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Research Article

**ALOPECIA: EXPLORING CONVENTIONAL TREATMENTS  
AND THE PROMISING POTENTIAL OF HERBAL MEDICINE**Sumit Kumar Tantway<sup>1\*</sup> and Mukesh Patel<sup>1</sup>

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**Abstract:**

*Hair, a vital component of the human body, originates from the skin's ectoderm and acts as a protective appendage. It includes two types: terminal hair, which is dark and thick, and vellus. Hair growth follows three cyclical phases: anagen, catagen, and telogen. Typically, 50–100 hairs are shed daily; excessive loss leads to a condition known as alopecia. Alopecia, characterized by hair loss, affects millions worldwide and presents in various forms, each with distinct etiologies and clinical manifestations. The primary types include androgenetic alopecia, alopecia areata, telogen effluvium, and cicatricial alopecia. While numerous treatment options are available, ranging from topical applications like minoxidil and corticosteroids to systemic therapies such as finasteride and immunosuppressants, these often have limited efficacy and potential side effects. Therefore, the quest for more effective and safer treatments continues. Herbal medicine has emerged as a promising alternative, offering natural remedies with fewer side effects. Herbs such as ginseng, saw palmetto, rosemary, and peppermint essential oils have demonstrated potential in promoting hair growth and improving scalp health. These herbal treatments are valued for their holistic benefits and minimal adverse effects, making them an attractive option for many patients. Integrating herbal remedies with conventional therapies could provide a more comprehensive and personalized approach to managing alopecia. This review underscores the need for ongoing research into both conventional and herbal treatments to develop effective strategies for combating hair loss and improving patients' quality of life.*

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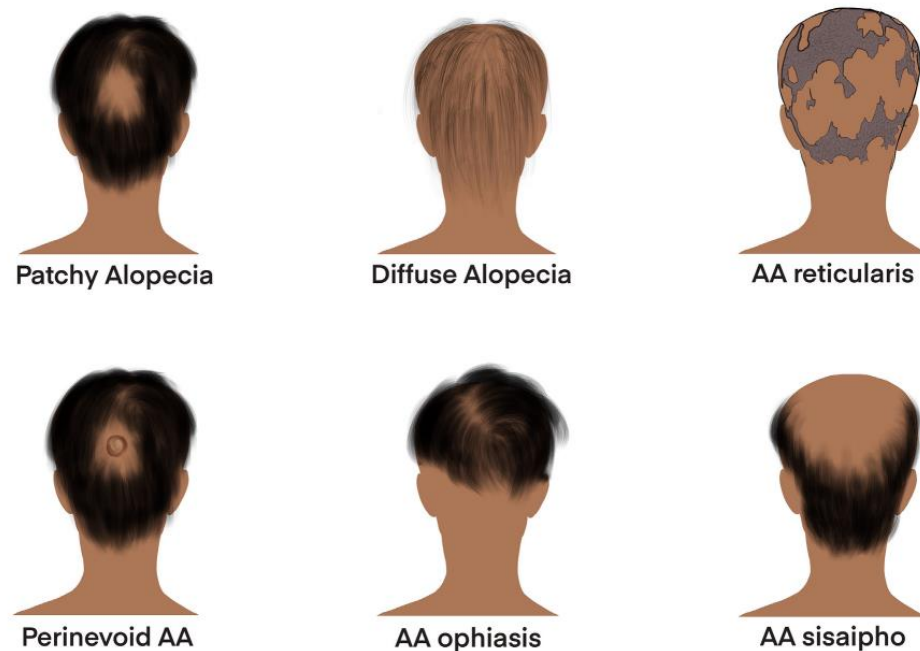


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**INTRODUCTION:**

One of the essential components of the body, hair is made from the ectoderm of the skin and serves as a protective appendage. In addition to nails and sebaceous glands, the integument has an accessory structure.<sup>[1]</sup> There are two kinds of hair: terminal hair and vellus hair. Terminal hairs are darker, thicker, and sometimes curling, whereas vellus hairs are thin, light-colored, and straight. Terminal hair grows on the scalp, in the area between the eyebrows and eyelashes, and on the newborn. Because they are independent of testosterone levels, these hairs are also known as nonsexual hair. An increase in plasma testosterone levels leads to the development of both sexual and ambisexual hair. 5  $\alpha$ -reductase transforms the testosterone in circulation into dihydrotestosterone (DHT). Ambisexual and sexual hair growth is caused by this DHT's action on hair follicles. There are three cyclical periods to hair growth. Phases of anagen, catagen, and telogen.<sup>[2]</sup> Generally speaking, 50–100 hairs randomly fall out each day. Alopecia is the term for a condition in which there is an average daily growth of more than 100 hairs lost. Alopecia areata is a medical illness primarily characterized by hair loss from the scalp and other regions of the body, often all at once. Because of the development of bald patches, it is also occasionally referred to as male pattern baldness or spot baldness. This type of alopecia typically begins as one or more small, smooth, circular bald patches on the head and can proceed to full body or scalp hair loss. Males are more likely than females to experience alopecia. It might be because men have higher levels of the androgenic hormone, testosterone. Treatment challenges arise from the lack of knowledge regarding the precise cause of alopecia.<sup>[3]</sup> There are several allopathic medications on the market for the treatment of alopecia, including minoxidil and finasteride. However, many of these do not meet the necessary standards and have a variety of side effects, including hypotension, recurrence of alopecia, loss of libido, impotence, decreased ejaculate volume (each in 3-4%), lip swelling, skin rashes, etc. Management of alopecia with agent

