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Brief Treatment Modalities of Alopecia Areata: Review Article

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ABSTRACT

Background: Hair loss on the scalp and/or body due to Alopecia Areata (AA) is a frequent autoimmune, inflammatory, nonscarring form of baldness. Hair loss can occur in a variety of patterns, from thinning in discrete areas to a complete loss of hair from follicle. All treatments of AA are considered off-label. While these therapies may help stimulate hair growth, they are not considered curative because they cannot alter the disease's normal progression. Age, general health, and the severity of hair loss all play a role in determining the best course of therapy.

Objective: Assessment of current treatment modalities of alopecia areata.

Methods: Treatment, and alopecia areata were all looked for in PubMed, Google scholar, and Science direct. References from relevant literature were also evaluated by the authors, but only the most recent or complete study from September 2007 to May 2021 was included. Due to the lack of sources for translation, documents in languages other than English have been ruled out. Papers that did not fall under the purview of major scientific investigations, such as unpublished manuscripts, oral presentations, conference abstracts, and dissertations, were omitted.

Conclusion: Because of their low risk of adverse effects and simple administration, topical corticosteroids of varying strengths are often the first line of defense in the treatment of AA in children. In more advanced cases of AA, systemic therapies are performed. Most of these drugs work by modulating or suppressing the immune system.

Keywords: Treatment protocol AA, Alopecia Areata, Hair loss.

INTRODUCTION

Common hair loss on the scalp and/or body due to an autoimmune, inflammatory, nonscarring condition called alopecia areata. Hair thinning can occur in a variety of patterns, from discrete bald spots to thinning at every hair follicle **(1).**

After androgenetic alopecia, alopecia areata is the most frequent autoimmune disorder associated with hair loss. Globally, 2% of the population will have AA at some point in their lives, with a 1%-2% lifetime risk **(2).**

AA is equally likely to affect men and women of any age and any racial or cultural origin. Prevalence is higher in kids than in grownups. In most cases, the beginning of AA occurs before the patient is 40, with a peak incidence in the twenties and thirties **(2).**

The precise mechanisms by which this illness develops remain unknown. However, a combination of genetic and environmental variables has been proposed to trigger an autoimmune reaction in the hair follicles, leading to AA (Figure 1)**(3).**

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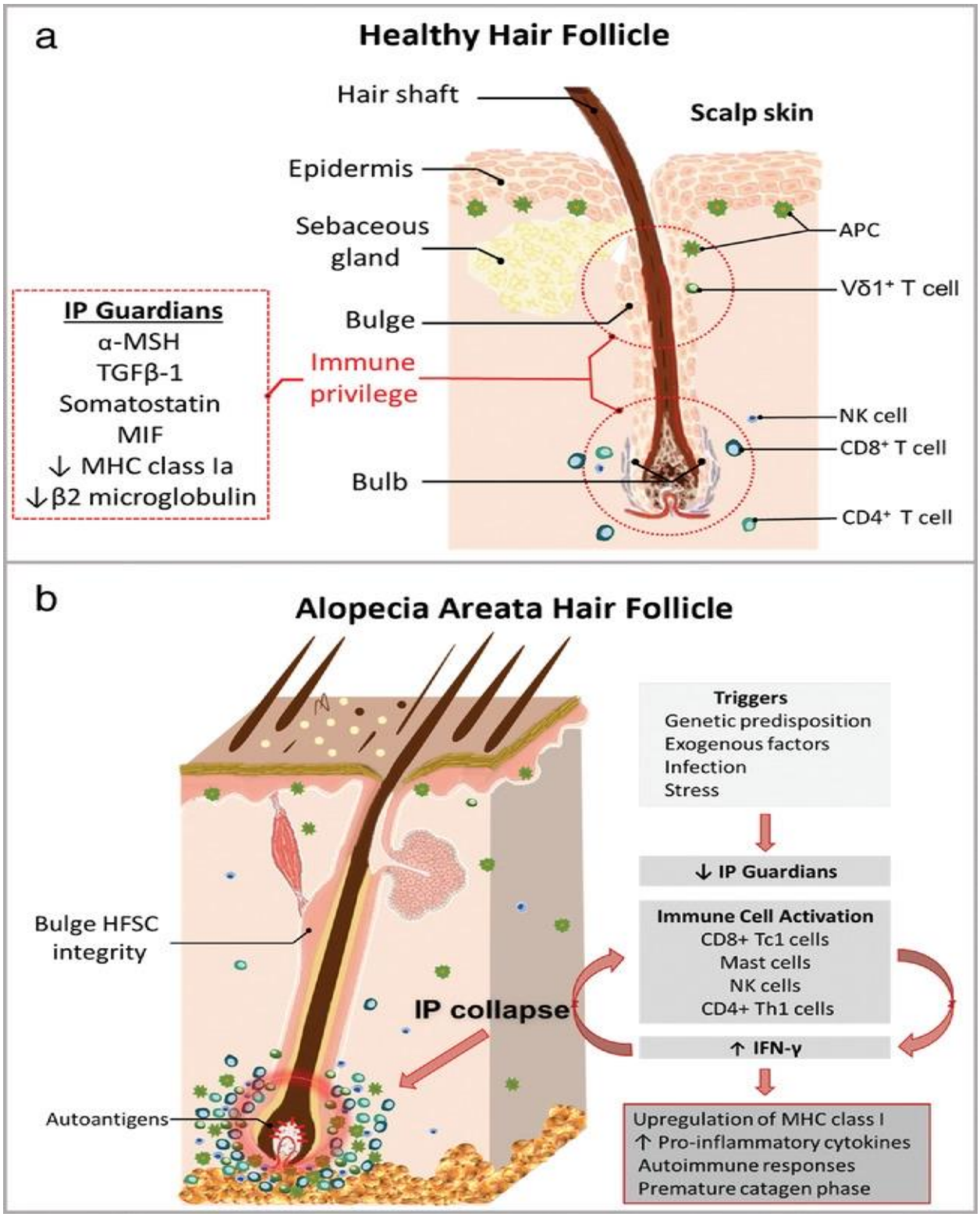

