A clinician's guide to pediatric and adolescent alopecia areata treatments

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Background: Available treatment options for pediatric alopecia areata are limited and may even exacerbate the physical and psychosocial burden.

Objective: This article aimed to provide a treatment classification system and counseling recommendations.

Recommendations: Topical corticosteroids are appropriate for initial treatment due to their ease of use and safety. Topical minoxidil and dithranol are often used as adjunctive treatments based on mixed data. Intralesional corticosteroids are effective for localized disease, but injections may be challenging in children. Contact immunotherapy, systemic corticosteroids, and immunosuppressive agents such as topical and oral Janus kinase (JAK) inhibitors, dupilumab, and low-dose methotrexate demonstrate varying effectiveness, adverse effects, and cost. Although ineffective in adults, topical JAK inhibitors may sometimes be effective in children. Conditions that impact the quality of life in pediatric patients with alopecia areata such as depression and social isolation should be addressed.

Limitations: Some data originate from case reports and series, which may limit validity and generalizability.

Conclusions: Shared decision-making and patient-centered communication are essential in managing pediatric alopecia areata. Treatment should be based on disease severity, individual patient goals, and adverse effect profiles. As topical and systemic JAK inhibitors, dupilumab, and related compounds advance the treatment of alopecia areata in children, clinicians should be alert to cost, potential side effects, and the enduring role of older topical therapies. (JAAD Reviews 2024;2:57-66.)

Key words: adverse effects; biologic therapy; contact immunotherapy; immunosuppressive agents; intralesional corticosteroids; Janus kinase (JAK) inhibitors; oral corticosteroids; oral minoxidil; pediatric alopecia areata; pulse-dosed corticosteroids; shared decision-making model; side effects; systemic corticosteroids; topical calcineurin inhibitors; topical corticosteroids; topical dithranol; topical minoxidil; treatment classification.

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INTRODUCTION, PATHOPHYSIOLOGY, AND EPIDEMIOLOGY

Alopecia areata (AA) is a chronic disease characterized by a nonscarring T cell-mediated attack on hair follicles. It affects approximately 2% of the population, with a predilection for patients aged <40 years.[1] Our understanding of AA has evolved through studies of genetics, immune cell types, antigenic targets, and environmental factors. This clinician's guide serves as an overview of disease pathogenesis and treatment guidelines to address concerns about disease severity and potential treatment risks in pediatric patients. Alopecia can have a significant impact on quality of life. The visible hair loss paired with the chronic and unpredictable nature of the disease may be a source of emotional and psychological distress for both children and families, thereby exacerbating the overall burden of disease.

CAPSULE SUMMARY

- The lack of a definitive treatment classification system for pediatric alopecia areata and potential adverse effects of current treatments may contribute to a greater burden of disease.
- This clinical review discusses our treatment approach for pediatric alopecia areata and the need for a multidisciplinary approach to ensure optimal treatment.

CLINICAL FEATURES

Pediatric hair loss presents a wide range of differential diagnoses (Fig 1).[2] AA presents as smooth, round, sharply demarcated patches of hair loss (Fig 2). Exclamation points or tapering hairs, which are thinner at the base of the scalp, may be seen at the periphery of the patch. Dermatoscopy findings indicating active disease include "yellow dots," which represent keratin within dilated follicular infundibula. Similarly, "black dots" indicate dystrophic hairs. Less common variants exist, such as AA ophiasis (band-like hair loss across the temporal and occipital scalp), sisaipho (hair loss sparing the temporal and occipital scalp), totalis (complete hair loss on scalp and face), and universalis (complete hair loss on scalp and whole body).

COUNSELING

Pediatric patients with AA have higher rates of separation anxiety, generalized anxiety disorder, social phobia, and major depressive disorder than the general pediatric population.[3] AA lowers patients' sense of self-esteem, which may manifest negatively in school, in social life, and at home. Furthermore, higher severity of disease in pediatric patients with AA is correlated with lower quality of life among their parents.[4] Clinicians may recommend support groups, which can help patients and families replace feelings of isolation with a sense of community to cope with challenges. The benefits of hair prosthesis (custom-made wigs) on quality-of-life measures have been noted in adult studies and may translate to the pediatric population.[5] Referral to pediatric specialists, such as clinical psychologists or educational psychologists, and support groups such as those associated with the National Alopecia Areata Foundation may help guide disease management.

