic procedures. Scalp dermatoscopy is a potential new technique for

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diagnosing scalp and hair diseases that may be difficult to diagnose otherwise. A large number of publications have been published about the video dermatoscopic aspects of alopecias in various stages [1].

## Alopecia Areata

Alopecia Areata (AA) is a frequent recurrent dermatological autoimmune illness that causes patchy regions of hair loss on the scalp and/or body. It can afflict people of any age or skin type, and it affects both men and women [2]. In the United States, the cumulative lifetime incidence of Alzheimer's disease (AA) is estimated to be roughly 2 percent of the population [3]. Among contrast, the worldwide incidence of hepatitis C varies from 0.57 percent to 3.8 percent in individuals who present to a hospital setting [4-7]. The manner in which the illness manifests itself varies from one sufferer to the next, ranging from hair loss in distinct, well-circumscribed circular or oval spots on the scalp or body to complete hair loss on the entire scalp (Alopecia Totalis) and to complete hair loss on the entire body (Alopecia Universalis) (Alopecia Universalis) [2, 8].

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Infection can affect any hair-bearing location, while the scalp, beard area, and brows are the most noticeable [9]. Men are more often than women to be diagnosed with alcoholism [10], which may explain why, despite the fact that both men and women are affected equally by the disease. The clinical indications of AA extend beyond the hair follicle, with nail and eye pathologies being more common than hair follicle diseases. AA patients with nail involvement have been observed in 2 to 44 percent of cases, and it is more prevalent in children and adolescents (>40 percent) than in adults (20 percent), particularly in severe instances [11-13]. A typical finding [11] is nail pitting and trachyonychia, with leukonychia [14], red lunulae, and transverse grooving of the nail plate [15] following closely after. AA has been linked to a variety of ocular problems, including focal retinal hypopigmentation, lens opacities, cataract, Horner's syndrome, miosis, and palpebral ptosis, among others

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[16]. Within a year following the first occurrence of alopecia, 80 percent of patients experience spontaneous hair regrowth; however, recurrence or advancement of the condition cannot be anticipated and is completely reliant on prognostic variables exhibited by the individual [4-5, 7, 17-18].

Alopecia Areata is not limited to the gender and can affect male and female sex equally and is independent of the age group of patients. It has been found that about 0.1% to 0.2% of the population has been suffering from Alopecia Areata. It has been reported that diagnosis of AA among males is diagnosed at earlier age as compared to female sex. Some studies have also reported that pediatric populations is more prone to AA [19].

AA is neither linked with the inflammation nor with the scarring signs on the scalp, but is a spontaneous loss of hair with well-circumscribed patches of hair loss on the scalp, [20]. Pull test can be used to confirm the active state of the disease, especially at the periphery of the lesion [21]. Patients with AA are found to be asymptomatic in most of the cases. In some cases the occasional reporting of tingling, itching, and dysesthesia is found before the events of hair loss. AA at severe situation may include the loss of hairs all over the scalp, which is known as Alopecia Totalis (AT). If the disease progresses to the whole body and scalp, it is known as Alopecia Universalis (AU). Loss of hairs at beard is likely to occur more (50.5%) than the involvement of scalp (39.3%) [22]. It is found that the pathogenesis of AA is incompletely understood but it is viewed as an organ-specific autoimmune disease. The studies showed 1.7% life time risk of AA [23]. The treatments available are not sufficient for AA and the only FDA-approved treatment option available for the management of hair loss includes finasteride, dutasteride and minoxidil which are not only the expensive options but possessed various adverse effects [24].

### **Androgenetic Alopecia**

Male pattern baldness, also known as androgenetic Alopecia, is the most prevalent kind of hair loss in both men and women. It is characterised by gradual reduction in hair width, length, and colour over time. The genetic inheritance of androgenetic alopecia is widely understood, yet the genes responsible for the condition have not yet been identified. Several factors, including medicines, acute stresses, weight loss, and pregnancy, can cause androgenetic alopecia, which can then be aggravated by the disease known as telogen effluvium. To give an example, medicines that have androgenetic effects, such as oral

contraceptives that include androgenic progestins and hormonal therapies for menopause, can cause or aggravate androgenetic alopecia. Because androgenetic alopecia is a degenerative condition, therapies should be initiated early and continued for an extended period of time to ensure effectiveness[25].