devoid of any side effect is still challenge to the medical profession. Current survey suggests that, in many developing countries, a large proportion of the population relies heavily on traditional practitioners and medicinal plants to meet the primary health care needs. Although modern medicine may be available in these countries, herbal medicines have often maintained popularity for historical and cultural reasons. Human beings and their ancestors have always been afflicted by disease. It is quite possible that human beings are relied on medicinal plants for a considerable time. The medical community is still faced with the difficulty of managing alopecia with a drug that has no adverse effects. According to a recent poll, a sizable section of the populace in many developing nations primarily depends on traditional healers and medicinal herbs to cover their basic medical needs. For historical and cultural reasons, herbal treatments have frequently remained popular even if modern medicine may be accessible in certain nations. Disease has plagued humans and their ancestors for all recorded history. It's probable that for a very long time, humans have relied on therapeutic plants. According to Sofowara, humans were aware of the medicinal qualities of plants as early as 5,000 years ago. Many significant contemporary medications, including atropine, morphine, and digoxin, were first identified by studying the ancient healing practices of native Americans. In the cosmetic and hair care industry, natural products are strongly endorsed. Approximately 1000 plant extracts have been tested for their ability to stimulate hair growth; proanthocyanidine from grape seeds (*Vitis vinifera*) and [beta]-sitosterol from saw palmetto (*Serenoa serrulata*) have demonstrated impressive results.<sup>[4]</sup> With the development of allopathic or modern medicine, scientists' focus shifted from plant sources to synthetic preparations as the foundation for contemporary medications.<sup>[5]</sup> Many herbal medications are touted by India's traditional medical system as aids in promoting hair growth, but their usage is restricted due to a lack of scientific backing.



**Fig. 1.** Clinical classifications of alopecia areata variants.

#### **TYPES OF ALOPECIA <sup>[6]</sup>: -**

1. **Alopecia Areata (primary stage)-** Alopecia areata is a prevalent autoimmune condition that causes hair loss on the scalp and other areas. One or more small, round, smooth, non-scarring spots are typically where it begins.
2. **Mild Transient Alopecia Areata-** Patient who frequently experiences temporary alopecia areata but does not develop universalis or totalis.
3. **Transient Alopecia Areata -** Patients with progressive Alopecia areata may advance to Alopecia totalis or Alopecia universalis.
4. **Ophiasis Alopecia Areata -** Alopecia areata of the ophiasis variety manifests as band-like hair loss. Since most medications have a delayed effect on the temporal or occipital regions of the head, where it mostly occurs, treatment can be more challenging.
5. **Alopecia Totalis -** Complete hair loss on the scalp.
6. **Alopecia Universalis -** Hair loss over the body, encompassing the lashes and eyebrows.
7. **Tricotilomania -**Compulsive pulling or repetitive self-pulling by the patient is the term used to describe this form of hair loss.
8. **Traction Alopecia -** Tight hairstyles can lead to significant traction at the hair roots, which can result in traction alopecia.
9. **Chemotherapy and hair loss -** Chemotherapy is only used to treat cancer patients; yet, it also damages healthy cells and hair follicles. This type of alopecia is known as anagen effluvium and results in hair loss.
10. **Diffuse Alopecia-** excessive hair loss throughout the scalp that doesn't result in a patch.
11. **Hair loss due to side effect of the beauty treatments-** For certain people, strong chemicals used in beauty treatments such as hair color, dye, straightening, softening, rebounding, perming, etc., can cause hair loss.
12. **Telogen effluvium (TE) and chronic telogen effluvium-** Dietary deficits and crash dieting (CTE) Telogen effluvium-type hair loss can be brought on by high fever,

anemia, blood loss, hormonal imbalances, pregnancy, and other factors. Effluvium signifies letting go, whereas telogen denotes the hair's resting period.