Should all pediatric patients with AA receive treatment? Given the potential for spontaneous regrowth, some patients may opt for watchful waiting; however, treatment is generally recommended, given the psychosocial impact and the potential for worsening disease. Although most remain off-label, multiple treatment options are now available for pediatric AA.[6,7] In determining the optimal treatment course, clinicians should consider the evidence and adverse effect profile of each treatment.[8-10] Shared decision-making is the recommended model, because it helps incorporate patients' goals for care, perception of self-image, coping strategies, and social support.[11]

TREATMENT

Treatment options studied in pediatric patients are discussed below. Further details on recommended dosage, duration of therapy, and potential adverse effects may be found in Table I[11-45] for all treatments listed below.

Topical corticosteroids

Topical corticosteroids remain the initial choice of treatment for pediatric patients due to their ease of use, accessibility, and safety, especially among patients aged <12 years, who may be less tolerant of intralesional delivery (Table I). A 6-month study of patients with refractory alopecia totalis (AT) and alopecia universalis showed hair regrowth within 14 weeks after using clobetasol propionate 0.05% under occlusion. Only 3 of the 28 patients did not see results, and 17.8% of patients saw long-term benefit.[12] Another randomized controlled trial (RCT) among 41 children with AA showed that 0.05% clobetasol propionate resulted in a significantly greater decrease in hair loss surface area than 1% hydrocortisone.[13] Topical corticosteroids may be

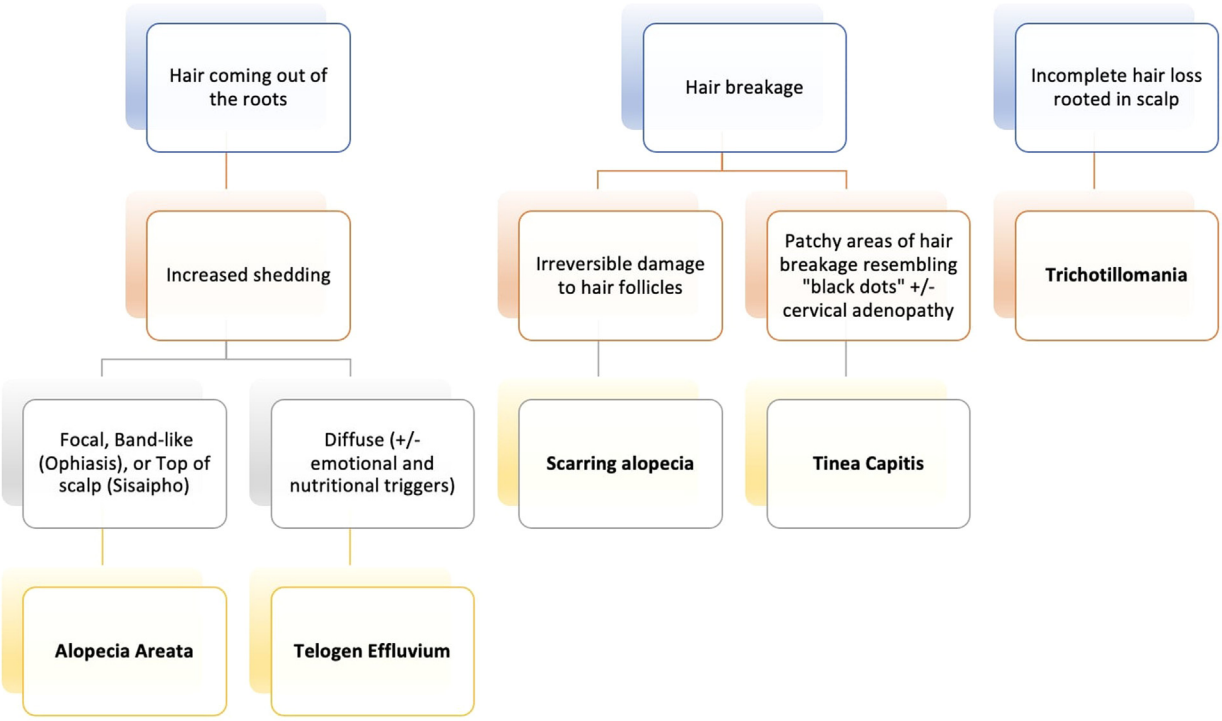

Fig 1. Pediatric hair loss: presenting a diagnostic algorithm.[2]