### **Cicatricial Alopecia**

Hair follicle inflammation and subsequent destruction are two characteristics of Cicatricial Alopecia, which results in irreversible hair loss and is caused by inflammation and subsequent destruction. It is possible to have either primary or secondary cicatricial alopecia. Primary Because it is caused by illnesses that mostly affect the hair follicle, Cicatricial Alopecia can be classified as either lymphocytic or neutrophilic, depending on the kind of inflammatory cell that predominates in the hair follicle. All of the conditions listed above, including lichen planopilaris and its clinical manifestation frontal fibrosing alopecia, as well as discoid lupus erythematosus, are instances of lymphocytic Cicatricial alopecia. There are several types of lichen planopilaris, and one of them is lichen planopilaris that damages hair follicles. Secondary Cicatricial alopecia has been associated with sclerosis, granulomatous inflammation (such as sarcoidosis), and malignancy, among other conditions. The pathological investigation of Cicatricial alopecia is required for the diagnosis. Early detection of cicatricial alopecia is critical since the objective of treatment is to decrease the course of the disorder[25].

### **Chemotherapy Induced Alopecia (CIA)**

While various kinds of chemotherapy occur, the drug itself can be generally interpreted as a medication that prevents cell division or development. Alopecia considered as most severe skin side effects that damages the skin. Several drugs influence the growth of hair and a few others induce loss of hair, the symptoms of the illness of which are important for hair growth as a direct consequence of drug action on different cells. These cells are keratinocytes, hair matrix cells, peri-follicular blood vessels, in addition those of hair bulbs connective tissues. Keratinocytes considered a first affected cite for environmental or xenobiotic damaging effects. This can be attributable to the normal feature that up to 90% from total HFs are in a rapid growth process and the high blood flow rate around hair bulbs contributes to a strong bioavailability of many drugs at these locations [26-27].

There appear to be two types of alopecia that can be generated by chemotherapy: telogen effluvium and anagen effluvium. Telogen effluvium is a kind of alopecia that appears to be caused by chemotherapy. Despite the fact that telogen Effluvium is regarded less severe than anagen Effluvium, it often leads in significant thinning, with the patient losing around half of their hair while undergoing cancer treatment. In comparison to anagen effluvium, which also causes alopecia, this form of CIA is less noticeable to the general public's eyes. Since chemotherapy medications attack rapidly proliferating cell groups and kill neoplastic cells as well as rapidly growing normal cells such as anagen hair matrix cells, the anagen effluvium is frequently associated with chemotherapy. Hair development occurs during the anagen phase, which is the process during which productive growth occurs. Anagen is one of three phases of hair development, and it is the period during which productive development occurs. It has been reported that chemotherapy disrupts the anchoring of hair to the point that it falls out of the scalp or breaks down before reaching the top surface of the scalp[28].

For many cancer survivors, alopecia considered a traumatic experience. The change in appearance resulted from alopecia may cause some survivors to encounter a sense of loss of identity and discomfort as they face difficulty to accept their distorted identity. The quickly growing HF's of cells within the anagen stage suffers from systemic chemical toxins and from premature apoptosis, which contributes to earlier catagen beginning [29].

### **Alopecia Inducing Factors**

Alopecia is caused by a variety of factors, including hormonal disorders, impaired hepatic and liver function, lupus erythematosus, heat and chemical injury, conception, and fungal infection. Other factors include chemotherapy, fatigue, diabetics, vicious disorders, injuries, autoimmune diseases, and rheumatoid arthritis, among others [30]. The age at which the disease first manifests itself is also considered to be a less favourable prognosis factor [19].

There has been evidence that AA is associated with a range of medical illnesses, including asthma, atopic dermatitis (AD), allergic rhinitis (AR), thyroid problems, vitiligo, psoriasis, and rheumatoid arthritis [19, 24, 26-30]. Werth and colleagues also discovered a higher frequency of AA in individuals with systemic lupus erythematosus [31].