## CAUSES OF ALOPECIA

### Seasonal variation.

Seasonal variations in hair production are observed in numerous mammalian species. Two molting episodes, during which hair or fur is shed in early autumn to replace a summer coat with a winter coat and again in spring to replace a winter coat with a summer coat, are a frequent pattern in some mammals (such as ferrets).<sup>[7]</sup> Certain species also experience a shift in their hair color. One such species is the arctic hare, whose coat becomes white in the winter and brown in the summer. These seasonally driven hair changes are linked to changes in hormone levels as well as differences in the duration of the day.<sup>[7]</sup> Long days in temperate climates are linked to lower levels of prolactin and melatonin as well as shorter hair (summer coat); on the other hand, short days are linked to greater levels of both hormones and longer hair (winter coat). Prolactin has a well-established effect on seasonal molting patterns in Djungarian hamsters, goats, sheep, and deer, but little is known about how seasonal variation on pelage is controlled in nonhuman primates.<sup>[8]</sup> An analysis reveals that prolactin-treated mice have delayed hair renewal cycles, providing additional evidence for the hormone's involvement.

### Aging.

The aging process has also been linked to hair loss. As people age, even those who do not exhibit noticeable hair loss exhibit a decline in both mean hair density and anagen hair growth rate.<sup>[9, 10]</sup> Figure 1 depicts the thinning of hair that is typical of elderly rhesus monkeys. Age has been the subject of two studies<sup>[11, 12]</sup> examining hair loss in nonhuman primates. When older and younger rhesus monkeys (mean ages, 25 and 10 years, respectively) were compared, it was found that the older monkeys had thinning hair and skin abnormalities, such as increased areas of reddish skin, scaling, wrinkles, and subacute dermatitis.

### Nutritional factors.

While vitamin and mineral deficiencies have been proposed as hair loss causes<sup>[13]</sup>, a thorough evaluation of the nutritional characteristics that could control hair production has not been done. The three main areas of attention have been protein, zinc, and vitamin D. Research on the connection between zinc and hair loss illustrates the idea that an excess or

deficiency of any material might have negative consequences. Alopecia in children, talapoin monkeys, marmosets, and rhesus and bonnet macaques has been linked to moderate to severe zinc deficiency. Skin and hair significantly improved in each of these cases when zinc was added to meals or drinking water. Alopecia was not consistently produced by a lesser deficit that was enough to impede skeletal growth and maturation in adolescent rhesus monkeys.<sup>[14]</sup> On the other hand, alopecia has also been linked to exposure to hazardous quantities of ambient zinc; typically, this occurs in conjunction with anemia and achromotrichias, sometimes known as "white monkey syndrome."

### Hormonal imbalances or changes

Hormone fluctuations have long been implicated with hair loss, especially in humans. The most well-known instance is probably male pattern baldness, also known as androgenetic alopecia, which results from androgens being converted to dihydrotestosterone in hair follicles that are genetically susceptible. Follicles shrink and the anagen phase is shortened by dihydrotestosterone.<sup>[15]</sup> Although the stump tailed macaque, with its distinctive bald forehead, has been used as a model to explore the effects of minoxidil on hair development, this disease does not appear to have a nonhuman primate equivalent.<sup>[16, 17]</sup> But in both people and animals, hair loss can also be related to other hormones. Here, we'll concentrate on the potential links between pregnancy, pituitary or adrenal tumors that cause hyperadrenocorticism, and hypothyroidism and hair loss.

### Immunologic and genetic factors

Autoimmune diseases of the hair. Between the ages of 20 and 40, both men and women are typically affected by the immunologic condition alopecia areata.<sup>[18]</sup> The disease-causing cells, mostly CD4-positive T lymphocytes and macrophages, target particular anagen hair follicle antigens. A comparable type of immune-mediated hair disorder has been observed in chimpanzees and rhesus monkeys. In one case that has been documented, a female rhesus monkey had no hair at all and had been hairless since birth.<sup>[19]</sup> The authors determined that the monkey had alopecia universalis based on a clinical examination, serologic tests, and histologic and immunocytochemical investigations of the skin demonstrating perifollicular lymphocytic infiltration. When determining the reason of a female chimpanzee's complete body hair loss, a similar result was made.<sup>[20]</sup>

### Bacterial infections

Numerous microorganisms can cause sores and hair loss on the skin. Adults in their middle years may develop a scarring alopecia known as folliculitis decalvans due to *Staphylococcus* bacteria. [21] It has also been demonstrated that alopecia and skin lesions can result from *Staphylococcus* infections in dogs, horses, and sheep. In addition, different subjects experienced different levels of hair loss in response to the creation of chronic salmonellosis in guinea pigs, depending on the *Salmonella* strain and the route of administration. No evidence currently available indicates that bacterial infections are the primary cause of hair loss in captive nonhuman primates, despite the fact that they should be ruled out as a potential cause of hair loss.

### Stress

There is mounting evidence to support the theory that stress and hair loss are related. Early uncontrolled research and, occasionally, anecdotal reports suggested that stressful life events could be the cause of alopecia areata in humans. An analysis of this corpus of data [22] highlighted the significant drawbacks of these strategies. More carefully monitored research afterwards did discover some proof that stress may play a part in initiating hair loss episodes. However, the review's authors advised against drawing firm conclusions about a clear link between stress and the development or aggravation of alopecia areata.