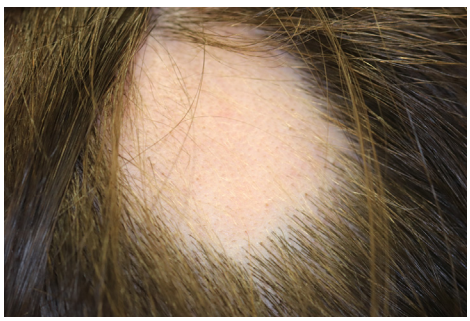

Fig 2. Pediatric alopecia areata, seen as a circular region of nonscarring hair loss on the scalp.

better suited to early stage disease (Table II), as one study found that children aged <10 years with AA duration of <1 year responded more favorably to 0.2% fluocinolone acetonide cream.[14] Another formulation, 0.25% desoximetasone cream, was found to result in a higher rate of complete regrowth than placebo in 54 patients with patchy AA, although the difference between the groups was not statistically significant.[15] Clinicians must be mindful of continuous use, especially with high-potency preparations, due to the risk of skin atrophy and dyspigmentation (Table I).

Topical jak inhibitors

Topical ruxolitinib 1.5% cream showed no significant results in adults with AA per a vehicle-controlled study.[16] Topical tofacitinib 2% showed better results in a case series of 11 pediatric patients with AA, totalis, or universalis, with 72% reporting partial regrowth.[17]

Topical minoxidil

Topical minoxidil is an adjunctive treatment with promising results. In a double-blind placebo-controlled trial, 63.6% of patients (adults and children) who applied topical 3% minoxidil experienced hair regrowth. Using minoxidil under occlusion was shown to have a greater effect in leading to regrowth in extensive AA.[18] In another comparative study (nonrandomized and uncontrolled), patients (n = 66) with >75% hair loss receiving 5% minoxidil experienced greater rates of terminal hair growth (81%) than those receiving the 1% concentration (38%).[19] One limitation to both aforementioned studies is the absence of subgroup analysis. In one case, using minoxidil over a large surface area led to hypotension and cardiovascular side effects.[20] More common and less severe side effects were limited to the skin. An optimal dose of topical minoxidil has yet to be established in children.

Oral JAK inhibitors

New developments and increased availability of small molecule inhibitors have led to the use of these agents in pediatric patients. Baricitinib, an Food and

Table I. Available pharmacologic treatments, dosage, and response per clinical data for pediatric alopecia areata.

Treatment	Dosage	Duration of therapy	Main outcomes	Adverse effects	Highest level of evidence
Topical corticosteroids[12-15]	0.05% clobetasol propionate	Up to 14 wk	High rates of hair regrowth; moderate rates of long-term resolution	Reversible skin atrophy, itching, burning, acneiform eruptions, striae, telangiectasia	Randomized controlled trial
Topical minoxidil[16-18]	2% or 5% minoxidil	3-6 mo	High rates of hair regrowth when used as an adjunctive treatment with other topicals	Hypertrichosis, irritant contact dermatitis, atopic dermatitis flare	Randomized controlled trial
Intralesional corticosteroids[19]	0.05-0.1 mL per site, triamcinolone acetonide (10 mg/mL)	Every 4 wk for 3 mo	High rates of initial hair regrowth (>75%) with potential for recurrence	Pain, dyspigmentation, skin atrophy	N/A
Topical contact immunotherapy—(DPCP)[23-25]	Increasing concentrations beginning with 0.0001%	6-12 mo	Limited efficacy (sustained regrowth in 10%-20% patients with AA)	Dermatitis, edema, urticaria, and lymphadenopathy	Case series
Topical contact immunotherapy—SADBE[23-25]	Increasing concentrations beginning with 0.0001%	6-12 mo	Limited efficacy (sustained regrowth in up to 10% patients with AA)	Irritant contact dermatitis	Case series
Systemic corticosteroids[26-28]	0.5 mg to 0.8 mg/kg/d	2-3 mo	Moderate rates of total regrowth (30%-50%), including severe AA	Weight gain, leukopenia, pneumonia, skin atrophy, acne, mood changes, psychosis, and GI symptoms	Randomized controlled trial
Pulsed systemic corticosteroid therapy[26-28]	5 mg/kg/mo) (300 mg/mo)	3 mo	Moderate rates of total regrowth (50%-60%), including severe AA	GI distress and headache	Randomized controlled trial
Methotrexate[29-31]	2.5-5 mg weekly, increased to up to 7.5-15 mg weekly	4-12 mo	Less effective for total resolution in pediatric patients vs adults (12% vs 45%) but relapse less frequently noted (32% vs 52%)	GI symptoms, elevated hepatic transaminases, and lymphopenia	Meta-analysis of observational studies
JAK Inhibitors and Biologics[32-38]	Varying dosages of Tofacitinib, Baricitinib, Ruxolitinib, Ustekinumab Dupilumab	3-12 mo	>80% rate of partial or complete resolution in recalcitrant cases	GI distress, headache, weight gain, fatigue, transaminase elevations, and infections	Randomized controlled trials and case series
Topical dithranol[25,39,40]	1% ointment	1-3 y	High rates of total or partial hair regrowth (>70%) when used as sole or adjunctive agent	Skin irritation	Bilateral half-head study
Topical calcineurin inhibitors[41-45]	1% pimecrolimus, 0.1% tacrolimus	6 mo	Not recommended due to multiple reports of unsuccessful use	Localized skin burning sensation	Randomized controlled trial
Energy-Based Devices (308 nm excimer laser)	Minimal intensity 50 MJ/cm^2 (ramp-up thereafter)	Twice weekly for 3 mo	High rates of total or partial regrowth (63%) but recurrence noted (44%)	Mild darkening, erythema, and peeling of skin	Cohort comprised study