### **Influence of Hereditary Factors**

The influence of hereditary factors is also seen in AA which is further aggravated by environmental triggers such as emotional/physical stressors including exposure to ultraviolet light, chemicals, physical trauma and emotional disturbances, hormones & infections [31]. According to studies, at least 23% of patients had an emotional incident or a personal psychological difficulty prior to the onset of AA [32-33]. 10% to 42% of people with AA have a positive family history of the illness [34-36]. According to prior studies, AA is more prevalent in the younger age group with 82.6%-88% of patients experiencing their initial symptoms by age 40 years while 40.2% by age 20 years [4, 12 and 37]. It has been shown that AA has a polygenic origin and so cannot be linked to a single gene locus, multiple genes participate creating an increased propensity to acquiring the illness [38].

### **Epidemiology of Alopecia**

The occurrence of hair loss is reported to increase gradually with increasing age in each of these males and females in populous-based surveys of White people aged 20-70 aged, often utilizing updated classification measures, the occurrence of hair loss ranged from 45% to 90%. In the Hamilton-Norwood classification scale, the incidence of alopecia was approximately 50% [39].

### **Psychological impact of Alopecia Areata:**

Hair has a significant social and psychological role in human life, hair loss can be regarded as a mark of abnormality and a failure to live up to societal standards of physical attractiveness. There are studies to support the idea that alopecia is mentally destructive, causes great emotional pain and leads to personal, social, and occupational issues [40]. (32) Alopecia patients, both adults and children have a higher risk of developing psychiatric illnesses than the general population, implying that persons with alopecia are more likely to experience a major depressive episode, anxiety disorder, social phobia, or paranoid disorder [41-44].

An article outlines the suicidal deaths of four Australian males aged 14–17 years with no known pre-existing psychiatric issues who were affected by AA and had social disengagement that began after the beginning of alopecia [45]. This demonstrates how alopecia may have a detrimental psychological influence on a person's life and can have a profound impact on how individuals think about themselves and others.

### **Quality of life (QOL)**

Quality of life (QOL) studies have shown to be quite beneficial in identifying how a disease impacts affected patients. According to studies, nearly 50% of individuals with AA have low health-related QOL [46]. Even when compared to patients with chronic skin conditions like psoriasis and atopic dermatitis [47-48]. The AA patients with the lowest QOL scores were <50 years old, female, and had more extensive hair loss [46]. The age at which AA initiates has an impact on the rate of psychiatric comorbidity, patients aged <20 years have a higher chance of depression, whereas those aged 40–59 years have a higher risk of anxiety and obsessive-compulsive disorder (OCD) [43].

### **Clinical Evaluation**

Alopecia Areata is difficult to diagnose due to its wide spectrum of symptoms and overlapping signs with other dermatological conditions eg. Female pattern hair loss (FPHL) and Telogen Effluvium (TE) [34, 49]. One of the most important aspects in identifying a sickness or determining the existence of another clinical problem is the clinical history [50]. With the use of procedures like the hair pull test and the Jacquet's sign, a thorough physical examination will allow an accurate diagnosis to be made aiding on to the clinical history [50, 51]. Olsen et al. created the severity of alopecia tool (SALT) score to quantify the severity of AA and define the afflicted region [52-55]. The surface area of the scalp is split into four sections (posterior 24%, superior 40%, temporal 18% each). The percentage of hair loss in each of the four sections is calculated and totaled. Severe AA was characterized as a SALT score of more than 50% [53-54]. The Alopecia Areata Progression Index (AAPI), created in 2016, also considers the results of hair pull tests and dermoscopy [54].

During a physical examination, the appearance of the nails and hair should also be addressed as they are commonly involved in AA patients [11-13]. The clinical history could also be able to identify a positive family history of Alopecia Areata or other autoimmune diseases linking to AA.