### Evaluation of Alopecia in Women

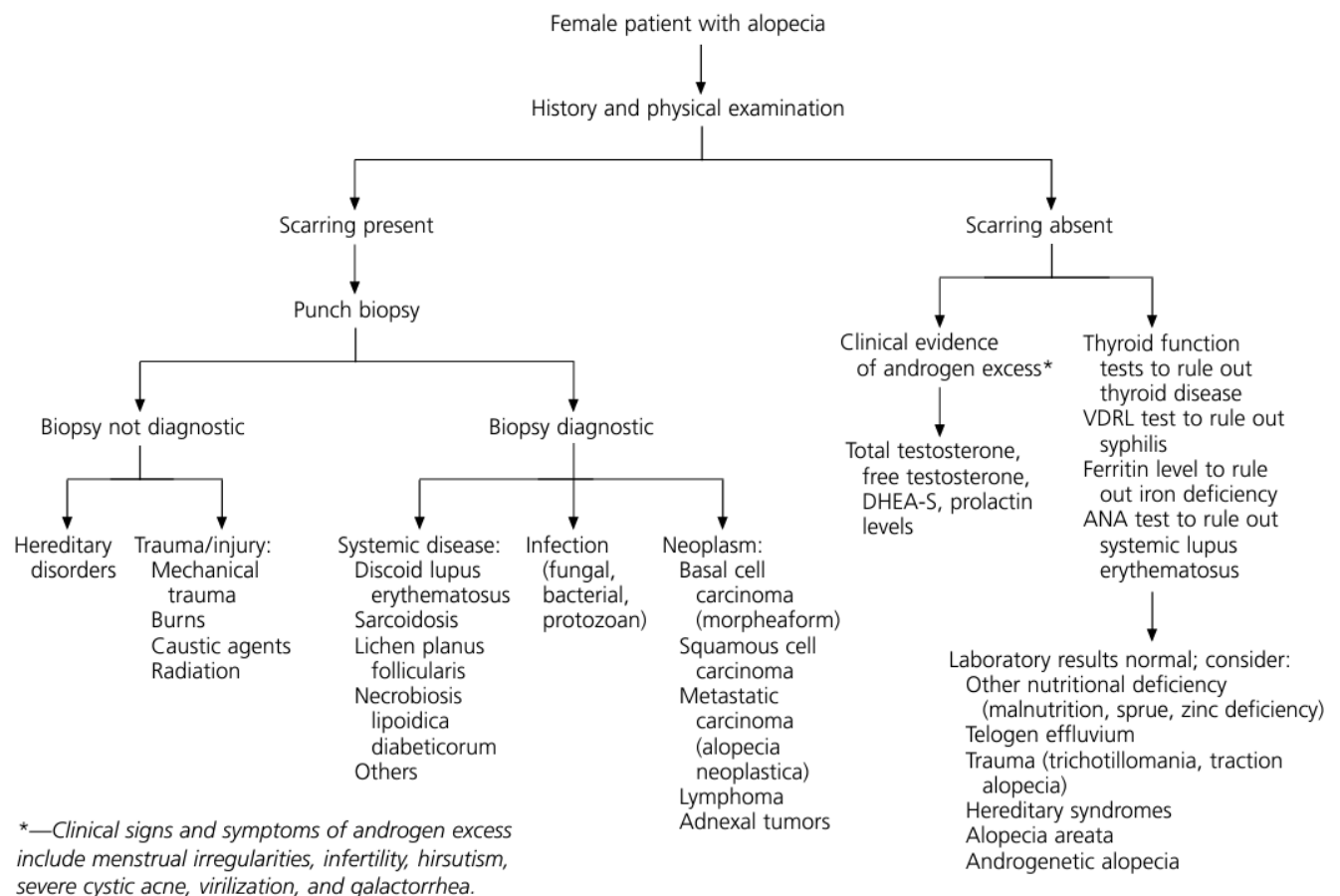


Fig. 2 Suggested approach to the evaluation of alopecia in women.