AA, Alopecia areata; *cm*, centimeter; *DPCP*, diphenylcyclopropenone; *FDA*, Food and Drug Administration; *GI*, gastrointestinal; *J*, joules; *JAK*, Janus Kinase; *kg*, kilogram; *mg*, milligram; *mL*, milliliter; *mo*, month; *N/A*, not applicable; *RCT*, randomized control trial; *SADBE*, squaric acid dibutyl ester.

Table II. Suggested treatment classification system for pediatric alopecia areata

Category	Definition	Treatment considerations	Patient-centered approach
Mild-to-moderate disease	Limited, patchy alopecia areata	*Initial considerations*: Topical steroids ± topical minoxidil if age <10 y; Intralesional steroids ± topical minoxidil if age >10 y *Additional considerations*: Contact immunotherapy (DPCP and SADBE) *Considerations for refractory disease*: Topical dithranol	Shared Decision-Making Model Recommendations for involvement in AA-related support groups Scalp Prosthesis Referral to pediatric specialists (clinical or educational psychologist)
Moderate disease	Rapidly progressing, extensive alopecia	*Initial considerations:* Topical steroids ± topical minoxidil if age <10 y; Intralesional steroids ± topical minoxidil if age >10 y Topical JAK inhibitors *Additional considerations:* Dupilumab Pulsed systemic corticosteroids Cryotherapy Contact immunotherapy (DPCP and SADBE)	
Severe disease	Chronic, refractory, extensive alopecia areata, or alopecia totalis, or alopecia universalis	*Initial considerations*: Topical steroids ± topical minoxidil if age < 10 y; Intralesional steroids ± topical minoxidil if age > 10 y with adjunctive dithranol *Additional considerations:* Topical JAK inhibitors Dupilumab Oral JAK inhibitors Pulsed systemic corticosteroids Cryotherapy Methotrexate *Considerations for refractory disease:* 308 nm excimer laser Contact immunotherapy (DPCP, SADBE) *Additional:* Consider referral to expert center for evaluation and potential trials of experimental options	

AA, Alopecia areata; *DPCP*, diphenylcyclopropenone; *JAK*, Janus Kinase; *nm*, nanometer; *SADBE*, squaric acid dibutyl ester.

Drug Administration-approved JAK inhibitor for severe AA in adults is currently undergoing clinical trials in pediatric patients. Ritlecitinib recently received Food and Drug Administration approval in patients aged >12 years after RCT data showed that it was well-tolerated and achieved a Severity of Alopecia Tool score of 20 or less with optimal dosing (200/50 mg daily) in up to 28% of patients with moderate-to-severe AA in the trial's pediatric subgroup (age, 12-17 years) (comparable with 29% in the adult subgroup).[21] To date, one pediatric case refractory to 12 mg per day of oral prednisone showed complete response with baricitinib, 7 mg per day uptitrated to 11 mg per day over the course of 6 months, with tapering of corticosteroids to 3 mg per day, maintained at follow-up 1 year later.[22] Oral tofacitinib has been studied in 70 pediatric patients across several case series, indicating >80% rate of partial or complete resolution as either adjuvant or monotherapy.[23-30]