### **Treatment Options**

There is no agreed-upon standard treatment guideline for AA. Each patient should be advised before starting therapy that there is currently no cure for AA and that all treatments are aimed at decreasing hair loss and increasing regeneration. Treatment does not guarantee regrowth, and due to AA's unpredictable nature, relapse is always a possibility [56]. The age, illness activity, disease severity, and therapeutic responses of each patient determines treatment

options. Patients with mild types of AA are usually advised to avoid using unnecessary medications because spontaneous regrowth is anticipated [57], which has been seen in 34%-50% patients within one year after onset of AA [58].

### **Corticosteroids**

This class of drug is further divided into topical (TC), systemic and intralesional (IC) according to the mode of administration. In both the adult and paediatric population, TCs are considered the first-line treatment for patch-type AA [59-61].

In adults, high-potency topical corticosteroids (TCs) such as 0.05 percent clobetasol propionate in different formulations or 0.25 percent desoximetasone cream are often used, with response rates of around 47 percent and 60%, respectively. The use of mid-potency TCs in children, on the other hand, is becoming more common. In one study, the use of 5 percent minoxidil in conjunction with other treatments resulted in an 80% response rate. The use of intralesional corticosteroids (IC), usually triamcinolone acetonide at concentrations of 2.5–10 mg/mL at 4–6-week intervals as monotherapy or in combination with TCs is common in adult patients with chronic inflammation. In general, the response rate of IC ranges between 64 and 97 percent [52, 62-63].

In the treatment of Alopecia Areata, systemic corticosteroids (SCs) are one of the most commonly used medications. In a range of AA types, it has been demonstrated that SCs are efficacious. Typically, oral daily therapy with a beginning dosage of 0.5 mg/kg of prednisolone and a tapering over a 6- to 12-week period has been prescribed. SCs have been used for decades, but the high risk of recurrence after dosage reduction or termination, along with the side effects (such as suppression of the pituitary-adrenal axis and weight gain, as well as ocular and bone abnormalities, as well as worsening hypertension or diabetes) during long-term therapy, has kept them from being widely used [64-68].

### **Minoxidil**

Minoxidil, a vasodilator used to treat high blood pressure, has been linked to hair growth and is used to treat a variety of hair loss illnesses, including AA [69]. The linear growth rate of hair re-growing within an AA patch can be accelerated by topical minoxidil [70]. Topical minoxidil is frequently used in conjunction with other treatments, such as corticosteroids [71-72]. According to a study, low-dose (0.25–5 mg) b.i.d oral administration of minoxidil

improved clinical outcomes in 18–82.4 % of patients, with only minimal side effects such as fluid retention, palpitations, and face hypertrichosis [73]. Before beginning treatment, inform the patient that the medication must be continued for at least three to four months to show any benefit, and that stopping will reverse the minoxidil effect [56].

### **Contact Immunotherapy**

Whenever more than 50 percent of the scalp is afflicted, contact immunotherapy is the first line of treatment [56]. Topical sensitizers such as diphenylcyclopropenone (DPCP) and squaric acid dibutylester (SADBE) are used to begin and sustain a contact allergic reaction in order to stimulate hair regrowth. After treatment, mild to severe redness and/or itching are observed in the treated area for up to 24 hours. Individuals with stable conditions can benefit from CI therapy provided at home, which can be just as safe and effective as CI therapy administered in a clinic setting. According to the National Cancer Institute, regional lymphadenopathy, urticaria, widespread eczema, and localised vitiligo are only a few of the adverse effects of CI [65, 74-76].

### **Other treatment options**

Other therapies include anthralin, azathioprine [77], cyclosporine, methotrexate, sulfasalazine [51], adalimumab [78], tofacitinib, and ruxolitinib [79-80]. Weight gain, avascular necrosis, hypertension, diabetes, sleep problems, mood swings, acne, allergy sensitivity, abnormal hair colouring, and even disorders like vitiligo, which can be induced by diphenylcyclopropenone, should all be carefully monitored [51, 81-82].

#### COMPETING INTERESTS DISCLAIMER:

Authors have declared that no competing interests exist. The products used for this research are commonly and predominantly use products in our area of research and country. There is absolutely no conflict of interest between the authors and producers of the products because we do not intend to use these products as an avenue for any litigation but for the advancement of knowledge. Also, the research was not funded by the producing company rather it was funded by personal efforts of the authors.

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