## Treatment of alopecia

### TOPICAL TREATMENTS

#### 1. Anthralin

Four case studies or reports including 69 individuals showed the use of the irritant anthralin to treat AA in paediatric patients.<sup>[23-26]</sup>

Relapse rates varied from 9.5% to 64%, with complete response rates falling between 32% and 33.3%. When paired with leflunomide, there was a case report of full regrowth. Nine to fifteen months was the average period to peak response. Anthralin results in skin discoloration and localized lymphadenopathy (LAD), both of which go away when the medication is stopped. Systemic adverse effects are infrequent, but other side effects include burning, leaking, itching, and bullous eruptions.<sup>[23-26]</sup>

#### 2. Diphenylcyclopropenone (DPCP)

Sensitization before the first treatment and increasing dosage concentrations are two aspects of treating the afflicted areas with DPCP. DPCP is a popular and effective therapy option for the pediatric population because of its practically painless application approach.<sup>[27-33]</sup>

The percentage of complete responses varied from 0% to 33.3%, which is comparable to the meta-analysis's (30.7%) findings.<sup>[34]</sup> With relapse rates ranging from 12.5% to 58.3%, relapses are frequent.<sup>[27,29,32]</sup> Iquimod may increase the effectiveness of DPCP, according to one case-control research.<sup>[12]</sup> Eczematous skin responses, pruritus, localized LAD, vesiculation, and infrequently, a secondary infection are examples of side effects.<sup>[27]</sup>

125% There were no systemic side effects observed, except from headaches.<sup>[34]</sup>

#### 3. Squaric acid dibutyl ester (SADBE)

The efficacy of SADBE was investigated in a study including 78 pediatric patients. Complete response rates ranged from 0% to 33.3%.<sup>[36-38]</sup>

In comparison to DPCP (30.7%), SADBE had somewhat better complete response rates (38.4%) in a meta-analysis covering both adult and pediatric patients.<sup>[35]</sup> Relapse rates range from 62.5 to 100%. Side effects include irritation, itching, contact dermatitis, and LAD.<sup>21</sup> In addition, one patient experienced epidermolysis *bullosa acquisita* while receiving AA therapy with SADBE; the illness resolved after the medicine was stopped.<sup>[37]</sup> No evidence exists to support the idea that topical application results in systemic absorption.

#### 4. Minoxidil

For adult AA, minoxidil's effectiveness is unclear, and the only case studies assessing its treatment in children are available. Most often, minoxidil is

utilized as an adjuvant treatment.<sup>[38, 39]</sup> Extensive hypertrichosis is one of the side effects of minoxidil.<sup>[40-44]</sup> The usual twice-daily dosage is usually safe, even though excessive topical application may cause systemic absorption (seen as palpitations, hypotension, etc.).<sup>[45]</sup>

#### 5. Topical Calcineurin Inhibitors

Tacrolimus and pimecrolimus, topical calcineurin inhibitors, are not useful in treating AA, according to the agreement of four investigations including seven juvenile AA patients. Of those that responded, about 29% did so only minimally the remaining 71% did not respond at all and frequently had their disease worsen.<sup>[46-47]</sup>

#### 6. Topical and Intralesional Corticosteroids

The use of topical corticosteroids, especially high-potency topical corticosteroids, as a first-line therapy option for children with patchy AA that is both safe and efficacious. In a randomized controlled trial comprising forty-one pediatric patients, high-potency topical corticosteroids proved to be more effective than low-potency topical corticosteroids. Additionally, they are frequently utilized as adjuvant therapy and are superior to topical tacrolimus and anthralin.<sup>[48,38,49,50]</sup> Children typically tolerate high-potency topical corticosteroids well. Folliculitis, telangiectasias, and skin atrophy are among the side effects. Although studies using intralesional corticosteroid therapy (triamcinolone) are beneficial, they are uncommon in youngsters because of the pain involved with injections.<sup>[44]</sup> The most frequent adverse effects, according to data on adult patients, are dyspigmentation, discomfort, and skin atrophy.

#### 7. Prostaglandins

Topical prostaglandins, such as latanoprost and bimatoprost, may enhance the regrowth of hair on the scalp and eyelashes in AA.<sup>[51,52,53,54,55]</sup> but an RCT looking at the growth of eyelashes in children with AA patients did not find statistically significant differences between bimatoprost and vehicle. 46 Although uveitis, eyelash curling, conjunctival hyperemia, and irreversible iris and eyelid darkening can be caused by prostaglandins, particularly latanoprost, these adverse effects have not been observed in alopecia areata patients.<sup>[51-56]</sup>

### 2. SYSTEMIC THERAPIES

#### 1. Corticosteroids

Systemic corticosteroid therapy is the most researched treatment option for AA in both adults and children. The studies included oral corticosteroid maintenance or tapered therapy<sup>[57,58,59-62]</sup> intramuscular corticosteroids, intravenous pulse-

dosed corticosteroids<sup>[63,64]</sup> and oral pulse-dosed corticosteroids<sup>[53,65]</sup>. About 45% (range 0-100%) of patients receiving intravenous or oral pulse-dosed corticosteroids showed a complete response, whereas 34% (range 0-55.5%) of patients receiving conventional oral corticosteroid regimens showed a complete response, despite doses and frequencies varying amongst each route of administration. It was discovered that a stronger response to pulse-dosed therapy was linked to shorter disease duration, younger age at disease onset, and multifocal disease (as compared to AT and AU).<sup>[66]</sup> Relapse rates were 16.7–100% for corticosteroids administered by pulse and 50–100% for those not.<sup>[59,66]</sup> Significant adverse effects were noted, and these were more severe and more common for non-pulse-dosed regimens. These side effects included weight gain, cataracts, infections, hypertension, Cushingoid characteristics, striae, and acne.<sup>[68, 69]</sup>