Biologics

The interleukin (IL) 12/23 blocker ustekinumab has been reported in 2 studies with pediatric patients, with mixed data from no response to complete response among 7 total patients.[31] Other studies have investigated the use of dupilumab in pediatric populations. In a case series of 16 patients with severe AA refractory to initial topical and oral treatment options, switching to the IL-4/13 blocker dupilumab lowered or maintained Severity of Alopecia Tool scores in all patients. Notably, all patients with atopic dermatitis and asthma demonstrated clinical improvement in their respective comorbid diseases.[32]

Intralesional corticosteroids

In adults, the use of intralesional corticosteroids (ILCs) has been well-documented. Triamcinolone acetonide at concentrations of 2.5 to 10 mg/mL may be injected at 0.05 to 0.1 mL per site on the scalp and at 2.5 mg/mL for eyebrows and other facial areas (0.5 mL per eyebrow).[33] Successful use with relatively few adverse effects has led to the use of ILCs as the preferred agents in the treatment of adult AA.[33]

Despite promising data, use of ILCs in children is limited due to the barriers of pain and needle phobia. Future studies that attempt to assess this treatment modality in pediatric AA cohorts may consider various strategies to limit pain. These include using smaller gauge needles, topical anesthetic creams, vibrating devices and peripheral ice packs to provide desensitization or distraction, and needle-free intradermal drug delivery through jet injection. Studies in adults with varying levels of disease severity indicate partial regrowth in up to 75% of patients receiving ILC via jet injection.[34,35] These studies suggest the efficacy of ILC versus saline; however, the comparative efficacy and tolerability of ILC via jet injection versus standard needle has yet to be assessed.

Contact immunotherapy

In cases where topical or ILC use may fail, topical immunotherapy may be considered. Efficacy data in adult cohorts are mixed (10%-90%) with similar distribution in pediatric populations but higher relapse rates, especially in patients with more severe disease.[36]

The most commonly used topical immunotherapy agents include the contact sensitizers diphenylcyclopropenone (DPCP) and squalene acid dibutyl ester (SADBE).[37,38] Resolution rates in adults have been reported to be 50% to 60% in recent studies. However, studies in pediatric patients are few and of notably lower efficacy. A retrospective study assessing SADBE in 33 children with AA or AT found sustained improvement in 9% (n = 33) because of high relapse rates after the treatment course. A similar study with 108 patients using DPCP showed low rates of completed regrowth (13%) and partial regrowth (25%). Adverse effects were noted in >50% of patients taking DPCP.[39] With limited efficacy, DPCP and SADBE may be best limited to refractory cases in pediatric groups, given the potential side effects.

Systemic corticosteroids (pulsed and nonpulsed)

Systemic corticosteroid therapy has been the most widely studied treatment option for pediatric alopecia to date. Different routes of systemic administration have been studied, including oral pulse-dosed, intravenously (IV) pulse-dosed, intramuscular, and oral with adjuvant methotrexate or cyclosporine. Attempts to avoid the well-established profile of adverse effects associated with long-term systemic corticosteroid therapy include pulse-dosed administration. A recent review noted that complete resolution of AA was achieved in 45% of patients receiving IV pulse-dosed steroids and in 34% of patients receiving oral corticosteroids among an aggregate cohort of 272 patients from various case series and controlled studies.[25] Treatment was generally less effective in patients who had a longer duration of disease and AT or alopecia universalis. High rates of relapse were noted in several studies. In one study where oral corticosteroids were pulsed with weight-based dosing for 3 months, 60% of patients (n = 12) experienced total resolution of AA or AT. Systemic treatment (nonpulse dosed) was associated with

more side effects and a higher rate of treatment discontinuation due to side effects (Table I).[40,41]

Other systemic immunomodulatory agents

Oral immunosuppressive agents such as methotrexate, sulfasalazine, and hydroxychloroquine have been studied in smaller populations. In a case series of pediatric patients prescribed hydroxychloroquine, 1 of 11 patients experienced total resolution, and 5 of 11 patients experienced partial resolution.[42] In a study where pediatric patients were treated with 3 g/mL of sulfasalazine, all 10 patients demonstrated partial improvement.[43] The use of methotrexate, both as a single and adjunctive agent, has been more widely studied in the pediatric population. A recent meta-analysis indicated that methotrexate was slightly less effective in pediatric patients than adults (12% vs 45%) but that relapse was less frequently noted in pediatric patients (32% vs 52%).[44]