Figure (1): Pathophysiology of alopecia areata [(3)]

Treatment of Alopecia Areata:

All treatments of AA are considered off-label. These treatments can induce hair growth, but they aren't considered curative as they can't affect the natural course of the disease. Age, overall health, and the severity of hair loss all play a role in determining the best course of therapy (Figure 2)[(4)].

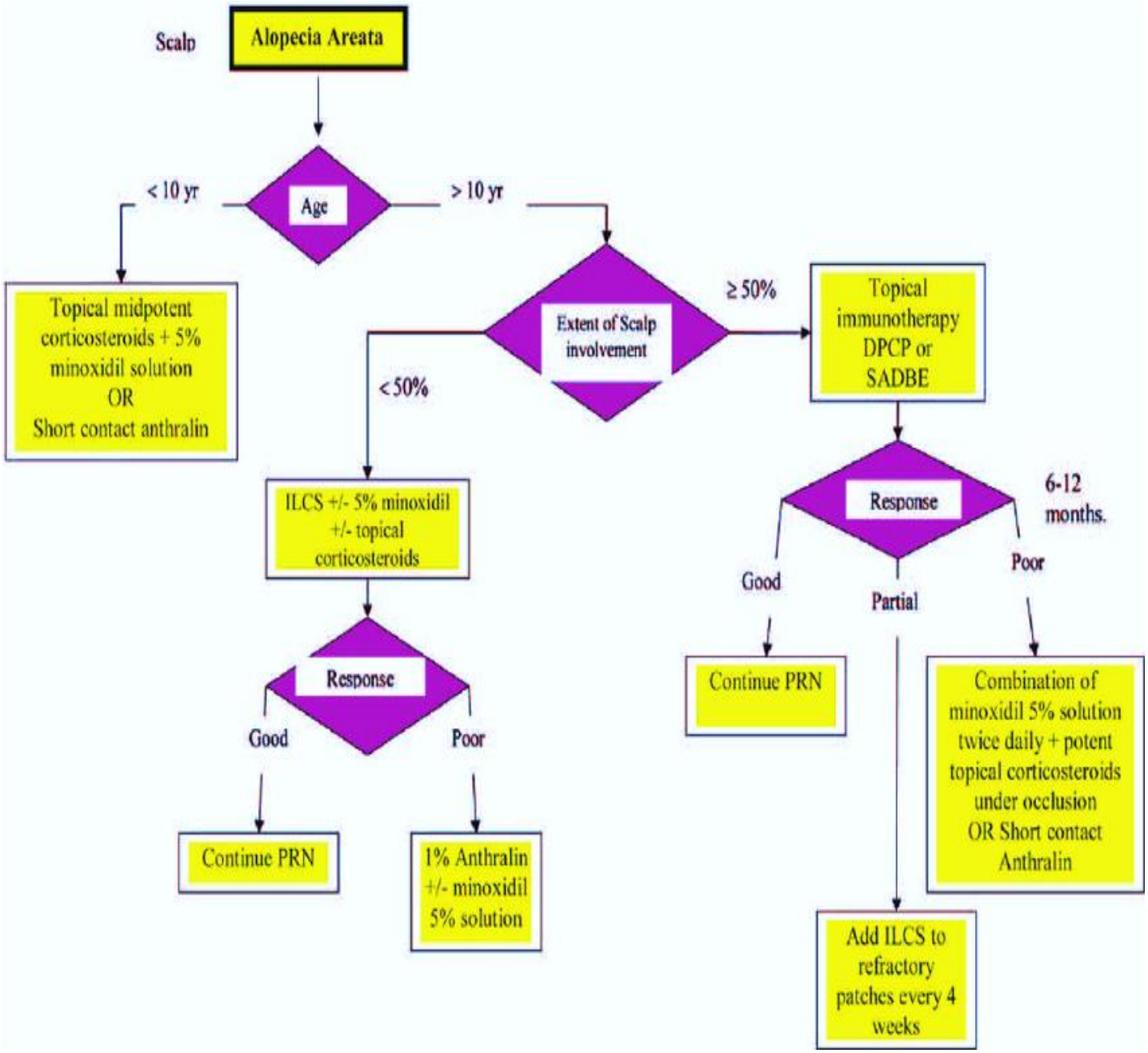

Figure (2): Treatment protocol of alopecia areata [4].