## 2. Hydroxychloroquine

A single case study with nine juvenile patients looked at the usage of hydroxychloroquine. Eleven percent of patients experienced a complete response and fifty-five percent saw a partial response when topical corticosteroids and/or minoxidil were used. Headache and stomach pain were mentioned as negative effects.<sup>[38]</sup>

## 3. Methotrexate

Eight studies examined the use of methotrexate for the treatment of AA in 42 pediatric patients either alone or in combination with oral, intravenous, or corticosteroids.<sup>[63-65, 70-74]</sup> With doses ranging from 2.5 mg to 25 mg per week, complete response was observed in 17.9% (range 0-50%; Table II) and partial response in 47.9% (range 0-100%) of cases.<sup>[64,65,69,70,]</sup> Although the relapse rate in children was much lower than that in adults (31.7% vs. 52%), a meta-analysis showed a larger complete response in adult AA patients compared to pediatric patients (44.7% vs. 11.6%). Hematologic abnormalities, increases in hepatic transaminases, and nausea were among the reported adverse effects.

## 4. Sulfasalazine and Mesalazine

The use of mesalazine and sulfasalazine for pediatric AA is limited in the available evidence. One case study of five juvenile patients showed a complete response to mesalazine, with or without concurrent oral or topical corticosteroids and minoxidil. In two investigations,<sup>[75]</sup> adolescents with AA who received oral sulfasalazine therapy showed a partial response to a starting dose of 1 g/week, which was increased to a final dose of 3 g/week.<sup>[76,77]</sup> Similar to the adverse

effect profile in adults, which includes gastrointestinal discomfort, rash, headache, and lab abnormalities, the side effects of sulfasalazine included dizziness, headache, and dyspepsia.<sup>[78]</sup>

## 5. Ustekinumab

Ustekinumab, a monoclonal antibody used to treat psoriasis that blocks interleukins 12 and 23<sup>[79]</sup> was shown to have improved the symptoms of AA in three people. This led to the treatment of juvenile children with AA and AT. Three patients in one case series had a full or partial response, whereas no reaction was observed in the other research.<sup>[80,81]</sup> Although ustekinumab is known to cause injection-site reactions, infections, nausea, and vomiting, none of these side effects were observed in these two investigations.

## 6. Janus Kinase (JAK) Inhibitors

JAK inhibitors may be useful in treating AA, according to mounting evidence, yet there is less data on this impact in pediatric patients. Hematologic malignancies, infections, diarrhoea, hypertension, thrombosis, gastrointestinal perforation, and incorrect lab results are among the side effects.<sup>[82]</sup>

## 7. Baricitinib

Although not yet for children, clinical trials have been started to assess the safety and effectiveness of baricitinib for the treatment of AA in adults.<sup>[83,84]</sup> There has only been one pediatric case documented. When given baricitinib 7 mg once day, a 17-year-old male patient with a history of resistant AA first displayed a partial response; however, when the dose was increased to 11 mg once daily, the patient demonstrated a complete response.<sup>[85]</sup> Relapses were not mentioned.

## 8. Ruxolitinib

There was just one juvenile patient in a case series of eight AA patients treated with ruxolitinib; the child had 10 mg of ruxolitinib twice a day for ten months, and his Severity of Alopecia Tool (SALT) score improved by 91% without any side effects.

## 7. Tofacitinib

Adult AA patients are undergoing clinical trials to assess tofacitinib's effectiveness in treating the condition.<sup>[86]</sup> Systemic tofacitinib was assessed in six case series and reports for the treatment of AA in 28 pediatric patients.<sup>[86-91]</sup> Eighty-two percent of these patients exhibited a complete or partial response, and the remaining patients all had AU. Tofacitinib response was also less common in persons with severe AT or AU that had been present for more than

ten years. Diarrhea, headaches, upper respiratory infections, increased hunger, weight gain, exhaustion, and momentary elevations in transaminases were among the side effects.<sup>[89]</sup>

### 8. Topical Tofacitinib & Ruxolitinib

Thirteen of the eighteen pediatric patients in three reports responded to topical treatment.<sup>[92-94]</sup> Instances of borderline leukopenia in a patient with a baseline low white blood cell count were among the side effects, along with application site discomfort.<sup>[93]</sup>

### 3. LASER AND PHOTOTHERAPY

#### • Laser Therapy

A 308 nanometer (nm) excimer laser was used twice a week to treat 17 patients, with a response rate of 58.8% . Mild scalp erythema and desquamation were among the side effects.<sup>[95-98]</sup>