Topical dithranol

Anthralin (dithranol) is a synthetic compound modeled after chrysarobin, found naturally in goa powder. In one case series, 37 patients aged <17 years on topical anthralin were monitored for 2.5 years. Most patients were receiving concurrent treatment with topical minoxidil, corticosteroids, or ILCs. Among 37 total patients, 12 experienced total regrowth. The remainder of patients experienced >50% regrowth with anthralin. Four patients stopped anthralin due to reported skin irritation.[45] In another case, a 14-year-old with recalcitrant ophiasis-phase alopecia, who failed ILCs, minoxidil, and oral betamethasone pulses, was successfully treated with a combination of leflunomide and anthralin.[46] In a bilateral half-head study of 30 pediatric patients with chronic, severe treatment-refractory alopecia, 16 achieved complete response and 5 achieved partial response after 2 years of anthralin 1% ointment. No serious adverse events were observed.[47] Anthralin ointment may be a helpful standalone or adjunctive agent for treatment-refractory alopecia with minimally reported adverse effects.

Topical calcineurin inhibitors

Topical calcineurin inhibitors have been investigated in RCTs and smaller-scale studies for AA. In 1 half-head study with 12 patients with untreated mild AA, tacrolimus led to inferior outcomes than a comparative regimen of clobetasol.[48] In another case series with 11 patients presenting with mild-to-moderate AA, tacrolimus ointment yielded no significant terminal hair growth after 6 months of treatment.[49] Another study of 15 patients with mild AA, topical pimecrolimus 1% did not lead to any significant improvement compared with placebo.[50] In a trial featuring 17 patients with mild-to-moderate AA, tacrolimus did not show significant hair growth in any patients.[51] There may be insufficient penetration of these topical formulations, and patients with long-standing disease may lack the T cell infiltrates commonly seen in active, early stage disease.[48-51]

Oral minoxidil

Oral minoxidil has emerged as an agent of interest in pediatric patients. One case series indicated a significant increase in hair regrowth among 45 patients, with an average improvement rate of 55% in hair density and 62% in hair diameter.[52] Another case series noted that tofacitinib + oral minoxidil yielded significant hair regrowth in adult patients with severe AA, highlighting the potential for this combination.[53] Further investigation is needed to determine the safety profile and overall efficacy of oral minoxidil in this context.

Phototherapy

Topical psoralen and UV-A therapy have been assessed in small pediatric cohorts with mixed results. Although some cases have resulted in markedly improved hair growth, these results have not been shown to be sustained long term.[54,55] Furthermore, the mechanism for improvement is poorly understood, most likely owing to immunomodulatory effects.[56] Multiple sessions were required in patients who demonstrated improvement.[57] Commonly reported adverse effects included erythema, irritation, and scaling. UV-B phototherapy failed to demonstrate positive results in 1 pediatric cohort.[58] The data for phototherapy are far from promising but suggest some scope for consideration as an adjunctive treatment.

Cryotherapy

Superficial hypothermic liquid nitrogen cryotherapy has been accepted as a considerable treatment for AA due to its minimal costs and adverse effects. In mixed adult-pediatric cohorts across 8 studies, partial or complete regrowth has been noted in 60% of patients who underwent liquid nitrogen cryotherapy.[59] Compared with ILC injections, treatment efficacy was similar, although lower relapse rates were noted in cryotherapy groups. One study highlighted outcomes specifically in a pediatric patient cohort.[60] This retrospective chart review at a department of dermatology in a Korean institution found >60% regrowth in 21% and partial improvement in 37% of a group of 24 pediatric patients aged <10 years, and >60% regrowth in 16% and partial

improvement in 37% of a group of 40 patients aged 10 to 20 years. Less than 3% of patients in the overall cohort experienced minor adverse effects, including pain, inflammation, and pruritus. These findings suggest that liquid nitrogen cryotherapy may be an effective agent and should be further studied in larger sample sizes with subgroup analysis for the level of disease severity.

Energy-based devices

One uncontrolled clinical trial investigated the role of 308 nm excimer laser therapy in pediatric AA.[61] Although improvement was noted in 7 of 11 cases of alopecia universalis or AA recalcitrant to topical therapy, disease recurrence (4/7) was an issue. This study has been referenced by systematic reviews and meta-analyses that discuss the efficacy of low-level light therapy on mixed adult and pediatric cohorts.[62,63] The 308-nm excimer laser may be used as an adjunctive treatment, given that it was well-tolerated but should be investigated in larger trials for more conclusive efficacy data. Additionally, other lasers, such as the pulsed-infrared diode laser, should be investigated because of promising initial data in mixed adult and pediatric cohorts.