Topical treatments:

Because of their low risk of adverse effects and simple administration, topical corticosteroids of varying strengths are often the first line of defence in the treatment of AA in children. Intralesional corticosteroids (especially triamicinolone acetonide) are considered also first-line therapy in adult patients [5]. Atrophy of the skin, telangiectasia and local folliculitis are common side effects with long-term application[6].

Topical minoxidil is used also in AA as an adjunctive therapy as it can stop or reverse the inflammatory infiltrate and it stimulates also hypoxia-inducible factor-1-alpha, which is essential for vascular endothelial growth factor induction that increase perifollicular vascularization. In addition, minoxidil allows hair follicles to grow continuously and maintain the anagen phase by increasing prostaglandin E2 production and decreasing prostacyclin production. Allergic contact dermatitis including erythema, pruritis and burning sensation are reported side effects of topical minoxidil [7].

Other topical treatments include anthralin as a topical irritation therapy, contact sensitizers as squaric acid dibutylester, topical retinoids, topical calcineurin inhibitors, topical vitamin D analogue, prostaglandin analogs, platelet-rich plasma and cryotherapy [8].

Systemic treatments:

Systemic treatments are used only in progressive forms of AA. Most of them are immunosuppressants or immunomodulatory. They include systemic corticosteroids, oral immunosuppressive agents as methotrexate, biological agents as oral Janus kinase

inhibitors, oral vitamin D and zinc supplementation, phototherapy as excimer laser and narrowband ultraviolet B. Other treatment modalities include aromatherapy, hypnotherapy and psychosocial support [5].

Cosmetic treatments for patients with AA are also used. They include dermatography (tattooing) and hair pieces. Hair pieces are useful for patients with extensive disease as they enhance their self-esteem and social adjustment [9].

Emerging therapy (Simvastatin-Ezetimibe combination):

Statins are a class of lipid lowering drugs that are used in the treatment of hypercholesterolemia. There are various forms of statins as atorvastatin, fluvastatin and simvastatin. They are also present in combination preparations with other agents such as ezetimibe/simvastatin [10]. It was found that AA was successfully treated with simvastatin-ezetimibe combination [11].

They have a role in the onset and progression of autoimmune diseases because they can alter the imbalance of Th1, Th2 and Th17 cell populations versus Treg cell populations. Statins also prevent IFN-γ signaling and lymphocyte activation, which represent a more important target in recent AA [12].

Since statins have immunomodulatory effects and can also inhibit the JAK-STAT (Janus kinase-signal transducer and activator of transcription proteins) pathway, they have recently been considered as a potential therapy for AA. These immune effects are such as interfering with production of proinflammatory mediators and expression of adhesive molecules II, inhibition of MHC class II and antigen presentation, impairment the function of natural killer cell, stimulating the Treg cells expressing, inhibiting antigen associated lymphocyte function and intercellular interaction of adhesion molecule [13].

Myopathy, rhabdomyolysis, elevated hepatic enzymes and diabetes mellitus are considered serious complication after statins administration. However, they can improve with cessation of the drug. Liver function tests, fasting lipid profile and renal function tests should be evaluated before the start of therapy [14, 15].

Statin-associated muscular complaints are the hallmark of statin poisoning, and they often manifest as asymmetrical and bilateral pain and weakness in the big proximal muscles, most notably those of the lower limbs. Symptoms often appear within a month of starting treatment or increasing the dosage and might manifest either at rest or soon after activity. Myopathy and rhabdomyolysis brought on by statins cannot be reversed because there is currently no treatment for them. In most cases, therapy is just stopping the offending substance and providing emotional support [16].

Ezetimibe is the pioneering chemical of a new class of lipid-lowering drugs that work by blocking the intestinal absorption of cholesterol. When combined with simvastatin, ezetimibe may be responsible for immunomodulatory and synergistic anti-inflammatory actions. Levels of C-reactive protein were not affected by ezetimibe alone, but when coupled with simvastatin, a substantial decrease was seen [17].

Patients with AA who were given simply ezetimibe did not have any substantial improvement in their hair regrowth. When simvastatin was introduced, though, hair began to grow [18].

CONCLUSION

Because of their low risk of adverse effects and simple administration, topical corticosteroids of varying strengths are often the first line of defense in the treatment of AA in children. In more advanced cases of AA, systemic therapies are performed. Most of these drugs work by modulating or suppressing the immune system.

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