#### • Phototherapy

26 pediatric AA patients receiving psoralen and UVA (PUVA) therapy were the subject of 6 reports.<sup>[99-101]</sup> In addition, five teenagers who received treatment with a towel drenched in psoralen before being exposed to the sun all showed signs of partial response.<sup>[102,103]</sup> Similar to adult outcomes, pediatric patients did not respond well to narrow-band UVB therapy .Similar to adult patients with AA, mild irritation, erythema, pruritus, and scaling were reported as phototherapy side effects<sup>[102-105]</sup>

### HERBAL MEDICINE USED FOR THE ALOPECIA

| S.No. | Plant Name                  | Family           | Part Uses        | Chemical Constituents   | Remark  |
|-------|-----------------------------|------------------|------------------|---|---|
| 1.    | Hibiscus rosa-sinensis Linn | Malvaceae        | Leaves & Flowers | Flavonoids; Anthocyanins and 5-diglucoside, Cyaniding-3, Quercetin-3-diglucoside, Cyaniding-3-sophoroside-5-glucoside, Quercetin-3,7-diglucoside <sup>[106]</sup> | Hibiscus rosa-sinensis leaf extract may help sustain hair development both in-vivo and in-vitro techniques. <sup>[107]</sup>  |
| 2.    | Cuscuta reflexa Roxb        | Convolvulaceae   | Stems            | Bergenin & Kaempferol, Cuscutin, Cuscutalin, - Sitosterol, Luteolin   | Cuscuta reflexa Roxb. hair growth activity. stem by periodically changing the telogen phase of the hair follicle into the anagen phase. <sup>[108]</sup>  |
| 3.    | Asiasari radix              | Aristolochiaceae | Roots & Rhizomes | methoxytoluenes & 3-benzodioxole derivatives, Methyl eugenol (18%), Safrole (18.4%)   | The Asiasari radix extract may promote hair development because of its regulatory actions on the expressions of both cell growth factor genes. <sup>[109]</sup>   |
| 4.    | Ocimum gratissimum Linn     | Lamiaceae        | Leaves           | Essential oil: Carvacrol, Eugenol, Nerol & Eugenol methyl ether <sup>[110]</sup>  | Ocimum gratissimum Linn. (Ocimum oil) leaf essential oil's ability to stimulate hair development in cyclophosphamide-induced hair loss and came to the conclusion that ocimum oil would be able to improve normal hair growth |



|    |                          |                    |        |  |   |
|----|--------------------------|--------------------|--------|--|---|
|    |                          |                    |        |  | and encourage follicular proliferation in cyclophosphamide-induced hair loss. <sup>[111]</sup>  |
| 5. | Aloe vera L. (Liliaceae) |                    | Leaves | Mucilage (Glucose, Galactose, Mannose, Galacturonic acid), Barbaloin (15-40%), Aloesodin, Aloesone, Alocutin A and B, Hydroxyaloin (3%) <sup>[112]</sup>                           | Gel made from Aloe vera L. or Aloe barbadensis is traditionally used to treat alopecia and promote hair growth. Aloenin is the primary component that stimulates hair growth without irritating the skin <sup>[113]</sup>   |
| 6. | Ginkgo biloba            | (Ginkgoaceae)      | Leaves | Bilobalide-A, Flavonols (24%), Lactones(6%): Diterpenoids, Ginkgolides A, B, C, Kaempferol, Quercetin, Isorhamnetin <sup>[114]</sup>   | Through its combined effects on cell proliferation and apoptosis in the hair follicle, ginkgo biloba leaf extract promotes hair renewal, suggesting potential use as a hair tonic. <sup>[115]</sup>   |
| 7. | Tridax procumbens L.     | (Compositae)       | Leaves | Flavonoid: Procumbenetin <sup>[116]</sup> , 1.7% Fumaric acid, sitosterol, Alkalodies, Tannin <sup>[117]</sup> , Quercetin, Glucoluteolin, Luteolin, Isoquercetin <sup>[118]</sup> | Tridax procumbens's ability to promote hair growth encourages the growth of hair. <sup>[119]</sup>  |
| 8. | Sophora flavescens (     | Leguminous plants) | Roots  | Alkaloids: Sophoranol, Oxymatrine, Losmatrine, Matrine, Sophocarpine, Bioflavones: Kuraridinol, Sophoraflavanone, Norkurarinone, Formoronetin & Fatty acids <sup>[120]</sup>       | the dried root extract Sophora flavescens has a remarkable ability to promote hair development. In dermal papilla cells, Sophora flavescens extract increased the mRNA levels of growth factors such KGF and IGF-1, indicating that the control of growth factors in dermal papilla cells may be the mechanism by which Sophora flavescens extract affects hair development. Furthermore, a strong inhibitory impact on type II 5 Y-reductase |