LIMITATIONS

For several treatment modalities, the paucity of data makes it more challenging to make recommendations about the efficacy or safety of the respective treatments. For certain treatment options, data were collected from case reports and series, which may limit validity and generalizability. However, these suggested that treatment guidelines indicate the need for more comparative studies to better assess the efficacy and safety of current treatment options.

Summary

Due to the heterogeneous presentation of the disease and its tendency to be refractory to initial treatment options, it is essential that clinicians and families of patients be informed of available treatments, their efficacy, and safety profiles. Topical treatment may be a reasonable initial approach for localized disease. Although data are sparse for combination therapy, when possible, the use of more than 1 topical agent, including minoxidil and adjuvant dithranol, may lead to more effective outcomes compared with a single agent.

Topical therapy may not always be a viable—or permanent—solution. ILC treatment is often poorly tolerated by pediatric patients. Systemic corticosteroids, although effective, are commonly associated with the greatest risk of side effects among all treatment options. Clinical trials and anecdotal evidence for biological treatments and immunosuppressants such as JAK inhibitors provide promising efficacy data, but the risk of adverse effects and cost of treatment are potential barriers. It is evident that the challenge of pediatric alopecia is the lack of a "one-size-fits-all" approach. Treatment should be tailored to each patient through shared decision-making when possible. Further studies should assess the use of these treatments and others, including laser treatment, aromatherapy, rosemary oil, and other topicals including cellium, for which limited data currently exist in pediatric cohorts.

Conflicts of interest

Dr Lio reports research grants/funding from AbbVie, AOBiome, Eczema Foundation, National Eczema Association; is on the speaker's bureau for AbbVie, Eli Lilly, Galderma, Hyphens Pharma, Incyte, La Roche-Posay/L'Oreal, MyOR Diagnostics, ParentMD, Pfizer, Pierre-Fabre Dermatologie, Regeneron/Sanofi Genzyme; reports consulting/advisory boards for AbbVie, Almirall, Amyris, Arbonne, Arcutis, ASLAN, Bristol-Myers Squibb, Burt's Bees, Castle Biosciences, Codex Labs, Concerto Biosci, Dermavant, Dermira, DermVeda, Eli Lilly, Galderma, IntraDerm, Janssen, Johnson & Johnson, LEO Pharma, Lipidor, L'Oreal, Menlo Therapeutics, Merck, Micreos, MyOR Diagnostics, Regeneron/Sanofi Genzyme, Skinfix, Sonica, Theraplex, UCB, Unilever, Verrica, Yobee Care; stock options with Micreos and Yobee Care. In addition, Dr. Lio has a patent pending for a Theraplex product and is a Board member and Scientific Advisory Committee Member Emeritus of the National Eczema Association. Dr Ungar is an employee of Mount Sinai and has received research funds (grants paid to the institution) from Incyte, Rapt Therapeutics, and Pfizer. He is also a consultant for Arcutis Biotherapeutics, Castle Biosciences, Fresenius Kabi, Pfizer, and Sanofi. Dr Guttman-Yassky is an employee of Mount Sinai and has received research funds (grants paid to the institution) from AbbVie, Amgen, AnaptysBio, AstraZeneca, Boehringer-Ingelhiem, Cara Therapeutics, Innovaderm, Janssen, KAO, Kyowa Kirin, Leo Pharma, Pfizer, Regeneron Pharmaceuticals, Inc., and UCB; and is a consultant for AbbVie, Almirall, Amgen, Arena, Asana Biosciences, AstraZeneca, Boehringer-Ingelheim, Bristol-Meyers Squibb, Cara Therapeutics, Connect Pharma, Eli Lilly, EMD Serono, Evidera, Galderma, Ichnos Sciences, Incyte, Janssen Biotech, Kyowa Kirin, Leo Pharma, Pandion Therapeutics, Pfizer, Ribon, RAPT Therapeutics, Regeneron Pharmaceuticals, Inc., Sanofi, SATO Pharmaceutical, Siolta Therapeutics, Target PharmaSolutions, UCB, and Ventyx Biosciences. The other authors report no conflicts of interest.

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