|     |  |                 |                  |  |   |
|-----|--|-----------------|------------------|--|---|
|     |  |                 |                  |  | activity was found in the <i>Sophora flavescens</i> extract <sup>[121]</sup>  |
| 9.  | <i>Citrullus colocynthis</i><br>Schrader | Cucurbitaceae   | Fruits           | Resinous Glycosides (Colocynthin and Colocynthitin), Phytosterol Glycoside, Citrullol, Albuminoides, Pectin, Cucurbitacins-Cucurbitacin E & I <sup>[122]</sup>   | <i>Citrullus colocynthis</i> encourages hair development. A minimum and maximum number of hair follicles in the anagenic phase were also discovered. 5-Reductase has been identified as a primary contributor to hair loss. If research is done using this bed to reveal the mechanism of action of herbal extracts, it could be fruitful.<br>[123].  |
| 10. | <i>Emblica officinalis</i>               | (Euphorbiaceae) | Fruits           | Alkaloids (Phyllantidine, Phyllantine), Vitamin C, Gallotannis (5%), Carbohydrates (14%), Pectin, Ellagic acid, Minerals, Amino acid (Alanine, Aspartic acid, Glutamic acid, Lysine, Proline), Phenolic acid, Gallic acid, Phyllemblic acid, Emblicol <sup>[124]</sup> | <i>Emblica officinalis</i> has increased its activities for hair development. It could be a powerful herbal substitute for minoxidil. <i>Emblica officinalis</i> (Euphorbiaceae), <i>Bacopa monnieri</i> (Scrophulariaceae), <i>Trigonella foenumgraecum</i> (Leguminosae), and <i>Murraya koenigii</i> (Rutaceae) also show outstanding outcomes of hair development in formula created by cloth pouch boiling method. |
| 11. | <i>Nordostachys jatamansi</i>            | (Valerianaceae) | Rhizomes & Roots | Volatile Essential Oil (0.5-2%): Jatmansone, Sesquiterpenoid (0.02-0.1%), Jatamol A & B Spirojatamol, Patchouli alcohol, Nardostachone, Jatamansic acid, Nardol <sup>[125]</sup>   | <i>Nordostachys jatamansi</i> possesses the ability to grow hair <sup>[126]</sup>   |
| 12. | <i>Eclipta alba</i><br>Hassak            | Asteraceae      | Whole plant      | Flavonoids & isoflavonoids: Desmethylwedolactone, Wedolactone, Triterpene glycosides & Saponins : Eclalbasaponins I-VI, -amyrin <sup>[127]</sup>   | Wedolactone and sitosterol are the active ingredients in hair growth. One factor in the treatment of androgenic alopecia is 5-reductase   |

|  |  |  |  |  |   |
|--|--|--|--|--|---|
|  |  |  |  |  | inhibition. In this study, 5-reductase inhibition by -sitosterol has been thoroughly reported. <sup>[128]</sup> |
|--|--|--|--|--|---|

Some Marketed Herbal Formulations for Hair Growth: <sup>[129]</sup>

Chirayu herbal oil- Amla, Bhringgraj, Brahmi

Sidha Shampoo - Tulsi, Neem

Regrow Massage Oil - Chamomile, Rosemary

Saini Herbal Hair Conditioner - Amla, Bhringgraj

Shyamla Shampoo - Amla, Heena

Nutrich Capsule - Bhringgraj, Godanti

Medhavi Oil - Amla, Bhringgraj, Brahmi

Keshmitra Tablets - Vacha, Jevanti

Keshamrit Oil - Amla, Bhringgraj

Kesh Rakshe Oil - Amla, Bhringgraj

Kesh Vardan Capsule - Ashwagandha, Shatawari

Hairich Capsule & Oil - H. roseus, E. alba, Osantum

Hairvit Oil - Brahmi, Bhringgraj, L.innerrmis

Hibril Oil - S.indicum, Bhringgraj, Brahmi

K-7 Taila Oil - Amla, Jatamansi

Hairbac Tablets - Amla, Bhringgraj, Guduchi

## CONCLUSION:

Alopecia, encompassing various types and causes, significantly impacts individuals both physically and psychologically. Despite numerous treatment options ranging from topical applications to systemic therapies, effective management remains challenging. Each treatment has its limitations and potential side effects, highlighting the need for ongoing research and innovation.

In addition to conventional treatments, herbal remedies have shown promise in managing hair loss. Ingredients like ginseng, saw palmetto, and essential oils such as rosemary and peppermint have been noted for their potential to stimulate hair growth and improve scalp health. These natural options are often favored for their minimal side effects and holistic benefits. Integrating these herbal treatments with conventional therapies could offer a more comprehensive approach to alopecia management, catering to diverse patient needs and preferences. Ultimately, a personalized treatment plan, considering both medical and herbal options, may provide the most effective solution for those suffering from alopecia.

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